ORTHOPAEDIC PHYSICAL THERAPY

SECRETS
DEDICATION

To my father and mother, Edward and Lillian for their love, support, and discipline. To my wife Laura, my best friend, a true blessing from heaven. To my four angels, Alexis, Baily, Lily and Addison, my life’s true joy and deepest love.

Jeffery D. Placzek

To my colleagues, my students, my patients, and my family, for constantly challenging me to be my best and press forward. Thank you!

David A. Boyce

In loving memory of the authors of this text who have passed away since the publication of our first edition.

Jack Echternach (February 23, 1932 – July 11, 2013) In memory of Dr. John “Jack” Echternach, Sr, PT, DPT, Ed. D, ECS, FAPTA. A leader, advocate, and mentor who inspired us to love what we do, and do what we love.

“A man knows when he has found his vocation when he stops thinking about how to live and begins to live.”

Thomas Merton

Dick Erhard (March 21, 1942 – October 4, 2009) A master clinician who mentored numerous physical therapy students and graduate manual therapy residents. A humble and skilled instructor who possessed a wonderful, warm, healing touch and demeanor. Those who were fortunate enough to spend time with him have been blessed and are better people because of it.

Harry Herkowitz (January 13, 1948 – June 7, 2013) As the Chairman of Orthopaedic Surgery at William Beaumont Hospital from 1991 to 2013, Dr. Harry Herkowitz, was deeply committed to the education of residents and fellows in orthopaedics and spine surgery. Dr. Herkowitz provided countless contributions to the realm of orthopaedic surgery in research, education, and patient care. He brought out excellence in those around him and expected perfection as a loving father would for his children. He will be remembered warmly by all those he mentored and cared for.
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We are pleased to announce the third edition of Orthopaedic Physical Therapy Secrets. The popularity of this text as a study guide for the orthopaedic and sports certification specialty examinations as well as a home and office reference guide has led us into a new updated edition. The new electronic version will allow for quick referencing as well as testing ones knowledge. One substantial improvement of the third edition is the addition of over 200 sample questions that are consistent with the level of difficulty one would encounter on the orthopedic or sports specialty examinations.

New chapters on innovative rehabilitation techniques such as therapeutic dry needling, functional movement screening, and selective functional movement assessment have been added. Significant updates related to concussion management, pelvic floor dysfunction, and foot orthoses is reflected in their respective chapters. And as always, all chapters have been edited to reflect contemporary practice standards.

The success of Orthopaedic Physical Therapy Secrets is due to the contributions of its authors in specialties ranging from anterior knee pain to X-ray. We would like to thank all of the authors that have contributed their time and expertise to making this text such a popular and sought after study and reference guide.

Jeffrey D. Placzek, MD, PT
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1. What is the organizational hierarchy of skeletal muscle, and how is it achieved?
The hierarchy:
- Muscle fascicles
- Muscle fibers or cells
- Myofibrils (arranged in parallel)
- Sarcomeres (arranged in series)

Achieved as follows: The connective tissue that surrounds an entire muscle is called the epimysium; the membrane that binds fibers into fascicles is called the perimysium. Two separate membranes surround individual muscle fibers. The outer membrane of fibers has three names that are interchangeable: basement membrane, endomysium, or basal lamina. An additional thin elastic membrane is found just beneath the basement membrane and is termed the plasma membrane or sarcolemma.

2. Describe the characteristics of the sarcomere.
- In the middle of the sarcomere, the areas that appear dark are termed anisotropic. This portion of the sarcomere is known as the A band.
- Areas at the outer ends of each sarcomere appear light and are known as I bands because they are isotropic with respect to their birefringent properties.
- The H band is in the central region of the A band, where there is no myosin and actin filament overlap.
- The H band is bisected by the M line, which consists of proteins that keep the sarcomere in proper spatial orientation as it lengthens and shortens.
- At the ends of each sarcomere are the Z discs. The sarcomere length is the distance from one Z disc to the next.
- Optimal sarcomere length in mammalian muscle is 2.4 to 2.5 μm. The length of a sarcomere relative to its optimal length is of fundamental importance to the capacity for force generation.

3. What are the contractile and regulatory proteins?
The most prominent protein making up the myofibrillar fraction of skeletal muscle is myosin, which constitutes approximately one half of the total myofibrillar protein. The other contractile protein, actin, comprises about one fifth of the myofibrillar protein fraction. Other myofibrillar proteins include the regulatory proteins tropomyosin and the troponin complex.

4. Name the structural proteins in skeletal muscle.
- C protein—part of the thick filament; involved in holding the tails of myosin in their correct spatial arrangement
- Titin—links the end of the thick filament to the Z disc
- M line protein—also known as myomesin; functions to keep the thick and thin filaments in their correct spatial arrangement
- α-Actinin—attaches actin filaments together at the Z disc
- Desmin—links Z discs of adjacent myofibrils together
- Spectrin and dystrophin—have structural and perhaps functional roles as sarcolemmal membrane proteins

5. What are the characteristics of myosin?
Myosin is of key importance for the development of muscular force and velocity of contraction. A myosin molecule is a relatively large protein (approximately 470–500 kD) composed of two identical
myosin heavy chains (MHCs) (approximately 200 kD each) and four myosin light chains (MLCs) (16–20 kD each). In different muscle fibers, MHCs and MLCs are found in slightly different forms, called isoforms. The isoforms have small differences in some aspects of their structure that markedly influence the velocity of muscle contraction.

6. Describe the components of myosin.
Light-meromyosin (LMM) is the tail or backbone portion of the molecule, which intertwines with the tails of other myosin molecules to form a thick filament. Heavy-meromyosin (HMM) consists of two subfragments: S-1 and S-2. The S-2 portion of HMM projects out at an angle from LMM, and the
S-1 portion is the globular head that can bind to actin. S-1 and S-2 together are also termed a myosin cross-bridge. There are approximately 300 molecules of myosin in one myofilament or thick filament. Approximately one half of the MHCs combine with their HMM at one end of the thick filament; the other half have their HMM toward the opposite end of the thick filament—a tail-to-tail arrangement. When molecules combine, they are rotated 60 degrees relative to the adjacent molecules and are offset slightly in the longitudinal plane. As a consequence of these three-dimensional structural factors, myosin has a characteristic bottlebrush appearance, with HMM projecting out along most of the filament.

7. Explain the role of the enzyme myosin adenosinetriphosphatase (ATPase).
   A specialized portion of the MHC provides the primary molecular basis for the speed of muscular contraction. The enzyme myosin ATPase is located on the S-1 subfragment. In different fibers, the myosin ATPase can be one of several isoforms that range along a functional continuum from slow to fast. The predominant isoforms of MHC are the slow type I and the fast types IIa, IIX, and IIb.

8. What are the characteristics of actin?
   Actin consists of approximately 350 monomers and 50 molecules of each of the regulatory proteins—tropomyosin and troponin. The actin monomers are termed G-actin because they are globular and have molecular weights of approximately 42 kD. G-actin normally is polymerized to F-actin (ie, filamentous actin), which is arranged in a double helix. The polymerization from G-actin to F-actin involves the hydrolysis of ATP and the binding of adenosine diphosphate (ADP) to actin; 90% of ADP in skeletal muscle is bound to actin. The actin protein has a binding site that, when exposed, attaches to the myosin cross-bridge. The subsequent cycling of cross-bridges causes the development of muscular force. The actin filaments also join together to form the boundary between two sarcomeres in the area of the A band. α-Actinin is the protein that holds the actin filaments in the appropriate three-dimensional array.

9. Explain the sliding filament theory of muscle contraction.
   A muscle shortens or lengthens because the myosin and actin myofilaments slide past each other without the filaments themselves changing length. The myosin cross-bridge projects out from the myosin tail and attaches to an actin monomer in the thin filament. The cross-bridges then move as ratchets, forcing the thin filaments toward the M line and causing a small amount of sarcomere shortening. The major structural rearrangement during contraction occurs in the region of the I band, which decreases markedly in size.

10. What are the functions of muscle?
   - Movement
   - Support and protection
   - Heat generation
   - Energy storage

11. List the functions of myonuclei and satellite cells, and identify the number of nuclei found in the skeletal muscle fiber.
   - Growth and development of muscle
   - Adaptive capacity of skeletal muscle to various forms of training or disuse
   - Recovery from exercise-induced or traumatic injury. Approximately 200 to 3000 nuclei per millimeter of fiber length

12. List the energy production systems in skeletal muscle.
   - Creatine kinase reaction
   - Adenylate kinase reaction
   - Glycolysis
   - Tricarboxylic acid (TCA) cycle and oxidative phosphorylation

13. What are the major steps of fatty acid metabolism in muscle that result in the release of energy?
   - Fatty acid activation and transport into the mitochondria
   - Beta-oxidation
   - Tricarboxylic acid (TCA) cycle
   - Oxidative phosphorylation

14. What is the range of muscle fiber lengths?
   Muscle fiber lengths range from a few millimeters in the intraocular muscles of the eye to >45 cm in the sartorius muscle.
15. Discuss the role of satellite cells in the formation of a new muscle fiber.
Satellite cells are normally dormant, but under conditions of stress or injury, they are essential for the regenerative growth of new fibers. Satellite cells have chemotactic properties, meaning they migrate from one location to another of higher need within a muscle fiber and then participate in the normal process of developing a new muscle fiber. The process of new fiber formation begins with satellite cells entering a mitotic phase to produce additional satellite cells. These cells then migrate across the plasma membrane into the cytosol, where they recognize each other, align, and fuse into a myotube, an immature form of a muscle fiber. The multinucleated myotube then differentiates into a mature fiber.

16. Identify and define or describe muscle growth factors.
Muscle growth factors are proteins that either promote muscle growth and repair or inhibit muscle protein breakdown. Examples include insulin-like growth factor, fibroblast growth factor, hepatocyte growth factor, and transforming growth factor.

17. What are the characteristics of myofibrils?
Individual myofibrils are approximately 1 μm in diameter and comprise approximately 80% of the volume of a whole muscle. The variable number of myofibrils is regulated during the hypertrophy of muscle fibers that is associated with growth; for example, the number of myofibrils ranges from 50 per muscle fiber in the muscles of a fetus to approximately 2000 per fiber in the muscles of an untrained adult. The hypertrophy and atrophy of adult skeletal muscle are associated with certain types of training and disuse and result from the regulation of the number of myofibrils per fiber. Training and disuse have negligible effects on the number of fibers in mammals.

18. Describe the characteristics of individual muscle fibers.
The cross-sectional area of an individual muscle fiber ranges from approximately 2000 to 7500 μm², with the mean and median in the 3000 to 4000 μm² range. Muscle fiber and muscle lengths vary considerably. For example, the length of the medial gastrocnemius muscle is approximately 250 mm, with fiber lengths of 35 mm, whereas the sartorius muscle is approximately 500 mm, with fiber lengths of 450 mm. The numbers of fibers range from several hundred in small muscles to >1 million in large muscles, such as those involved in hip flexion and knee extension.

19. What are the factors that upregulate protein synthesis in skeletal muscle?
• Amino acids
• Insulin
• Anabolic hormones such as growth hormone and testosterone
• Resistance training/muscle contraction

20. What is a strap or fusiform muscle? List examples of fusiform muscles.
Muscles that have a parallel-fiber arrangement are strap or fusiform muscles. In a parallel-fiber muscle, the muscle fibers are arranged essentially in parallel with the longitudinal axis of the muscle itself. Muscles with a parallel-fiber arrangement generally produce a greater range of motion (ROM) and greater joint velocity than muscles with the same cross-sectional area but with a different fiber arrangement.
• Sartorius
• Biceps brachii
• Sternohyoid

21. What are the factors that upregulate protein degradation in skeletal muscle?
• Inflammation
• Oxidative stress
• Catabolic hormones such as cortisol
• Energy stress such as starvation

22. Explain the role of pennation in force production.
When muscles are designed with angles of pennation, which is the most common architecture, more sarcomeres can be packed in parallel between the origin and insertion of the muscle. By packing more sarcomeres in a muscle, more force can be developed. As the angle of pennation increases, an increasing portion of the force developed by sarcomeres is displaced away from the tendons. As long as the angle of pennation is <30 degrees, the force lost as a result of the angle of pennation is more than compensated for by the increased packing of sarcomeres in parallel, producing an overall benefit to the force-producing capacity of muscle.
23. Describe the differences among unipennate, bipennate, and multipennate muscles.
   - In unipennate muscles, such as the flexor pollicis longus, the obliquely set fasciculi fan out on only one side of a central muscle tendon.
   - In a bipennate muscle, such as the gastrocnemius, the fibers are obliquely set on both sides of a central tendon.
   - In a multipennate muscle, such as the deltoid, the fibers converge on several tendons.

24. Define the force-velocity relationship.
   The muscle shortens at different velocities depending on the load placed on the muscle. As the load increases, the velocity decreases. When the load exceeds the maximal force capable of being developed by the muscle, a lengthening contraction ensues. The force developed during a shortening contraction is less than the isometric force. The force developed during a lengthening contraction exceeds the isometric force by 50% to 100% because of the increased extension of the attached cross-bridges.

25. Describe additional factors influencing muscle strength.
   The myosin structural state, the ratio of strong binding and weak binding cross-bridges to actin, muscle innervation, motor unit recruitment, and synchronization are all factors influencing muscle strength.

26. What is active insufficiency at the sarcomere level?
   Active insufficiency is the diminished ability of a muscle to produce or maintain active tension when elongated to the point at which there is no overlap between myosin and actin. It may also refer to the muscle being excessively shortened.

27. Define the all-or-none principle of muscle contraction.
   When a motor neuron is activated, all the muscle fibers innervated by that motor neuron contract maximally.

28. What is active insufficiency at the muscle level?
   This type of insufficiency is most commonly encountered when the full ROM is attempted simultaneously at all joints crossed by a two-joint or multi-joint muscle. During active shortening, a two-joint muscle becomes actively insufficient at a point before the end of a joint range, when full ROM at all joints occurs simultaneously. Active insufficiency also may occur in one-joint muscles, but this is not common.

29. Define excitation-contraction coupling.
   Excitation-contraction coupling is the physiologic mechanism whereby an electric discharge at the muscle initiates the chemical events that lead to contraction.

30. Summarize how excitation-contraction coupling occurs in skeletal muscle.
   1. Action potentials in the alpha motor neuron propagate down the axon to the axon terminals.
   2. Acetylcholine, the neurotransmitter at the neuromuscular junction, is released from the axon terminals.
   3. Acetylcholine diffuses across the neuromuscular junction and binds with acetylcholine receptors on the sarcolemma of the muscle.
   4. A muscle action potential is generated at the motor end plate.
   5. The muscle action potential travels along the sarcolemma and into the depths of the transverse tubules, which are continuous with the sarcolemma.
   6. The action potential (voltage change) is sensed by the dihydropyridine receptors in the transverse tubules.
   7. The dihydropyridine receptors communicate with the ryanodine receptors of the sarcoplasmic reticulum, a mechanism poorly understood.
   8. Calcium is released from the sarcoplasmic reticulum through the ryanodine receptors.
   9. Calcium binds to the regulatory protein, troponin C, and the interaction between actin and myosin can occur.
   10. Myosin cross-bridges, previously activated by the hydrolysis of ATP, attach to actin.
   11. The myosin cross-bridges move into a strong binding state, and force production occurs.
31. What are the characteristics of the different skeletal muscle fiber types?

<table>
<thead>
<tr>
<th>PROPERTY</th>
<th>I (S) (SO)</th>
<th>IIA (FR) (FOG)</th>
<th>IIB (FF) (FG)</th>
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<tbody>
<tr>
<td>Contraction speed</td>
<td>Slow</td>
<td>Fast</td>
<td>Fast</td>
</tr>
<tr>
<td>Force production</td>
<td>Small</td>
<td>Intermediate</td>
<td>Large</td>
</tr>
<tr>
<td>Fatigue resistance</td>
<td>High</td>
<td>High (intermediate)</td>
<td>Low</td>
</tr>
<tr>
<td>Fiber diameter</td>
<td>Small</td>
<td>Intermediate</td>
<td>Large</td>
</tr>
<tr>
<td>Red color</td>
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<td>Dark</td>
<td>Pale</td>
</tr>
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<td>Myoglobin</td>
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<td>High</td>
<td>Low</td>
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<tr>
<td>Capillary supply</td>
<td>Rich</td>
<td>Rich</td>
<td>Poor</td>
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<tr>
<td>Respiratory type</td>
<td>Aerobic</td>
<td>Aerobic</td>
<td>Anaerobic</td>
</tr>
<tr>
<td>Mitochondria</td>
<td>Many</td>
<td>Many</td>
<td>Few</td>
</tr>
<tr>
<td>Z line thickness</td>
<td>Intermediate (wide)</td>
<td>Wide (intermediate)</td>
<td>Narrow</td>
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<tr>
<td>Glycogen content</td>
<td>Low</td>
<td>High (intermediate)</td>
<td>High</td>
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<tr>
<td>Alkaline ATPase</td>
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<td>High</td>
<td>High</td>
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<tr>
<td>Acid ATPase</td>
<td>High</td>
<td>Low</td>
<td>Moderate</td>
</tr>
<tr>
<td>Oxidative capacity</td>
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<td>Medium-high</td>
<td>Low</td>
</tr>
<tr>
<td>Glycolytic ability</td>
<td>Low</td>
<td>High</td>
<td>High</td>
</tr>
</tbody>
</table>

32. Define the type IIX myosin heavy chain in human fibers.

The type IIX myosin heavy chain was first described in animals (rat, mouse). Type IIX myofibers have maximal shortening velocity and maximal isometric tension, which are intermediate between types IIA and IIB. The fiber type IIB in animals is not found in humans. Rather, the type IIB that is described in humans has a myosin composition very similar to that of type IIX.

33. Define the function of muscle spindles, and describe their appearance.

Muscle spindles provide sensory information concerning changes in the length and tension of muscle fibers. Their main function is to respond to the stretch of a muscle and, through reflex action, to produce a stronger contraction to reduce the stretch.

The spindle is fusiform in shape and is attached in parallel to the regular or extrafusal fibers of the muscle. Consequently, when the muscle is stretched, so is the spindle. There are more spindles in muscles that perform complex movements. There are two specialized cells within the spindle, called intrafusal fibers. There are two sensory afferents and one motor efferent innervating the intrafusal fibers. The gamma efferent innervates the contractile portion—the striated ends of the spindle. These fibers, activated by higher cortex levels, provide the mechanism for maintaining the spindle at peak operation at all muscle lengths.

34. What is the size principle of motor unit recruitment?

When skeletal muscles contract voluntarily against a load, motor units are recruited from smallest to largest.

35. Discuss the function of the Golgi tendon organs.

Connected in series to 25 extrafusal fibers, these sensory receptors also are located in the ligaments of joints and are primarily responsible for detecting differences in muscle tension. The Golgi tendon organs respond as a feedback monitor to discharge impulses under one of two conditions: (1) in response to tension created in the muscle when it shortens and (2) in response to tension when the muscle is
passively stretched. Excessive tension or stretch on a muscle activates the tendon’s Golgi receptors. This causes a reflex inhibition in the muscles they supply. The Golgi tendon organ functions as a protective sensory mechanism to detect and inhibit subsequently undue strain within the muscle-tendon structure.

36. Describe the adaptations in muscle structure that occur with progressive resistance exercises.
The major adaptation is an increase in the cross-sectional area of muscle, which is termed hypertrophy. The number of muscle fibers is minimally affected. Progressive resistive exercise involves 10 repetitions a day at 60% to 90% of maximal capacity; this results in an increase in strength by 0.5% to 1.0% per day over a period of several weeks. The fast-twitch type II fibers are more responsive to progressive resistance exercise than slow-twitch type I fibers. There are increases in the amounts of transverse tubular and sarcoplasmic reticulum membranes as well. Furthermore, neural adaptations result in an increased ability to recruit high-threshold motor units. The functional significance of this morphologic change is primarily a greater capacity for strength and power development.

37. List the effects of progressive resistance exercise.
- Increased mass and strength
- Increased cross-sectional area of muscle (increased number of myofibrils, leading to hypertrophy)
- Increased type I and type II fiber area
- Decreased mitochondrial density per fiber and oxidative capacity
- Increased intracellular lipids and capacity to use lipids as fuel
- Increased intracellular glycogen and glycolytic capacity
- Increased intramuscular high-energy phosphate pool and improved phosphagen metabolism

38. Describe the adaptations in muscle structure that occur with endurance exercises.
Endurance exercise has minimal impact on the cross-sectional area of muscle and muscle fibers. The smaller cross-sectional area allows better diffusion of metabolites and nutrients between the contractile filaments and the cytoplasm and between the cytoplasm and the interstitial fluid. There is a decrease in fatigability. The number of capillaries increases around each fiber, and there is an increase in mitochondria, especially in the type I fibers. The increased mitochondria can provide a good supply of ATP during exercise. The more extensive capillary bed improves the delivery of oxygen and circulating energy sources to the fibers, whereas the products of muscle activity are removed more efficiently. The functional significance of these changes is observed during sustained exercise, in which there is a delay in the onset of fatigue.

39. List the effects of endurance exercise.
- Improved ability to obtain ATP from oxidative phosphorylation
- Increased size and number of mitochondria
- Less lactic acid produced per given amount of exercise
- Increased myoglobin content
- Increased intramuscular triglyceride content
- Increased lipoprotein lipase (enzyme needed to use lipids from blood)
- Increased proportion of energy derived from fat; less from carbohydrates
- Lower rate of glycogen depletion during exercise
- Improved efficiency in extracting oxygen from blood
- Decreased number of type IIb fibers; increased number of type IIa fibers

40. What are the consequences of muscle disuse?
- The most striking consequence is atrophy—a reduction in muscle and muscle fiber cross-sectional area.
- The slow type I fibers show greater atrophy with disuse than the fast type II fibers.
- A few fibers undergo necrosis, and there is an increase in the endomysial and perimysial connective tissue.
- The muscles develop smaller twitch and tetanic tensions, beyond those expected on the basis of fiber atrophy.
- There is an increase in fatigability.
- There is a tendency for slow-twitch fibers to be transformed into fast-twitch fibers, with changes in the isoforms of the myofibrillar proteins.
• In the sarcolemma, there is a spread of acetylcholine receptors beyond the neuromuscular junction, and the resting membrane potential is diminished.
• The motor nerve terminals are abnormal in showing signs of degeneration in some places and evidence of sprouting in others.
• There is a loss of motor drive, such that the motor units cannot be recruited fully.

41. What adaptations occur if muscles are immobilized in a shortened position?
• Decreased number of sarcomeres
• Increased amount of perimysium
• Thickening of endomysium
• Increased ratio of collagen concentration
• Increased ratio of connective tissue to muscle fiber tissue
• Atrophy
• Altered strength
• Increased stiffness to passive stretch
• Increased fatigability

42. Define the term sarcopenia.
Sarcopenia is the term used to describe age-related loss of skeletal muscle mass and strength.

43. What occurs as a result of lengthening the muscles?
Sarcomeres are added.

44. What are the changes in skeletal muscles that occur with aging?
• Decreased size of muscle cells
• Decreased number of muscle cells
• Preferential loss of type 2 muscle fibers

45. Define disease-associated muscle atrophy, such as cachexia.
Disease-associated muscle atrophy occurs as a result of accelerated proteolysis. This form of skeletal muscle atrophy is systemic and associated with metabolic and/or inflammatory factors.

46. Differentiate apoptosis from necrosis as applied to skeletal muscle.
Apoptosis, or programmed cell death, is a regulated physiologic process critical to cellular homeostasis, which can become dysregulated, leading to disease states including muscle disease or dysfunction. Apoptosis results in cell shrinkage, DNA fragmentation, membrane blebbing, and disassembly into apoptotic bodies (membrane-bound cell fragments). Necrosis is a pathologic process caused by the progressive degradative action of enzymes that is generally associated with severe cellular trauma in muscles, leading to cell death.

47. What are the hallmarks of muscles undergoing degeneration-regeneration?
• Central nuclei
• Increased variation of fiber sizes

BIBLIOGRAPHY
CHAPTER 1 QUESTIONS

1. Which description about age-related changes in skeletal muscles is incorrect?
   a. Sarcopenia is the term describing the loss of muscle mass and strength with aging.
   b. Not only the quantity, but also the quality of muscles, decreases with aging.
   c. The physical activity level does not affect the changes of skeletal muscles with aging.
   d. Fast-twitch muscles show more significant age-related changes than slow-twitch muscles

2. Which factor does NOT upregulate protein synthesis in skeletal muscles?
   a. Cortisol
   b. Resistance training
   c. Insulin
   d. Amino acids

3. Which step is NOT involved in muscle fatty acid metabolism that results in the release of energy?
   a. \( \beta \)-oxidation
   b. TCA cycle
   c. Oxidative-phosphorylation
   d. Glycolysis
1. **Does kinematic similarity ensure kinetic similarity?**
   No. Kinematics is the description of motion without reference to the cause of motion. Kinetics refers to the causes of motion (forces). Although two movements may appear similar (kinematics), the underlying forces causing those movements (kinetics) may be very different. This fact should be appreciated when using readily available motion analysis tools (such as recording movements on smartphones or tablets). For example, some patients who have undergone ACL reconstructive surgery and subsequent rehabilitation have gait patterns that look normal compared with healthy controls but are produced by altered joint kinetics (larger contributions from the hip and decreased contributions from the knee). These differences have been found to persist even a year after surgery.

2. **Explain how impulse can be manipulated to prevent injury.**
   Impulse is the area under the force-time curve and accounts not only for the magnitude of the force but also for the duration over which the force is applied. Impulse determines the change in a body’s momentum, which is the product of mass and velocity. Applying a smaller force over a longer period of time will have the same impulse (and effect on a body’s momentum) as applying a larger force over a shorter period of time. Increasing the time of the impact, which can be accomplished by cushioned shoes and/or bending the knees when making contact with the ground, can attenuate the magnitude of an impact force and may decrease the risk of injury.

3. **What are some considerations to keep in mind when using elastic resistance?**
   Elastic materials, such as bands and tubes, are often used as a form of resistance and follow Hooke’s law (the force is proportional to the stiffness and elongation). The stiffness is determined by the manufacturer (which uses different colors for different levels of stiffness) and will decrease with time as the material fatigues. It is also important to keep in mind that the elongation is related to the resting length of the band or tube and not just the elongation during the exercise. For example, consider two scenarios. In the first, an exercise starts with the band at its resting length and is elongated by a certain amount, “x.” In the second scenario, the exercise starts slightly elongated by a certain amount “a” but is still elongated by the amount “x.” In the first case, the amount of elongation is “x,” and in the second case the elongation is “a + x.” The band is providing greater resistance in the second scenario, even though the elongation during exercise is the same. This highlights the need to ensure patients are using the same starting length each time they perform an exercise.

4. **Define commonly used biomechanical terms and equations.**

<table>
<thead>
<tr>
<th><strong>Common Biomechanical Terms and Equations</strong></th>
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<tbody>
<tr>
<td><strong>TERM</strong> (LINEAR; ANGULAR)</td>
</tr>
<tr>
<td>Displacement ($\Delta x$, $\Delta \theta$)</td>
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<td>Velocity ($v$, $\omega$)</td>
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</table>
5. **What is the relation between the linear motion at the joint surface and the angular motion of a bone around the joint axis?**

A theoretical construct, developed to describe this relation and advocated by Kaltenborn, is known as the convex-concave rule. In brief, if the convex surface of one bone is moving on the fixed concave surface of another bone, rotation and translation will occur in opposite directions. Additionally, if the concave surface of one bone is moving on the fixed convex surface of another bone, rotation and translation occur in the same direction. This rule should be appreciated when joint mobilizations are performed. It is proposed that in order to restore rotational motion at a joint, a linear mobilization is performed in relation to the treatment plane, which is parallel to the concave joint surface. Mobilization can be performed on either segment in accordance with the convex-concave rule.

6. **Has the convex-concave rule been experimentally verified?**

No, at least not for all joints. For example, it has been demonstrated that the glenohumeral joint contradicts the convex-concave rule during external rotation when the humerus is abducted to 90 degrees, and there is no clear consensus that the femur translates anteriorly when the knee is flexing in a weight-bearing position. However, these findings may not violate the convex-concave rule if the amount of translation in the direction of rolling is less than what the curvature of the convex segment would predict. The amount of rolling in one direction may be greater than the sliding in the opposite direction.
This “rule” pertains to the shape of the articular surfaces but does not account for the regional characteristics of the para-articular tissues, which may present as directional restrictions. Therefore the application of the convex-concave rule to treatment may need to be further informed by the direct method of assessing a restriction of joint gliding.

7. **Is the axis of rotation for a joint fixed?**
   In a nonpathologic joint, the AOR is generally fixed within the convex joint member and may stay in the same location (fixed AOR). A degenerated joint may lose its integrity, and the AOR may change its location throughout the range of motion. To reflect that change, the axis (or center) of rotation is called the instantaneous axis (or center) of rotation. Cartilage degeneration often accompanies the presence of a nonfixed axis of rotation.

8. **What is the difference between an absolute and relative joint angle?**
   An absolute angle is the angle that the distal point of a segment (e.g., foot, shank, thigh) makes with respect to some reference line (such as the horizontal for sagittal plane movements). A relative angle is the joint angle made by two segments (e.g., the knee angle is the angle between the shank and thigh). Relative angles can be stated as either internal (included) or external (anatomic) angles. An internal angle is the angle between the longitudinal axes of the two segments comprising a joint, while the external angle is the angular displacement from the anatomic position. For example, in the anatomic position, the internal knee angle is 180 degrees, while the external angle is 0 degrees. If this angle were decreased by 30 degrees, the internal angle would be 150 degrees and the external angle would be 30 degrees.

   ![Diagram of three types of angles: (A) absolute angle from the horizontal; (B) relative, internal angle; and (C) relative, external angle.](image)

   It is important to understand the distinction between these three measures and to be consistent in their use. In observational gait analysis, for example, ankle and knee measures are usually external relative angles, while the thigh is usually an absolute angle with respect to the vertical; many motion capture systems, on the other hand, report internal angles for all three joints.

9. **Are the terms valgus and varus used unambiguously?**
   Surprisingly, no. In accordance with their Latin derivations, in early writings varum referred to “knock-knees” and valgus referred to “bow-legged.” Taber’s Cyclopedic Medical Dictionary (21st ed.) still uses these definitions, which are the exact opposite of how they are generally used clinically. Because of the confusion this discrepancy can cause, it is prudent to understand how these terms are being used by various authors and not to assume that there are unambiguous and universally accepted definitions.
10. Provide examples of the concept of moment.

The moment of a force (“moment” for short), or torque, is the turning effect of a force. A force will have a tendency to rotate a body according to its magnitude, its direction, and the perpendicular distance between its line of application and the axis of rotation (this perpendicular distance is known as the moment arm). Knowing that the moment is the product of the force and the moment arm, the length of the moment arm can be manipulated to increase or decrease the force required to complete a task. For example, low back injury prevention strategies are based on the premise of decreasing the moment about the low back during lifting by keeping the load as close to the spine as possible, thus reducing the moment arm of the external resistance. Similarly, flexing the elbows during abduction will decrease the moment arm about the shoulder, thus making the movement easier to perform. On the other hand, during manual muscle testing, the therapist can increase the demand on a muscle by applying the resistance as far from the axis of rotation as possible.

11. What is the effect of a muscle’s force on a joint system?

Just as forces can be combined together to determine a resultant, they can also be broken into components. The components are useful in identifying the different effects of a force on a joint. For example, a muscle force can be divided into the component that is perpendicular to the bone (causing it to rotate and create a shear force across a joint) and the component that is parallel to the bone (usually increasing the compressive force across a joint). Therefore in addition to causing movement at a joint, all muscle forces will affect the amount of compression at a joint. During rehabilitation of certain joint pathologies, it may be necessary to identify which therapeutic exercises will increase the force of a muscle (to strengthen it) without applying excessive compressive forces across the joint. For example, performing unilateral (as opposed to bilateral) exercises for the lumbar extensors will decrease compressive forces on the spine while increasing the demand on those muscles.

12. Explain how torque-producing capabilities of a muscle vary over a joint's range of motion.

Over the range of motion of a joint, the magnitudes of moment arms and forces may vary. The amount of force a muscle can produce is influenced by several properties, including the length-tension relationship, which states that when a muscle is too stretched or too shortened it cannot actively produce as high amounts of force as it can when the muscle is at its optimal length. For example, when using the deltoid to abduct the shoulder from 0 to 180 degrees, the moment arm of the deltoid increases and the force-producing capabilities also increase to a position of optimal length and then decrease. These variations result in an initial increase of torque production, until the optimal position is reached, and then a relatively constant amount of torque production from the deltoids ensues for the remainder range of motion. Based on this, it is important to keep the same manual muscle testing position, especially if the therapist wants to compare among patients or examine the effect of training over a period of time.

13. Can a muscle’s action at a joint change?

Yes. A muscle’s action at a joint is determined by the magnitude of the force and the direction of the force vector (a line roughly extending from the effective origin to the effective insertion). Tendon-transfer surgeries will often make use of this fact when a certain muscle group is paralyzed. Additionally, with flat feet (pes planus) the tibialis anterior’s role can change from that of a subtalar invertor to an evertor. Even if the absence of pathology, this can occur most notably at the hip. For example, the piriformis is an external rotator when the hip is in a neutral position but becomes an internal rotator when the hip is flexed beyond 90 degrees.

14. When a study refers to a net joint moment, what does that mean and what are the assumptions behind it?

One of the greatest limitations in biomechanics is that we cannot, with current technology, measure muscle forces in a noninvasive way. However, we can measure the acceleration of the limbs and forces between the body and the ground to calculate the net joint moment (NJM), which is the moment required to accelerate a limb in accordance with Newton’s second law. Despite the fact that muscles and other
soft tissue structures contribute to the NJM, and cocontractions of the antagonists can make the actual moment much greater than the NJM, we usually equate high NJMs with high muscle forces needed to produce that moment. So when a research study suggests that exercise A has a greater extensor NJM at the knee than exercise B, it assumes that there is no cocontraction of the hamstrings during both exercises, and exercise A has a higher demand on the quadriceps. Studies will normally report internal moments (as described previously) or external moments (which are as a result of external forces and inertia). Internal moments are equal in magnitude and opposite in direction to the external moment.

15. What is joint instability, and how does it differ from hypermobility?
Joint stability is the ability of a joint to maintain a posture or trajectory similar to an undisturbed behavior in the presence of a perturbation. Although joint instability would represent the lack of this ability, the definition used by investigators and clinicians within studies is inconsistent, with three main definitions: (1) excessive and occasionally uncontrolled range of motion resulting in frank joint dislocation; (2) small, abnormal movement in an otherwise normal range of motion that may result in pain because of “impingement” at the joint; and (3) a small amount of force necessary to move a joint through its range of motion (or low stiffness). Joint hypermobility describes a laxity of the joint, where there is increased flexibility and range of motion, and is often used interchangeably with instability by clinicians. However, a hypermobile joint may still be stable because of muscular influence and motor coordination, whereas an unstable joint may not be lax because of neurologic and muscular control.

16. How are force and strength related?
Force is a push or pull of one object on another. Force is a vector quantity, having both a magnitude and a direction. Strength may be thought of as the ability to produce or absorb force. Measures of strength typically determine the maximum force a muscle or muscle group can produce.

17. Does the amplitude of the electromyography (EMG) signal quantify a muscle’s force-producing (absorbing) capability?
No. A muscle’s force-producing (absorbing) capability is primarily determined by the:
- Type of muscle action (concentric, eccentric, isometric)
- Length of muscle (force-velocity relation)
- Physiologic cross-sectional area of the muscle
- Number of motor units within a muscle that are activated (intramuscular coordination)
- Rate of motor unit activation (rate-coding)
- Intrinsic force-generating capability of the muscle (specific tension)
- Contractile history of the muscle (eg, prestretch)

The EMG signal quantifies the number of motor units and their rate of activation within the electrode field. In addition, because electrode placement can affect the number of motor units within the field, it is important to compare relative values (usually normalized to a maximum voluntary isometric contraction) rather than absolute values when comparing differences in EMG signals.

18. What are the benefits of having three different types of muscle actions?
Skeletal muscles are required to produce force, reduce (or absorb) force, or stabilize against a force. There is a different type of muscle action to fulfill each of these roles. A concentric muscle action produces force—the muscle moment is greater than the moment of an external force, and movement occurs in the direction of the muscle moment. An eccentric muscle action reduces force—the muscle moment is less than the moment of an external force, and movement occurs in the direction opposite of the muscle moment. The eccentric muscle action reduces the external force, and consequently decreases the acceleration caused by it. An isometric muscle action stabilizes against a force—the muscle moment is equal and opposite to the moment created by an external force, and no movement occurs.

19. What information can be obtained from studying the force-velocity curve?
Examining this relationship reveals that greater force can be produced isometrically (when the velocity is zero) than can be produced concentrically, and greater force can be produced eccentrically than can be produced isometrically.
Peak eccentric force is estimated to be between 120% and 140% of peak concentric force. Additionally, there is a negative relationship between force and velocity in the concentric range and a positive one between force and velocity in the eccentric range.

20. Is there a mechanical variable that can identify the types of muscle actions?
Yes; mechanical power is the product of the net joint moment and the angular velocity. If the NJM and the angular velocity are in the same direction, the power is positive and a concentric muscle action is controlling the velocity. If the NJM and angular velocity are in opposite directions, the work is negative and an eccentric muscle action is controlling the velocity. If there is an NJM but no angular velocity, the power is zero because there is no angular velocity, but the presence of an NJM indicates an isometric muscle action is preventing a velocity.

21. Why is eccentric strength important in the prevention of injury?
Although energy can be absorbed by all of the tissues of the body (eg, bone, ligament, muscle-tendon), the muscle-tendon complex has the greatest potential to safely absorb or distribute energy within the body. Eccentric muscle actions are the primary means by which energy is safely absorbed by the body. If the muscles are not strong enough, then other tissues must absorb this energy. Because the other tissues are not as capable of absorbing or distributing energy, energy levels can quickly exceed the tissues’ limits, resulting in injury.

22. Explain the length-tension relationship of muscle.
The amount of force or tension that a muscle can produce varies with the length of the muscle at the time of contraction. Maximum force is produced when the muscle is approximately at its resting length. When the fibers shorten beyond resting length, the force production decreases slowly at first and then rapidly. There is a progressive decline as the fibers are lengthened beyond resting length. This relationship can be used to help explain why surgically lengthened muscles are weak postoperatively (see figure). Although muscles typically do not operate over the entire length, this relationship helps explain the positions used for manual muscle tests, particularly for biarticular muscles. Biarticular muscles are
typically tested with one end of the muscle lengthened and the other end shortened, to place the muscle in the middle of its operating range. For example, when testing the hamstrings' action at the hip, the hip is usually extending (muscle shortening) while the knee is extended (muscle in a lengthened position).

23. **What is the stretch-shortening cycle?**
   The stretch-shortening cycle (SSC) involves 1) a well-timed preactivation of the muscle before an eccentric muscle action; 2) a short, rapid eccentric action; and 3) an immediate transition from an eccentric muscle action to a concentric muscle action. The subsequent concentric action is more forceful than it typically is because it was proceeded by the rapid eccentric action. The SSC is involved in many movements, from gait to jumping and throwing. Plyometric exercises are usually used to improve utilization of the stretch-shortening cycle.

24. **Is excessive force the cause of pain and injury?**
   Not directly. A better measure would be stress (force per unit area), which gives an indication of how that force is distributed. Although the term stress is used for reference to internal forces and pressure is used for external forces, clinically they can be used synonymously without much difficulty. Although a certain amount of stress is desirable, too much is believed to be the cause of injury and pain. Patellofemoral pain syndrome is believed to be the result of too much force (from the quadriceps) over too little area (patellofemoral contact area). The smaller contact area seems to have a stronger relationship to symptoms than does the increased amount of force. The insensate and poorly vascularized foot, in association with connective tissue changes, is vulnerable to increases in pressure and consequently the development of pressure sores. If the body weight transmitted to the foot can be dispersed over a larger surface area of the foot, the magnitude of pressure is decreased as is the chance for ulceration. The same factors apply to a person confined to prolonged bed rest; pressure sores may develop on areas where bony prominences contact the bed.

25. **What is the tissue response to a force (stress), and how is it measured?**
   The tissue response to a force (or load) is deformation, which is a change in the size or shape of the tissue. Deformation is usually expressed as the quotient of the change in tissue length divided by the tissue’s original length, or strain. Laboratory experiments usually apply a given force (N) to a tissue of known cross-sectional area (mm²) and specified length (mm), in which the resulting deformation (mm) is measured. Simple calculations will produce the applied stress and resulting strain. In vivo, force, either exerted by subject (active) or caused by an apparatus (passive), is measured using a dynamometer and the deformation (here displacement) is measured using an imaging technique (ie, ultrasound).
   
   Not all tissues can be measured in this way; musculotendinous units are accessible to testing in vivo, but cartilage is not.

![Length-tension curve.](image)
26. What information can be ascertained from studying force-deformation curves?

Plotting force on the vertical axis and the corresponding deformation on the horizontal axis produces a force-deformation curve, which graphically represents the relationship between the two (see figure).

Several important tissue qualities can be determined from this curve, including:

- **Ultimate strength**—the point on the curve where the tissue fails
- **Yield point**—the point at which a permanent deformation occurs
- **Elastic region**—the portion of the curve preceding the yield point
- **Plastic region**—the portion of the curve following the yield point
- **Stiffness**—the slope of the curve in the elastic range
- **Energy**—the area under the curve

When force is normalized to the area over which it is distributed and elongation is normalized to the resting length, we will have a stress on the vertical axis and strain on the horizontal axis. This curve provides insight into the material properties of the studied tissue, and its slope is the Young’s modulus.

![Force-deformation curve](image)

27. Do human tissues respond to all stresses in the same way?

No. Depending on the tissue and its role, tissues respond quite differently, and this difference in response is called anisotropism. For example, a tendon responds well to tension, but not as well to shear, and not at all to compression. Cartilage, on the other hand, responds well to compression. Human bone can handle compressive force best (such as pushing both ends of the bone toward each other), followed by tension (such as pulling both ends of the bone away from each other), and then shear force (such as pushing the top of the bone to the right and the bottom of the bone to the left). A bending force basically subjects one side of the bone to compression, while the other side experiences tension; therefore, the side subjected to tension usually fails first (immature bone may fail in compression first). For torsional loading (such as twisting the top part of the bone, while holding the bottom of the bone in a fixed position), fracture patterns typically show that the bone fails as a result of shear forces and then tension.

28. When the force is applied to the tissue externally, does the tissue return to its original state after the force is removed?

It depends on the amount of force applied. At lower levels of force the tissue returns to its original form, and therefore this stage is called the elastic region. It is in the elastic region that the characteristics of the tissue are stable and therefore are used to describe the tissue’s stiffness. If the force continues to increase, it reaches a transitional point—the yield point. The yield point is where the material changes from the elastic range to the plastic range. Beyond this yield point, permanent deformation will occur even after the load is removed.
29. Give an example of the clinical implications of the force-deformation curve.
The force-deformation curve can be appreciated clinically most easily during ligamentous testing. If the injurious force did not exceed the yield point, the ligament would return to its original length with no detectable changes in joint laxity. This injury would be classified as a first-degree sprain. If the injurious force exceeded the yield point but did not reach the ultimate strength of the ligament, the ligament would experience a permanent deformation that would be manifested as an increase in joint laxity. This injury would be classified as a second-degree sprain. If the injurious force exceeded the ultimate strength of the ligament, the ligament would catastrophically fail, and the subsequent force applied during ligamentous testing would be met with no resistance. This injury would be classified as a third-degree sprain.

30. Discuss some factors that affect the biomechanical properties of tendons and ligaments.

### The Most Commonly Cited Factors Affecting the Biomechanical Properties of Tendons and Ligaments

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>PHYSIOLOGIC EFFECT ON COLLAGEN</th>
<th>MECHANICAL EFFECT</th>
</tr>
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<tbody>
<tr>
<td>Physical activity</td>
<td>↑Glycosaminoglycan content</td>
<td>Strengthens</td>
</tr>
<tr>
<td></td>
<td>↓Cross-linking</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑Alignment of fibers</td>
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</table>
31. Is cartilage the same in all joints?
   No. There are morphologic, biomechanical, metabolic, and histologic differences between types of cartilage in the joints of the lower extremities. Those differences, in part, are the reason why osteoarthritis is more prominent in the knee and hip joints than in the ankle joint.

32. Do all tissues adapt to change at the same rate?
   No. An obvious example would be the difference in change in volume response to resistive exercise by a muscle and a tendon. A tendon adapts to change more slowly than muscle because it has fewer cells (in this case, tenocytes) that are capable of facilitating adaptation. Bone adapts more slowly than muscle. Evidence on the rate of adaptation of ligaments, cartilage, and intervertebral discs is scarce, but it is believed that they develop more slowly than muscle. It is important to realize, during rehabilitation, that a muscle will regain its strength before the other tissues of the musculoskeletal system, and therefore muscle strength alone is not a good indicator of the rehabilitation process.

33. What does it mean that a tendon is more compliant?
   A more compliant tendon, typically accompanying degeneration, is a tendon where more displacement (m) occurs as a result of the same amount of force (N) produced by the muscle on the contralateral extremity. Compliance is the opposite of stiffness, which is the rate of change in force over displacement (N/m). Mechanical stiffness and patient-reported perceived sensation of stiffness are not related.

34. Are tissue responses to a submaximal stress time dependent?
   Yes; tissue responses do change with time of application (or loading rate). Even if the amount of load is in the elastic range, but applied for a longer time, it will continue to cause a deformation. This type of deformation is reversible and is called creep. Creep is caused by the exudation of interstitial fluid. The fluid exits most rapidly at first and diminishes gradually over time. Human cartilage takes 4 to 16 hours to reach creep equilibrium, and this is why humans become slightly shorter as the day passes. Creep can also be associated with injury. Prolonged flexion of the lumbar spine results in a creep of the posterior ligaments, which decreases joint stiffness and may predispose the low back to injury. It is prudent to advise patients to allow this flexion-creep to reverse itself before performing activities that require lumbar stability.

35. What is hysteresis?
   When viscoelastic tissue is loaded and then subsequently unloaded, the amount of stress is lower for a given amount of strain. This phenomenon is a consequence of the tissue’s viscosity and is called hysteresis. The area between the loading and unloading curves (shaded area, see figure) is a measure of hysteresis and represents the energy absorbed by the tissue, which is usually lost in the form of heat (although it could cause tissue damage).
Repeated loadings, as well as acute and chronic stretching, increase a tendon’s compliance and decrease the amount of hysteresis. These changes increase the energy returned during the stretch-shortening cycle (improving performance) and can decrease the risk of injury. These changes show that stretching has beneficial effects other than just improving the range of motion of a joint.

36. What is the role of cartilage in joint lubrication, and how might pathology affect it?
There are three different types of joint lubrication processes: hydrodynamic (fluid film), elastohydrodynamic, and boundary. With fluid film lubrication, the fluid between two surfaces separates the contact surfaces and distributes the loading between them. Synovial fluid is attracted to the area of contact between the joint surfaces, resulting in the maintenance of a fluid film. Increased fluid pressure deforms the articular surface (cartilage), creating greater contact area and resulting in the elastohydrodynamic process. With boundary lubrication, the fluid is absorbed on the joint surface, preventing direct contact between two surfaces and decreasing friction.

Effective sealing of fluid within the joint is important for maintaining joint lubrication. For example, it has been found that a hip acetabular tear undermines the fluid sealing and joint lubrication processes, thereby leading to an elevation in the hip joint friction. This can ultimately cause cartilage degeneration and arthritis.

37. What is friction, and is it good or bad?
Friction is a force, parallel to the contact surface, that opposes motion between two objects. The interlocking of irregularities in the contact surfaces causes friction. The magnitude of the friction force will depend on the material characteristics of the two contacting surfaces and will be lower if there is relative motion between the two surfaces.

Friction may be good or bad, depending on the situation. A certain amount of friction between the ground and our shoes is necessary for efficient movement and to prevent slipping, but it also wears
the soles of our shoes. High friction forces between the ground and the shoe increase the risk of ankle and knee injuries in sports where there is a lot of sudden turning or stopping, and repetitive friction forces to the skin can cause blisters. High friction forces at the joint surfaces cause wear, which leads to articular degeneration.

38. List biomechanical factors that affect a joint implant
   - Initial stability—based mainly on the surgery technique used and the implant design
   - Late stability—determined by bone growth and remodeling of the bone around the implant (biologic fixation); if cement is used, late stability is determined by the bone-cement and cement-implant interfaces
   - Stress shielding—affects the bone around the implant as the load typically goes through the stronger implant and not the bone surrounding it
   - Wear of the implant—cobalt-chrome implants are typically used for the femoral head to decrease frictional wear on the acetabular component; ceramic acetabular cups and femoral heads are used because of their low coefficient of friction, but their implant strength has been questioned in some studies (they break and you never get all the fine ceramic debris out of the joint); highly cross-linked plastic has been used for acetabular components, but microscopic wear over time has proven to create an environment of aseptic loosening at the bone/cement/implant interfaces
   - Wear debris—polyethylene wear can cause osteolysis and potential aseptic loosening
   - Changing the anatomic alignments—by the manner in which the implant is installed or the correction of any preoperative deformity (hip dysplasia, knee varum or valgum)

39. List factors that affect the stability of an external fixator.
   - Pin diameter—bending stiffness increasing by an order of the fourth power as the diameter increases
   - Number of pins used
   - Distance from the surface to the bone
   - Stiffness of the frame
   - Number of fixation planes
   - Bone integrity into which the pins are placed (infection, avascular necrosis/poor vascularity)

40. What happens to the strength of an intramedullary rod when its diameter is increased?
   Strength increases as the rod size increases by an order of the third power.

41. What happens biomechanically with improper fixation size?
   In the case of total knee arthroplasty (TKA), if the plastic tibial insert is too big, then the space is “over-stuffed” and the knee will not have full flexion or extension capability. If the insert is too small, then the joint is unstable and the ligamentous and capsular structures are too lax; the muscular length tension characteristics are also compromised and both scenarios are often accompanied by pain.

   In the case of total hip arthroplasty (THA), if the acetabular component is too big, then the femoral implant “fulcrums” against the lip of the cup with flexion and can cause dislocation. If the stem is too long, then leg length discrepancy may occur. If the femoral head is too big, then range of motion is decreased in all planes. If any of the components is too small, then risk of joint dislocation increases. Furthermore, the muscular length tension characteristics are disrupted, and the supporting capsule and ligaments are lax. The patients often feel weak and walk with a limp.

42. How do holes in the bone (ie, missing screw or following removal of plate) affect its strength?
   - Decreases the cross-sectional area of the bone; less bone at the hole and strength is decreased
   - Decreases strength by causing a stress concentration point, determined by the geometry of the hole and bone
   - A hole of 20% of the bone diameter decreases strength by 50%
43. How long does it take for strength to return to normal levels after the removal of a screw?
   It takes between 4 months and 1 year for strength to return to normal (provided normal bone physiology).

44. List the types of metals that are closest biomechanically to bone.

<table>
<thead>
<tr>
<th>With Regard to Modulus:</th>
<th>With Regard to Biocompatibility:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Aluminum</td>
<td>• Titanium (and titanium alloys)</td>
</tr>
<tr>
<td>• Titanium (and titanium alloys)</td>
<td>• Cobalt-chromium</td>
</tr>
<tr>
<td>• Stainless steel</td>
<td>• Stainless steel</td>
</tr>
<tr>
<td>• Cobalt-chromium</td>
<td>• Aluminum</td>
</tr>
</tbody>
</table>

45. How much strength does a well-placed lag screw add to fracture fixation?
   One should be able to assume that the strength of the fixation is determined by the pull-out strength of the lag screw, or approximately a 40% increase in strength over plating alone.

46. Do movement screens have diagnostic value?
   It is doubtful. Although a movement screen may alert you that something could be wrong with the way a person is moving, it cannot tell you the cause of a dysfunction. Additionally, some dysfunctions may not even be picked up by visual inspection as a result of compensations. The concept of a support moment highlights the need to be cautious when evaluating multijoint movement. Collapse of the lower extremity requires flexion at all three joints (ankle, knee, and hip). A sufficiently large NJM at the ankle or hip can prevent flexion at the knee, and vice versa. Summing all three NJMs is called a support moment, and the support moment has been shown to be less variable during repeated gait trials than any of the individual NJMs. The support moment demonstrates how a decrease in one NJM can be compensated for by an increase in another.

BIBLIOGRAPHY


1. What is the body’s initial response to soft tissue injury? How is it identified?

The inflammatory response is characterized by a chemically mediated amplification cascade that represents the body’s initial reaction to injury, whether caused by trauma, surgery, or metabolic or infectious disease. The principal signs of the inflammatory response are erythema (rubor), swelling (tumor), elevated tissue temperature (calor), and pain (dolor). Local vasodilation, fluid leakage into the extracellular and extravascular spaces, and impaired lymphatic drainage are responsible for the erythema, swelling, and increased tissue temperature. The fourth cardinal sign of inflammation—pain—is the result of mechanical distention and pressure of the soft tissues and chemical irritation of pain-sensitive nerve receptors.

2. Describe the phases of soft tissue healing.

The acute inflammatory phase begins immediately after injury and lasts 24 to 48 hours, although some aspects may continue for up to 3 weeks. The proliferative phase may begin early in the inflammatory phase but is thought to be most extensive approximately 21 days after injury. The matrix formation/remodeling phase begins 3 weeks after injury and may last for up to 2 years, although in many cases the majority of remodeling has occurred by 2 months. Because the time frames for these three phases overlap considerably, the accepted delineations should be used as general guidelines only.

3. Describe the basic vascular and cellular activities associated with the inflammatory reaction and the primary function of each activity.

Blood vessels at the site of injury initially undergo vasoconstriction, which is mediated by norepinephrine and usually lasts from a few seconds to a few minutes. If serotonin is released by mast cells in the area of injury, a secondary prolonged vasoconstriction occurs to slow blood loss in the affected region. Additional cellular activities after soft tissue injury include margination of leukocytes, which adhere to the vessel wall, and chemotaxis (movement of white blood cells through the extravascular space toward the site of injury), which begins the process of phagocytosis and removes the cellular debris caused by the injury.

4. Identify the key chemical mediators of the inflammatory response.

Both histamine and serotonin (5-HT) are released from granules of mast cells in the area of the injury. Histamine results in elevated vascular permeability, whereas serotonin is a potent vasoconstrictor. Kinins, notably bradykinin, also cause a marked increase in vascular permeability, much as histamine does. It is now recognized that numerous cytokines and growth factors are involved in the cellular response to inflammation and injury. Proinflammatory prostaglandins are believed to sensitize pain receptors, attract leukocytes to the inflamed area, and increase vascular permeability by antagonizing vasoconstriction. The primary mode of action of aspirin, nonsteroidal antiinflammatory drugs (NSAIDs), and steroids is to inhibit prostaglandin synthesis by deactivation of a key enzyme (cyclooxygenase).

5. Which cell type is especially prominent in the proliferative and matrix formation phases of connective tissue healing?

The fibroblast is the most common connective tissue cell. It is responsible for synthesizing and secreting most of the fibers and ground substance of connective tissue. Soft tissue injury signals the fibroblast to multiply rapidly and mobilizes free connective tissue cells to the injured area. Tissue bleeding, in the case of trauma-induced inflammation, will result in deposition of fibrin and fibronectin in the tissues. These substances form a substratum that enhances the adhesion of various cells during later stages of repair.

6. Describe the elements that comprise the connective tissue matrix.

The connective tissue matrix is comprised of fibrous elements (such as collagen, elastin, and reticulin) and ground substance, which consists principally of water, salts, and glycosaminoglycans (GAGs). The matrix provides the strength and support of the soft tissue and also serves as the means for diffusion of tissue fluid and nutrients between capillaries and cells.
7. What general factors affect connective tissue repair after tissue injury?

Healing after soft tissue injury is affected by the availability of a number of factors, including blood supply, proteins, minerals, and amino acids. Enzymes and hormones also play a role in tissue healing, as do mechanical stress and infection. Steroids suppress the mitotic activity of fibroblasts, which results in diminished deposition of collagen fibers and reduction in tensile strength. Antibiotic medicines inhibit protein synthesis and may adversely affect wound healing and scar formation. Disease processes such as diabetes mellitus significantly retard wound healing because small-vessel disease inhibits normal collagen synthesis.

8. What is the association of antibiotic medicines and acute tendinopathy and tendon ruptures?

Fluoroquinolones (FQs) (Cipro) are a popular class of antibiotics with broad-spectrum coverage for a number of gram-negative pathogens. Beginning first in 1983 there have been many anecdotal and case-controlled studies reporting the incidence of tendon ruptures associated with FQs, with 90% occurring in the Achilles tendon. Tendon rupture is nearly always preceded by spontaneous onset of pain within 2 to 3 cm of the insertion point, thought to be closely correlated with reduced vascularity at this site. Other tendons reported to be affected include the biceps brachii, supraspinatus, and extensor pollicis longus.

9. What risk factors are associated with FQ antibiotic-induced tendon rupture?

Identified risk factors associated with FQ (eg, Cipro)-induced tendon rupture or tendinopathy include age over 60, previous corticosteroid use, renal failure, diabetes mellitus, and a history of musculoskeletal disorders such as other tendon ruptures. The latency period between the administration of FQ antibiotic treatment and occurrence of tendinopathy has a median onset of 6 days with half of tendon ruptures occurring within 1 week of taking the medicine. FQs are known to cause direct toxicity to type 1 collagen synthesis and promote collagen degradation.

10. What influence does nutrition play in the soft tissue repair process?

Collagen biosynthesis is especially sensitive to the availability of proper nutrients. Lack of vitamins C and A impedes the process of collagen synthesis. Glucosamine, found within collagen type II, is the critical compound in connective tissue repair and production. Glucosamine is the precursor for compounds important to connective tissue health, such as chondroitin sulfate and hyaluronic acid, and increases proteoglycan production. Whether dietary supplements such as glucosamine have a significant and lasting effect on joint disease has not been well established in controlled clinical trials, though mounting evidence suggests that such supplements are beneficial. Recent studies indicate that glucosamine may limit the advancement of joint space narrowing associated with osteoarthritis, resulting in improved functional scores. Minerals such as zinc contribute to the normal rate of cell proliferation and ultimate wound strength.

11. What role does aging play in altering the soft tissue injury healing process?

Age-related effects on wound healing include attenuated metabolic activity, decreased vascular supply, diminished cellular biosynthesis, delayed collagen remodeling, and decreased wound strength. Despite these differences, many of which have been confirmed in animal studies, clinical experience indicates that older patients often undergo surgical treatment with no adverse healing responses related to aging.

12. How does tendinitis differ from tendinosis?

Historically, painful tendon conditions were referred to as “tendinitis” and were assumed to be an inflammatory condition. Over time, however, further analysis revealed that the condition was not attended by inflammatory cells but was a degenerative change in the tendon—more accurately referred to as “overuse tendinosis.” Tendinitis (paratendinitis) is a rather rare condition that may occur occasionally in the Achilles tendon but almost always in conjunction with a primary tendinosis. The paratenon, a double-layered sheath of loose areolar tissue, is attached to the outer connective tissue surface of tendons that do not have a synovial lining. Paratendinitis refers to inflammation and thickening of the paratenon sheath. The pathology of tendinosis is characterized by a loss of collagen continuity and an increase in ground substance, vascularity, and cellularity. Research indicates that substance P, a neuropeptide known to contribute to tendon pain, is upregulated with tendon overuse. The cellularity increase associated with tendinosis results from fibroblasts and myofibroblasts, but inflammatory cells are absent. Because of this new understanding, it has been recommended that the term tendinopathy replace the term tendinitis for describing tendon pathology.
13. What is the appropriate treatment for tendinosis?
Treatment efforts to reduce pain and tenderness include ice application, oral NSAID administration, iontophoresis, rest, and cortisone injection. However, because tendinosis is by definition a chronic condition, treatment usually focuses on a controlled eccentric training program, often lengthy (10 to 12 weeks or more in some cases) in duration.

For Achilles tendinopathy, the most widely adopted approach is the Alfredson protocol of eccentric heel-drop exercises. This high-volume regimen has been shown to be particularly effective in athletic patients, though less so in nonathletic and older patients. Evidence clearly points toward exercise and mechanical loading as the best documented strategy for treating patients with tendinosis.

14. What tissue changes occur in response to a period of immobilization after soft tissue injury?
Immobility after soft tissue injury alters the rate of the biological process of remodeling. Changes that result in this alteration include an increased density of cells (usually fibroblasts), the presence of myofibroblasts, a reduction in hyaluronic acid and chondroitin sulfate levels in the periarticular connective tissue, and a 4% to 6% reduction in water content of the same tissues after only 9 weeks of immobilization. Further changes include a shift in the balance between collagen synthesis and degradation, which results in a reduction in total collagen.

15. What is the effect of immobilization on stiffness and strength of injured soft tissue?
Experimental evidence in rabbits indicates that 9 weeks of immobilization results in a 50% reduction in the normal breaking strength of the medial collateral ligament. At the same time a significant increase in the intermolecular cross-links of collagen leads to contracture formation. Therefore the remodeled connective tissue after immobilization is both thicker (tendency toward contracture) and weaker, possibly because of the random alignment of collagen fibers.

16. How do stress and motion affect connective tissue repair after injury?
Stress and motion have a profound effect on the quality of soft tissue repair after injury or surgery. Many studies have documented that scar tissue forms earlier in mobilized tendons, is well oriented, and is not attended by adhesions, in contrast to scar tissue that develops without physiologic stresses. Exposure of scar tissue to physiologic tensile forces during the healing process results in a more mature and stronger union of tendon and ligament. Healing of articular cartilage involves a greater amount of collagen and glycosaminoglycans, less cellularity, and fewer scar tissue adhesions when accompanied by modest joint movements. Some experimental evidence indicates that ultrasound application to tenotomized Achilles tendons improves tensile strength of the tissue if administered during postoperative days 2 to 4. This response appears to be time-dependent and may be related to limiting the inflammatory response and encouraging fibroplasia and fibrillogenesis. In a similar manner, high-voltage electrical stimulation appears to augment protein synthesis and the ultimate strength of the tendon if applied during the early stages of healing.

17. After ligament and tendon repair or reconstruction, when is the soft tissue the strongest and when is it the weakest?
Much of the information related to this question has been derived from studies using animal models (primates and others) and should be interpreted with caution. General data indicate that the strength of the patellar tendon autograft used in anterior cruciate ligament reconstruction cases is strongest on the day that it is surgically implanted. As the tissue heals in its new location, its strength diminishes to significantly <50% during the first 4 to 8 weeks postoperatively. In the ensuing 3 to 6 months, there is a slow transformation of collagen type and revascularization of the graft tissue. Stiffness and load to failure continue to increase for many months, and at 1 year the tissue is reported to have achieved 82% of its original strength. The clinical implications are fairly straightforward: protect the graft in the early stages of rehabilitation, encourage closed-chain axial loading activity to minimize shear forces (joint translation), and emphasize maximal motor unit activation throughout the rehabilitation process.

18. What is the response of articular cartilage to chondroplasty (microfracture technique, abrasion, and drilling) of the undersurface of the patella?
The microfracture technique is used to stimulate tissue repair of full-thickness articular cartilage defects. A drill is used to make multiple perforations in the subchondral bone in the area of the cartilage defect in an effort to produce a “super clot.” Over a period of 8 weeks or more the super clot heals with a hybrid mixture of fibrocartilage and type II (hyaline-like) collagen. This hybrid repair tissue may be functionally better than fibrocartilage alone; early animal and human studies suggest that it is durable enough to function like articular cartilage.
19. Describe the scientific evidence supporting articular cartilage repair.
Reproduced chondrocyte cells harvested from the patient are injected under a periosteal flap covering the articular defect. Two-year follow-up studies of patients with femoral condyle transplants indicate excellent results; most patients developed hyaline-like cartilage in the defect site. Patellar lesions have not done as well, possibly because of shear forces or noncorrection of underlying malalignment abnormalities. Research is encouraging for focal chondral defects but not for generalized osteoarthritis of the joint. In addition, there is evidence that articular cartilage exposed to electric and electromagnetic fields can lead to a sustained upregulation of growth factors, enhancing its viability. The degradative enzymes in the synovial fluid of osteoarthritic joints are not conducive to cell transfer with cartilage transplant experimental procedures.

20. What growth factors are involved with soft tissue healing?
- Chemotactic factors—prostaglandins, complement, platelet-derived growth factor (PDGF), and angiokines
- Competence factors—activate quiescent cells, PDGF, and prostaglandins
- Progression factors—stimulate cell growth, such as IL-1 and somatomedins
- Enhancing factors—fibronectin and osteonectin

21. What is the effect of NSAIDs on muscle recovery?
Short-term use (<1 week) of NSAIDs after muscular strain may improve recovery. However, long-term use (>1 month) may result in decreased recovery.

22. What factors affect allograft strength?
Freeze-drying reduces the immunogenic response but also decreases strength. Greater than 3-megarad irradiation will also decrease strength. Less radiation (2 megarad) in combination with ethylene oxide will decrease graft strength. Allografts have a slower, less predictable recovery than autografts.

23. What growth factors may aid in soft tissue repair?
Platelet-rich plasma (PRP) has been shown to improve soft tissue healing in horses with improved collagen abundance and organization. Macrophage-secreted myogenic factors may someday play a role in inducing muscle repair. Specific chondrocyte growth factors and bone morphogenetic protein (BMP) have shown promise in improving cartilage repair.

BIBLIOGRAPHY


**CHAPTER 3 QUESTIONS**

1. Risk factors associated with Achilles tendon rupture related to FQ antibiotic administration include which of the following:
   a. Age over 60
   b. History of previous corticosteroid use
   c. History of diabetes mellitus
   d. History of renal failure
   **e. All of the above**

2. The most widely adopted and evidence-based approach for the treatment of tendinosis/tendinopathy is:
   a. Steroid injection at the lesion site
   b. **Eccentric exercises (eg, Alfredson protocol)**
   c. Progressive concentric strength training of the musculo tendinous unit
   d. Iontophoresis of 4% acetic acid solution to the tendon lesion

3. Which of the following growth factors for soft tissue healing is correctly matched with the appropriate agent?
   a. Progression factors – interleukin 2
   b. Enhancing factors – prostaglandins
   c. Chemotactic factors – platelet-derived growth factor (PDGF)
   **d. A and C**
1. What are the components that make up bone?
   - Cells
   - Ground substance
   - Fibrous tissue network
   
   The cellular component consists of osteoblasts, which produce and initiate mineralization of new bone and cartilage, and osteoclasts, which are essential for the removal of the callus for lamellar bone to be laid down. A third cell type found in mature adult bone is the osteocyte.

   The ground substance component of bone contains mostly calcium phosphate, glycosaminoglycans, and hyaluronic acid. Calcium phosphate helps to add rigidity and hardness to the bone.

   The fibrous component consists of collagen fibers, which help resist tensile stress, and elastin fibers, which add a resilient aspect to the bone.

2. Describe the effects of aging on bone structure.
   The most commonly known age-related change is a calcium-related loss of mass and density. This loss ultimately causes the pathologic condition of osteoporosis. Osteoporosis is a major bone mineral disorder in older adults that decreases the bone mineral content; as a result, bone mass and strength decline with age. In geriatric patients, the hormonal system regulating calcium metabolism is less efficient and responds poorly to the challenge of a calcium-incorporating process, such as callous formation. Aging influences tissues (ie, the kidneys, gastrointestinal tract, and endocrine system) of the body that affect calcium metabolism and bone physiology. Thus the process of fracture healing in the geriatric patient is altered to some extent. Calcitonin, a hormone associated with decreasing serum calcium levels and possibly the remodeling of bone, has a decreased responsiveness to a calcium challenge with age. This decrease in calcitonin response may account, in part, for the slow bone healing in geriatric patients. Bones of older adults can withstand about half the strain of the bones of younger adults. Bones of older adults are less pliable and less able to store energy. Although there are physiologic changes that occur during the aging process that can affect bone health, the more sedentary lifestyle of many older individuals also may account for many of the age-associated changes in bone health.

3. How does Wolff’s law apply to bone healing?
   The ability of bone to adapt by changing size, shape, and structure depends on the mechanical stress on the bone. When optimal stress is placed on bone, there is greater bone deposition than bone reabsorption. This results in hypertrophy of periosteal bone and increased bone density. When bone is subjected to less than optimal stress, reabsorption of periosteal bone can occur, resulting in a decrease in strength and stiffness. Optimal stress within an appropriate range is essential for bone strength.

4. List the different types of bone fractures.
   - Compound (open)—occurs when sharp ends of the broken bone protrude through the victim’s skin or when some projectile penetrates the skin into the fracture site
   - Closed—skin remains intact
   - Perforating (eg, gunshot-bullet penetration)—may involve loss of bone from the effect of high-level energy at the fracture site
   - Depressed or fissured—occurs when a sharply localized blow depresses a segment of cortical bone below the level of the surrounding bone (eg, a skull fracture)
   - Greenstick—occurs on one side of the bone but does not tear the periosteum of the opposite side (seen in children)
   - Spiral—caused by opposite rotatory forces pulling on the bone (twisting)
   - Oblique—oriented at an angle of ≥30 degrees to the axis of the bone
   - Transverse—oriented at a right angle to the axis of the bone
5. What is a bone bruise and how does it relate to bone fractures?
Bone bruise (bone marrow contusion) is now considered to be one of four types of bone injuries that fall under the general heading of fracture – the others being stress fractures, osteochondral fractures and frank fractures (described in the list earlier in #3). The distribution of bone marrow edema has been likened to a footprint left behind by the musculoskeletal injury and is produced by compression and traction forces impacting adjacent bones. Further analysis indicates that there are actually three types of bone bruises:

a. Subperiosteal hematoma—a concentrated accumulation of blood underneath the periosteum after high-force trauma and most often seen in the lower extremities

b. Interosseous bone bruise—occurs most often with repetitive, high-compression forces that damage the blood supply in the bone marrow; usually occurs in the knee and ankle of professional athletes (eg, football and basketball players and elite runners)

c. Subchondral lesion—occurs beneath the cartilage layer of a joint and is usually caused by extreme compression or shear forces. Often there is microscopic separation of the cartilage and the underlying bone and is, again, most often seen in football and basketball players.

6. How are bone bruises identified?
Bone bruises are not visible with plain film radiographs, though this imaging modality may confirm that a frank fracture has not occurred. Most often bone bruises are visualized by means of T1- or T2-weighted fat-suppressed MRI. Patients with bone bruises tend to have a prolonged clinical recovery time with antalgic gait, slower recovery of motion, and persistent effusion compared with those with similar joint injuries who do not also have this complication.

7. Discuss the stages of bone healing.
The first stage is referred to as the inflammatory phase, or the granulation stage, fracture stage, or clot stage. During this phase surviving cells are sensitized to chemical messengers that are involved in the healing process. This initial aspect of the first stage is probably completed within 7 days.

A second feature of the initial stage is the development of a clot around the fracture site (not seen in stress fracture healing). After the formation of the clot, granulation tissue forms in the space between the fracture fragments. This granulation tissue activates macrophages, whose function is to remove the clot. This second aspect of the initial stage lasts about 2 weeks.

The second stage is known as the reparative phase or callous stage and can be divided further into soft callous and hard callous stages. Osteoblasts and chondrocytes within the granulation tissue begin to synthesize cartilage and weave bone matrices (soft callus). Approximately 1 week later, the newly formed soft callus begins to mineralize. This mineralization concludes several weeks later with the formation of a fracture (hard) callus. The hard callus is detectable on radiographs because of the calcium it contains. The creation and mineralization of the callus can require 4 to 16 weeks to complete.

The third stage is called the remodeling or consolidation phase and involves several processes. First the callus is replaced by woven bone, which, in turn, is replaced with packets of new lamellar bone. The callus plugging the marrow cavity is removed, restoring the cavity. It has been estimated that the complete replacement of the callus with functionally competent lamellar bone can take 1 to 4 years.

8. Name some conditions that have a negative effect on the bone healing process.

<table>
<thead>
<tr>
<th>Technical Factors*</th>
<th>Biological Failures†</th>
<th>Miscellaneous Conditions</th>
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<tbody>
<tr>
<td>Infection</td>
<td>Vascular injury</td>
<td>Poor nutrition</td>
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<tr>
<td>Poor reduction</td>
<td>Failure to make or mineralize callus (because of metabolic abnormalities)</td>
<td>Alcohol abuse</td>
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<tr>
<td>Distraction</td>
<td>Formation of scar and fat tissue instead of callus</td>
<td>Smoking</td>
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</tbody>
</table>
Repeated gross motion of fracture fragments
Inability to replace woven bone with lamellar bone (eg, children with osteogenesis imperfecta)
Loss of local blood supply because of injury and/or surgical procedure

*In these situations, the potential for normal healing is present, but problems during the treatment have prevented the healing process from proceeding, resulting in delayed union or nonunion.

†Biological failures refer to abnormalities in the biology of the healing process that delay or prevent union even with proper treatment.

9. Discuss the effect that smoking has on the bone healing process. In studies in which animals were administered nicotine, a significant decrease in callous formation and an increase in the prevalence of nonunions were documented. Nicotine-exposed bones have been shown to be significantly weaker in a three-point bending test compared with controls. Smoking and nicotine have been shown to delay the revascularization and incorporation of bone grafts and to increase the pseudarthrosis rate in spinal fusion patients. One study found that patients with fractured tibias who smoked took 62% longer to heal than nonsmokers. Nicotine has been shown to have a direct inhibitory effect on bone cellular proliferation and function. These changes, taken together with the vascular effects, result in a decrease in the quantity and maturity of the fracture callus. It has been estimated that the risk of fractures is two to six times higher in patients who smoke because of reduced bone density in these patients. Somewhat unexpectedly, current and previous smokers have been shown to be significantly more likely to develop infections (including osteomyelitis) after fractures. Damaged soft tissue and impaired nerve function (neurogenic inflammation) can impede fracture healing by increasing the metabolic demand of the tissue repair system and limiting the benefit of supportive muscle function around the fracture site. Such failures usually require downward revision of the rehabilitation timetable and ultimate recovery potential for the patient.

10. What steps may be taken by a patient to promote accelerated fracture healing?
   a. Traumatic fractures of the long bones and patients with multiple fractures require as much as three times the caloric intake compared with normal nutritional demands.
   b. Specifically, increasing protein intake enhances growth factors, such as insulin-like growth factor-1, which exerts a beneficial effect on skeletal integrity and bone renewal in particular.
   c. Vitamins C, D, and K, along with mineral intake and antiinflammatory nutrients, should be increased. Antiinflammatory nutrients (antioxidants) repair oxidative damage that would otherwise suppress fracture healing. Such antioxidants include vitamins E and C, lycopene, and alpha-lipoic acid.

11. Discuss the effect calcium nutrition has on bone healing. Calcium plays an important role in helping attain peak bone mass during bone development and in preventing fractures later in life. The daily recommended allowance of calcium for nonpregnant, nonlactating women is 800 mg/day. This level increases to 1500 mg/day in postmenopausal, estrogen-depleted women. It is estimated that 75% of all women ingest less than the recommended daily allowance. Men tend to meet their calcium needs more successfully by consuming twice as much calcium at the same age. Multiple factors can affect the bioactivity of calcium. High-fat or high-fiber diets can interfere with or decrease the activity of calcium. Large doses of zinc supplementation or megadoses of vitamin A can lower calcium bioactivity. High-protein diets can decrease calcium reserves by increasing urinary excretion of calcium.

12. What other factors affect calcium absorption? Alcohol consumption can decrease the absorption of calcium by a direct cytotoxic effect on the intestinal mucosa. Various medications, such as glucocorticoids, heparin, and anticonvulsants, can affect calcium activity.

   Vitamin D increases serum calcium levels by enhancing intestinal absorption of calcium and enhancing parathyroid hormone–stimulating reabsorption of bone. A low level of vitamin D impairs the ability of the body to adapt to low levels of calcium intake and may contribute to the pathogenesis of osteoporosis. Intake of vitamin D alone has never been shown to improve fracture healing.

- Closed reduction—use of casting or traction
- Open reduction—surgical intervention using plates, screws, or other internal fixation devices
- Rigid external fixation—combination of closed and open reduction using percutaneous pins and external stabilizing bars

14. What are the advantages of closed reduction?
Avoidance of surgery, reduction of the fracture, and usually (except in the case of traction) a shorter hospital stay are all advantages of closed reduction. Usually the patient can safely begin gentle range of motion exercises several weeks before the fractured limb is strong enough to return to normal weight-bearing function or to withstand resistance at the fracture site. In later stages of fracture healing, splints can be worn to protect the fractured limb, which is to be removed at intervals to permit joint mobilization or bathing.

15. List advantages and disadvantages of open reduction.

Advantages:
- Precise bone reduction
- Early mobilization of joints
- Immediate stability, allowing earlier return to full function

Disadvantages:
- Increased possibility of infection
- Increased hospital stay
- Metal devices may require subsequent removal

16. How does rigid fixation affect bone healing?
When rigid fixation is used, there is no stimulus for the production of the external callus from the periosteum or the internal callus from the endosteum (secondary bone healing). Instead the fracture healing occurs directly between the cortex of one fracture fragment and the cortex of the other fracture fragment (primary bone healing). Primary bone healing involves a direct repair of the bone lesion by new bridging osteons that become oriented through haversian remodeling to the long axis of the bone.

17. What effects can internal fixation have on bone healing?
- Improper placement or tightening of plates, screws, nuts, or bolts in bone surgery may cause bone reabsorption because of local stress concentration or decreased vascular perfusion.
- Plates that are too rigid may cause bone atrophy secondary to preventing the bone from perceiving intermittent compressive stress.
- If the hardware needs to be removed, a secondary inflammatory response occurs that leads to weakening of the bone; the bone needs to be protected until it regains strength.
- If the plates are left in place, problems with stress along the plate-bone interface can occur.

18. List some advantages of weight-bearing activities after sustaining a fracture.
- Enhanced rehabilitation (eg, improved range of motion)
- Shorter hospital stay
- Less overall postfracture morbidity

Fat-pad signs constitute radiologic evidence of an effusion in the elbow joint and appear as areas of translucency on the lateral radiograph of the elbow flexed to a right angle. The fat-pad sign has an overall high negative predictive value (87%). The absence of the fat-pad sign can exclude a fracture and is a reliable indicator of the absence of a fracture. The presence of a fat-pad sign should only raise the suspicion of a fracture being present, however, because there may be a positive fat-pad sign with no fracture.

20. What is the most commonly overlooked fracture in adults at the time of injury?
Carpal scaphoid fractures are easily overlooked. Because fractures of the scaphoid may result in loss of blood supply to the bone and consequent avascular necrosis, most physicians elect to treat wrist injuries as a fracture (immobilization) until properly interpreted radiographs indicate otherwise.

21. Discuss the role of ultrasound in the treatment of acute fractures.
Low-intensity pulsed ultrasound (LIPUS) stimulation can accelerate the normal repair process in a fresh fracture and may help stimulate the healing process of nonunions. In animal models low-intensity
pulsed ultrasound at 0.1 to 0.5 W/cm² accelerated fracture healing. Pulsed ultrasound at higher doses (1.0–2.0 W/cm²) significantly inhibited the synthesis of collagen and noncollagenous protein, however. In clinical double-blind studies, ultrasound has been shown to decrease significantly the time for overall healing of grade I open tibial fractures and distal radial fractures. Ultrasound has been shown to reduce significantly the prevalence of delayed union in nonsmokers and smokers. In animal studies ultrasound increased bone mineral content and density, increased peak torque, and accelerated the overall endochondral ossification process. Ultrasound stimulation may increase the mechanical properties of the healing fracture callus by stimulating earlier synthesis of extracellular matrix proteins in cartilage. Recent systematic reviews of the literature regarding LIPOUS clinical studies have questioned the quality of the supporting research to date. However, the current evidence indicates that LIPOUS may be useful for comminuted and/or open fractures that involve patients with associated risk comorbidities such as older age, smoking history, those with diabetes, and malnourished individuals. Simple fractures in otherwise healthy people should not be the target of LIPOUS therapy according to the most recent evidence.

22. What effect does bioelectric stimulation have on fracture healing?
Implantable electric stimulation and pulsed electromagnetic field (surface application) have been used for healing nonunion tibial fractures with some success. Electric stimulation generally is thought to convert fibrous connective tissue to bone, possibly by simulating mechanical stress in the bone. The best results with implantable electrodes in animal studies have been associated with the cathode located in the fracture gap and the anode in adjacent bone or in the soft tissue. Ionic migration in response to external direct current is believed to be one probable explanation for the apparent efficacy of electric stimulation on bone healing.

23. What is the effect of NSAIDs on bone healing?
Although there is still no well-defined answer, prostaglandins are known to participate in the inflammatory response and to stimulate osteoclasts as well as increase osteoblastic activity and subsequent new bone formation. Long-term excessive use of these medications may reduce normal bone healing.

24. What are stress fractures, and how do they occur?
Fatigue or stress fractures occur in otherwise healthy individuals usually in response to a sudden increase in physical activity of several weeks’ duration. First described in military training as “march fractures,” they are now fairly common in young individuals engaged in athletic activities and almost always represent a form of training error. In weight-bearing bones the overactivity causes microscopic fractures (debonding of osteons) that do not totally heal from day to day, eventually resulting in macroscopic bone failure and severe pain during ambulation or running. Though more common in the lower extremities, they can also occur in the medial epicondyle of the elbow with excessive throwing. Standard treatment involves early identification and rest of the involved extremity with avoidance of high-impact activities until healing has occurred. Signs of healing include resolution of bone tenderness with palpation and radiographic indication of healing—bone sclerosis.

25. What is the best imaging method for detecting stress fractures?
In spite of severe pain experienced by the patient, initial plain film radiographs of individuals suspected of a stress fracture are usually normal (up to 3 to 4 weeks after the initial onset of symptoms). Consequently, MRI and technetium bone scans are considered the best imaging studies for identifying stress fractures. Bone scans, in particular, may show signs of bone uptake as early as 72 hours after the onset of symptoms. However, radionucleotide (bone) scans have a disadvantage, compared with MRI, of exposing the patient to ionizing radiation. The American College of Radiology also recommends computerized tomography without contrast for early detection of a stress fracture, if MRI is contraindicated.

26. What is bone transplantation (replacement), and why is it used?
Bone transplantation (replacement) is an aggressive surgical technique whereby an entire diseased bone is excised and a cadaveric allograft replacement is transplanted in its place. This is usually necessitated by malignant bone tumors—primary or metastatic—and most of the descriptions in the current literature are of cases of femur transplantation. The alternative is typically an above-knee amputation or a hip disarticulation. Allograft replacement of the femur is prone to a number of complications, such as refracture, infection, nonunion, and resorption of the graft.

27. What treatments are available for nonunions?
- Autogenous bone grafting and appropriate stable fixation
- Vascularized bone grafting
• Use of allografts or autografts with the addition of platelet-rich plasma (contains high levels of PDGF and TGF-β1)
• Use of bone morphogenic proteins such as BMP-2
  • Mesenchymal stem cells
  • Muscle-derived stem cells

28. How do Salter-Harris fractures influence the pediatric population?
The growth plate appears on a radiograph as a lucent line near the joint, and a fracture through that line can be missed easily unless there is some disturbance in the alignment of the bone. When there is an injury to the growth plate, growth disturbances may occur in that bone. The younger the patient, the greater the growth potential remaining; however, there is also the danger of significant growth disturbance.

29. What are the roles of various growth factors on bone healing?
• BMP—Bone morphogenic protein induces metaplasia of undifferentiated perivascular mesenchymal cells into osteoblasts.
• PDGF—Platelet-derived growth factor is chemotactic for inflammatory cells at the fracture site.
• TGF-β—Transforming growth factor-β stimulates the production of type II collagen and proteoglycans at the fracture callus.
• IGF-II—Insulin-like growth factor II stimulates type I collagen production and cellular proliferation.

BIBLIOGRAPHY
CHAPTER 4 QUESTIONS

1. Bone bruises (bone marrow contusions) are most often visualized with which of the following imaging modalities:
   a. Plain film radiographs
   b. Dexa-scan bone density imaging
   c. T1- or T2-weighted fat-suppressed MRI
   d. Bone scan – scintigraphy

2. A variety of factors affect the bone healing process. Which of the following factors is correctly matched?
   a. Biological failure – vascular injury
   b. Technical factor – poor reduction
   c. Miscellaneous conditions – alcohol abuse
   d. All of the above

3. What steps may be taken by a patient to promote accelerated fracture healing?
   a. Increase caloric intake
   b. Increase protein intake
   c. Increase vitamin and antioxidant intake
   d. All of the above
1. What factor is considered to be the best indicator of an individual’s level of aerobic capacity?
Maximum oxygen uptake (VO2max) is the best indicator of aerobic capacity.

2. How is VO2max determined?
VO2max is the product of cardiac output (heart rate \times stroke volume) and arteriovenous oxygen difference (A – VO2 diff).

3. How is VO2max measured?
VO2max is measured via various methodologies, including, for example:
a. Indirect calorimetry using a metabolic chart to estimate oxygen consumption via Haldane transformation.
b. Field-based tests (e.g., Cooper 12-minute test)
c. Nonexercise algorithm taking into consideration the patient’s self-reported physical activity, age, gender, and body mass index

4. Why is VO2max considered the best indicator of aerobic fitness?
It is dependent on several factors:
- Cardiac output
- Ventilatory capacity
- Circulation
- Ability of the tissues to remove oxygen from the blood

5. What are limiting factors in determining VO2max?
- In healthy individuals, maximal cardiac output
- In individuals with asthma, chronic bronchitis, or emphysema, ventilatory compromise
- In individuals with emphysema, abnormalities in the ventilation-perfusion ratio of the lungs
- In individuals with peripheral vascular disease, decreased tissue perfusion

6. Are the VO2max values the same in an individual performing various exercises (e.g., treadmill, cycling, arm ergometry)?
No; the VO2max value is different for each exercise. Differences are thought to be a result of the amount of muscle mass involved in the exercise. If similar muscle mass is involved, the VO2max value is highest when the individual is performing the specific exercise for which he or she has trained.

7. Why is the cardiac output and arteriovenous oxygen difference larger in individuals who engage in regular physical activity?
Chronic, sustained physical activity increases stroke volume, mitochondrial size and density, and CA pillarization.

8. How does the VO2max of a well-trained man compare with the VO2max of a well-trained woman?
When VO2max is expressed per kilogram of body weight, the VO2max of a well-trained man is approximately 20% higher than that of a well-trained woman. If VO2max is expressed relative to lean body mass, it is only about 9% higher in men. The cause of the difference is not known, but it may be as a result of a greater oxygen-carrying capacity in men caused by a higher hemoglobin content, larger blood volume, and higher cardiac output.

9. Define other common indicators of physical fitness.
- Blood lactate threshold—the intensity of exercise when there is a sudden increase in the amount of lactate in the blood
- Ventilatory threshold—the intensity of exercise when there is an increase in ventilation corresponding to the development of metabolic acidosis during exercise
10. Differentiate between physical activity, exercise, and physical fitness.

- Physical activity is defined as any movement produced by a person’s skeletal muscles that results in the expenditure of energy
- Exercise is defined as a subset of physical activity that is planned, structured, and repetitive with the goal of improvement or maintenance of a person’s physical fitness
- Physical fitness is a set of attributes that can be either health- or skill-related and the degree of which can be measured with specific performance tests

11. What are the five components of physical fitness?

1. Cardiovascular fitness—also known as cardiorespiratory fitness, is the ability of the heart, lungs, and vascular system to deliver oxygen-rich blood to working muscles during sustained physical activity
2. Muscular strength—the amount of force a muscle or muscle group can exert against a resistance
3. Muscular endurance—the ability of a muscle or muscle group to repeat a movement many times or to hold a particular position for an extended period of time
4. Flexibility—the ability of a joint to move through its full range of motion, from a flexed to an extended position
5. Body composition—the amount of fat in the body compared with the amount of lean mass

12. What is the effect of regular exercise on cardiometabolic parameters?

Exercise has the potential to reduce levels of C-reactive protein (CRP), homocysteine, total cholesterol, low-density lipoprotein (LDL)-cholesterol, triglycerides, fasting glucose and insulin, hemoglobin A1c, blood pressure, and also increase high-density lipoprotein (HDL)-cholesterol

13. What is the effect of regular exercise on neurologic parameters?

Exercise is associated with a reduced risk of Parkinson’s disease, Alzheimer’s disease, and cognitive function, with possible mechanisms occurring from exercise-induced changes in the cerebral blood flow and metabolism, decreases in cortical accumulation of amyloid-β peptides, and increases in brain-derived neurotrophic factors.

14. What is oxygen deficit?

Oxygen deficit is the difference between the amount of oxygen that is consumed and the amount of oxygen that is required to perform an exercise.

15. What effect does warming up have on the oxygen deficit?

It decreases it. Warming up increases blood flow, muscle temperature, and mitochondrial respiration, and these factors enable oxygen to be delivered to and used by the tissues more rapidly. There is less time for a deficit to develop, and this results in a smaller deficit.

16. How do the resting stroke volume, heart rate, and cardiac output of a well-trained athlete compare with those of a sedentary individual?

The resting stroke volume of an athlete is greater than that of a sedentary individual because of hypertrophy of the cardiac muscle in the athlete, which results in an increase in contractility and an increase in venous tone that lead to more blood being returned to the heart. Both the increased contractility and increased venous tone cause an increase in the strength of contraction of cardiac muscle and in the stroke volume.

The resting heart rate of an athlete is lower than that of a sedentary individual (athlete, 40–60 beats/min; sedentary individual, 70–75 beats/min).

The higher stroke volume of an athlete is canceled out by the lower heart rate, resulting in the resting cardiac output of an athlete being similar to that of a sedentary individual.

17. How does the stroke volume response to exercise in the upright position differ between individuals who are physically fit and those who are not?

In a trained individual, stroke volume continues to increase until VO2max is reached; in an untrained individual, stroke volume increases as exercise intensity increases up to about 50% of VO2max and then remains steady. Maximal stroke volume is higher in fit individuals, and the stroke volume for any submaximal exercise intensity is higher in a fit individual.
18. How do heart rate, stroke volume, mean total peripheral resistance, mean arterial blood pressure, and respiratory rate change when exercise is performed using the upper extremities compared with a similar amount of exercise using the lower extremities?
These changes occur mainly because vasodilation occurs in exercising muscles, and vasoconstriction occurs in nonexercising muscles. Upper extremity exercise involves smaller muscles than lower extremity exercise. During upper extremity exercise, more vasoconstriction is occurring than vasodilation. This causes an increase in total peripheral resistance, and changes in the other variables occur as a result of this.

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<td>Respiratory rate</td>
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<td>Total peripheral resistance</td>
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19. What is the acute response of systolic and diastolic blood pressure to aerobic exercise?
During acute incremental exercise, systolic blood pressure increases while diastolic blood pressure remains steady or slightly decreases. Post acute exercise, both systolic and diastolic pressure can lower in both hypertensive and normotensive individuals, often referred to as postexercise hypotension.

20. Describe the normal interaction of inotropes and chronotropes during exercise.
During exercise the initial chronotropes and inotropes are the sympathetic nerves that directly innervate the heart. A slightly delayed chronotope and inotope come from the adrenal medulla. When sympathetic nerves innervating the adrenal medulla are stimulated, epinephrine and norepinephrine are released into the blood. These hormones travel to the heart and perpetuate the response that was initiated by the sympathetic nerves.

21. What effect does a low partial pressure of oxygen (Po2) have on blood vessel diameter in the lung and in the systemic circulation?
Vessels in the lung constrict when exposed to a low Po2, whereas vessels in the systemic circulation dilate. The constriction of vessels in the lung shunts blood to the areas of the lung that are better ventilated. This results in better ventilation-perfusion matching, which causes more effective oxygenation of blood. Dilation of systemic vessels enables more blood to be delivered to the area. This results in better oxygenation of the localized tissues.

22. Discuss the effect long-term endurance training has on the heart and on blood volume.
Increases in plasma volume occur shortly after the initiation of intense endurance training. This appears to be caused by an increase in plasma albumin levels, which osmotically draws fluid into the vasculature. Higher plasma volumes cause an increase in venous return, left ventricular end-diastolic volume, and stroke volume. These changes can occur within 1 week of the initiation of endurance training. Hypertrophy of myocardial muscle also occurs with endurance training, but this is a slower process.

23. Describe the contributions of stored adenosine triphosphate (ATP), creatine phosphate, glycolysis, and aerobic metabolism toward providing ATP during intense exercise over time.
- Stored ATP is used primarily for maximal intensity exercise, causing fatigue after about 4 seconds.
- If the intensity of exercise is such that fatigue occurs after about 10 seconds, creatine phosphate is used to supply the energy to replenish the ATP stores during the last 6 seconds of exercise.
- Intense exercise lasting between 10 seconds and 2 minutes depends on anaerobic glycolysis for ATP production. The maximal intensity of exercise is not as great as it was when creatine phosphate was being used.
- For intense exercise lasting longer than 2 minutes, aerobic metabolism provides most of the ATP, and the maximal intensity of the exercise that can be sustained is only about half of what it was during anaerobic glycolysis.
24. What can be done to improve the systems for providing ATP during intense exercise?
To improve the ability of creatine phosphate to provide energy, several bouts of intense exercise should be performed for 5 to 10 seconds with a 30- to 60-second rest between bouts. To improve anaerobic capacity, several bouts of intense exercise should be performed for at most 1 minute in duration with 3 to 5 minutes of recovery between bouts.

25. What are the main muscle fiber types and their characteristics?
Type 1—slow oxidative
Type 2a—fast oxidative, glycolytic
Type 2b—fast glycolytic

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<thead>
<tr>
<th>Properties</th>
<th>Type I</th>
<th>Type IIa</th>
<th>Type IIb</th>
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<tr>
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26. Which type of muscle fiber is activated during moderate-intensity, long-duration exercise, such as jogging?
Slow-twitch type 1 fibers are primarily activated.

27. Which type of muscle fiber is activated during high-intensity, short-term exercise, such as sprinting?
Slow-twitch type 1 and fast-twitch type 2 fibers are activated.

28. Why are specific muscle fiber types activated during different kinds of exercise?
The activation of a particular motor unit depends on the size of the $\alpha$-motor neuron that innervates it. Type 1 fibers are innervated by small $\alpha$-motor neurons, which have a lower threshold of stimulation than type 2 fibers; type 1 fibers always are stimulated first. Type 2 fibers are stimulated only if the intensity of the exercise requires it.

29. Explain why movements become less precise and refined as low-intensity exercise is continued for a prolonged period of time.
Initially, low-intensity exercise uses motor units consisting of slow-twitch muscle fibers. These motor units have fewer muscle fibers than motor units with fast-twitch fibers, and this accounts for better control during low-intensity exercises compared with high-intensity exercises. If low-intensity exercise is prolonged to the point that glycogen is depleted, the fast-twitch motor units are recruited. These motor units have more muscle fibers and result in less control of movements.

30. Can the three muscle fiber types be changed as a result of exercise?
Type 1 fibers cannot be converted to type 2 fibers, but type 1 fibers can improve their ability to use anaerobic metabolism, and type 2 fibers can improve their ability to use aerobic metabolism. Type 2b fibers can be converted to type 2a fibers with endurance training or strength training.

31. What changes occur in muscle with endurance training?
Endurance training results in improvements in oxygen delivery and use. This is caused by an increase in capillary and mitochondria content and aerobic oxidative enzyme activity. Type 2b muscle fibers are converted to type 2a. The cross-sectional area of the muscle decreases, resulting in shorter diffusion distances for oxygen and carbon dioxide.
32. What changes occur in muscle with resistance training, and how long does it take for those changes to occur?
Resistance training causes synthesis of proteins in thick and thin filaments, resulting in an increase in the cross-sectional area. The ratio of mitochondrial volume to contractile protein volume decreases. The aerobic capacity of the muscle decreases, which hinders performance in endurance activities. Type 2b muscle fibers are converted to type 2a. It takes about 6 to 8 weeks for the addition of protein filaments, but conversion of type 2b to type 2a fibers begins after about 2 weeks.

33. What causes improvements in strength with resistance training?
In the first 2 weeks, 90% of the improvements are attributed to neural changes, including improvements in the recruitment pattern of motor units, increases in CNS activation, more synchronization of motor units, and less neural inhibition. After about 6 weeks of training, 80% of the improvements are from an increase in contractile proteins.

34. What is the cause of athletic amenorrhea?
Women who train heavily have higher levels of catecholamines, cortisol, and $\beta$-endorphins. These hormones inhibit the release of luteinizing hormone and follicle-stimulating hormone, which results in decreased levels of estradiol. This contributes to the cause of athletic amenorrhea. Studies have shown that physical and emotional stress, diet, and the presence of menstrual irregularity before training also contribute. The exact mechanism is not known.

35. Is it true that pregnant women who are physically fit deliver more easily?
There is some evidence to suggest this, but there is also evidence stating otherwise. However, the perception of pain may be less in physically fit women.

36. Summarize some physiologic changes that occur during pregnancy that affect exercise.
The American College of Obstetrics and Gynecology (ACOG) recognizes the following:

a. After the first trimester, the supine position results in relative obstruction of venous return by the enlarging uterus and a significant decrease in cardiac output.
b. Stroke volume and cardiac output during steady-state exercise are increased significantly.
c. Exercise during pregnancy induces a greater degree of hemoconcentration than does exercise in the nonpregnant state.
d. There is a 10% to 20% increase in baseline oxygen consumption during pregnancy.
e. Because of the increased resting oxygen requirements and the increased work of breathing brought about by physical effects of the enlarged uterus on the diaphragm, decreased oxygen is available for the performance of aerobic exercise during pregnancy.
f. There is a shift in the physical center of gravity that may affect balance.
g. Basal metabolic rate and heat production increase during pregnancy.
h. Approximately 300 extra kilocalories per day are required to meet the metabolic needs of pregnancy; this caloric requirement is increased further in pregnant women who exercise regularly.
i. Pregnant women use carbohydrates during exercise at a greater rate than do nonpregnant women; adequate carbohydrate intake for exercising pregnant patients is essential.

37. What are the American College of Sports Medicine (ACSM) guidelines for physical activity?
ACSM's physical activity guidelines for healthy adults recommends participating in at least 30 minutes of moderate-intensity physical activity 5 days per week or 150 minutes total of moderate-intensity exercise. For vigorous intensity physical activity, 20 minutes for 3 days per week is recommended.

38. What are the American College of Sports Medicine (ACSM) guidelines for muscular fitness?
The American College of Sports Medicine (ACSM) recommends that a strength training program should be performed a minimum of two nonconsecutive days each week, with one set of 8 to 12 repetitions for healthy adults or 10 to 15 repetitions for older and frail individuals. Eight to 10 exercises should be performed that target the major muscle groups.
39. List the general American College of Sports Medicine (ACSM) guidelines for an exercise program to decrease body weight.
   a. The most successful program to decrease body weight is one that combines exercise with dieting. Such a program decreases weight, decreases fat mass, and maintains or increases fat-free mass. If one diets without exercising, one may lose more weight than by combining diet and exercise, but fat-free mass is lost in addition to fat mass.
   b. An aerobic exercise program is most effective.
   c. Exercise should be performed at least 3 days per week at an intensity and duration to expend 250 to 300 kilocalories per exercise session for a 75-kg person. This usually requires a duration of at least 30 to 45 minutes for a person in average physical condition.

40. What are the American College of Sports Medicine (ACSM) guidelines for sustaining weight loss?
   For sustaining weight loss, the ACSM recommendation is to engage in >250 minutes/week of moderate-intensity physical activity.

41. Describe the “fit-but-fat” paradigm.
   • An individual could have adequate cardiorespiratory capacity but still be of an undesirable bodyweight.
   • Evidence suggests that overweight (and possibly obese) adults who are physically active may be just as healthy, or even healthier, than inactive normal weight adults.

42. What are the American College of Sports Medicine (ACSM) guidelines for an exercise program to preserve bone health?
   a. Type of exercise should include weight-bearing endurance activities such as tennis, stair climbing, and jogging intermittently during walking; jumping activities such as volleyball and basketball; and resistance exercises that involve all major muscle groups, such as weight lifting.
   b. Intensity should be moderate to high, in terms of bone-loading forces.
   c. The frequency of weight-bearing endurance activities should be 3 to 5 times per week, resistance exercise 2 to 3 times per week.
   d. Duration should be 30 to 60 minutes per day.
   e. The older adult should also perform activities to improve balance for the prevention of falls.

43. How do exercise and training affect the endocrine system and the resting levels of hormones?
   Most hormone levels increase during submaximal, short-term exercise with the exception of insulin, which decreases, and thyroid hormones, which do not change. Resting levels of ACTH, cortisol, catecholamines, insulin, and glucagons decrease with training. This may be related to greater energy stores or a decreased perception of stress.

44. Discuss prolonged, moderate-intensity exercise training and blood glucose levels in individuals with type 1 and type 2 diabetes.
   Blood glucose levels do not seem to change with a prolonged exercise program in individuals with type 1 diabetes, but they decrease in individuals with type 2 diabetes. Exercise causes the cells of type 2 diabetic patients to be less resistant to insulin. This seems to be most effective if exercise is performed at an intensity of 60% to 75% of VO₂max. Most type 2 diabetic patients are overweight. Exercise may help to reduce body fat percentage, which results in an increase in the number of insulin receptors, an increase in their sensitivity, or both. Exercise reduces the cholesterol level in type 2 diabetic patients. This, along with the accompanying weight loss, decreases the cardiovascular risk factors of these individuals, which is the most significant benefit of performing exercise. Although exercise has not been shown to improve blood glucose levels in individuals with type 1 diabetes, it is still recommended for the same reasons that exercise is recommended for individuals without diabetes.

45. Does exercise affect the prevalence of upper respiratory tract infections (URTI)?
   Few studies have addressed the effect of moderate-intensity exercise on URTI. Preliminary results indicate a decrease in URTI with moderate exercise. More evidence indicates an increased prevalence of URTI during heavy endurance training and 1 to 2 weeks after a marathon-type event.
46. Should patients with chronic obstructive pulmonary disease (COPD) be encouraged to exercise?
Ambulation distance and feeling of well-being can increase significantly with an exercise program in individuals with mild or moderate COPD. There is controversy regarding the benefits of exercise for individuals with severe COPD. Some studies have shown improvements in endurance, whereas others have found no change. Only patients with stable COPD are encouraged to participate in an exercise program in a nonmedical setting.

47. How does the heart rate response to exercise differ between normal individuals and individuals who have had heart transplants?
In normal individuals, heart rate increases rapidly with moderate exercise as a result of a decrease in parasympathetic nerve activity and an increase in sympathetic nerve activity. Transplanted hearts are denervated. Any change in heart rate must be caused by changes in circulating levels of catecholamines, which takes more time than altering nerve activity. It takes longer for the heart rate to increase when exercise is initiated, and it takes longer for it to return to resting levels after exercise.

48. How does resting heart rate differ between normal individuals and individuals who have had heart transplants?
Resting heart rate is higher in individuals who have had a heart transplant because they no longer have the normal parasympathetic tone to slow the intrinsic rate of depolarization of the sinoatrial node.

49. Why are individuals with thoracic-level spinal cord injuries at risk for fainting after exercising in the upright position with the upper extremities?
There is no sympathetic innervation to the lower limb vasculature, and there may not be any innervation to the adrenal glands (depending on how high the injury is). This results in a lack of vasoconstriction of the vessels of the lower extremities, venous pooling occurs, and syncope follows.

50. What is the most common problem associated with exercising in cold environments?
When people know they are going to be exercising in cold environments, they usually overdress, resulting in hyperthermia.

51. List strategies to avoid hypothermia and hyperthermia when exercising in a cold environment.
- Dress in layers that can be removed as the exercise progresses.
- Stay dry; heat is lost much more rapidly when you are wet than when you are dry.

52. Describe the physiologic changes that occur with exercising in the cold.
Compared with a thermoneutral environment, exercising in the cold results in less lipid metabolism and free fatty acid use but greater lactate production and higher ventilation, oxygen consumption, respiratory heat loss, and peripheral heat loss.

53. List possible causes for decreased maximal muscle strength and power with hypothermia.
- Increased viscosity of skeletal muscle
- Increased resistance to blood flow
- Decreased maximal nerve conduction velocity

54. What are the two most common problems associated with exercising in hot environments?
Dehydration and hyperthermia are the two most common problems in this situation.

55. How can dehydration and hyperthermia be avoided?
These problems cannot be avoided completely, but they can be limited by ingesting fluid while exercising. There appears to be a similar benefit between ingestion of pure water compared with carbohydrate and electrolyte drinks as far as controlling core temperature and cardiovascular changes.

56. Describe the physiologic changes that occur with exercising in the heat.
The principal physiologic responses of exercise in the heat include skin and muscle vasodilation, nonactive tissue vasoconstriction, maintenance of blood pressure, and sweating. The hypothalamus plays a crucial role in thermoregulatory integration.
57. Does living at high altitude improve exercise tolerance at high altitude?

Yes. The exercise response of subjects at a high altitude who live at moderate altitudes compared with subjects who live at sea level shows that individuals who live at a moderate altitude have less of a decrease in VO2max and blood lactate accumulation. They also have larger maximal ventilation during maximal exercise. Hematocrit levels increase after about 25 days of exposure to high altitude, which should increase performance. Some studies indicate that pulmonary function, cardiac output, muscle enzyme capacity, and lean body mass decrease at high altitudes. World-class athletes performing endurance exercises consistently seem to perform better if they train at a moderate altitude.

**BIBLIOGRAPHY**


CHAPTER 5 QUESTIONS

1. Which of the following factors is considered the best indicator of an individual’s level of aerobic capacity?
   a. Maximum oxygen uptake (VO₂max)
   b. Resting heart rate
   c. Ventilatory threshold
   d. Blood lactate threshold

2. Which of the following muscle fiber types is activated primarily during prolonged, low-intensity exercise?
   a. Type 1
   b. Type 2a
   c. Type 2b
   d. Type 2x

3. Which of the following is a method to assess cardiorespiratory fitness?
   a. Indirect calorimetry
   b. Field-based tests
   c. Nonexercise prediction equation
   d. All of the above
1. List uses and potential side effects of medications commonly used to treat types of arthritis.

<table>
<thead>
<tr>
<th>Medication</th>
<th>Common Usage</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSAIDs and COX-2 inhibitors</td>
<td>OA, soft tissue injuries, inflammatory arthritis</td>
<td>GI upset and ulceration, renal dysfunction, heart disease, bleeding</td>
</tr>
<tr>
<td>Glucocorticoids</td>
<td>RA, SLE, other inflammatory disorders</td>
<td>Infection (including TB), weight gain, diabetes, muscle weakness, bone loss, poor healing</td>
</tr>
<tr>
<td>Antimalarials (hydroxychloroquine)</td>
<td>RA, SLE</td>
<td>Vision change, rarely myopathy</td>
</tr>
<tr>
<td>Colchicine</td>
<td>Gout, pseudogout</td>
<td>Diarrhea, rarely bone marrow suppression</td>
</tr>
<tr>
<td>Sulfasalazine</td>
<td>RA, psoriatic arthritis</td>
<td>Rash, hepatitis, bone marrow suppression</td>
</tr>
<tr>
<td>Methotrexate</td>
<td>RA, psoriatic arthritis, other inflammatory conditions</td>
<td>Infection (including TB), skin cancers, lymphomas, heart failure</td>
</tr>
<tr>
<td>Leflunomide</td>
<td>RA, psoriatic arthritis</td>
<td>Diarrhea, weight loss, neuropathy, hypertension, infection</td>
</tr>
<tr>
<td>TNF inhibitors</td>
<td>RA, psoriatic arthritis, other inflammatory conditions</td>
<td>Infection (including TB), skin cancers, lymphomas, heart failure</td>
</tr>
<tr>
<td>Rituximab</td>
<td>RA, myositis, other inflammatory disorders</td>
<td>Infection (including TB), low WBC</td>
</tr>
<tr>
<td>Other biologic agents</td>
<td>RA, +/- psoriatic arthritis</td>
<td>Infection (including TB), skin cancers, lymphomas</td>
</tr>
</tbody>
</table>

NSAID, nonsteroidal antiinflammatory drug; COX-2, cyclooxygenase-2 inhibitor; OA, osteoarthritis; RA, rheumatoid arthritis; SLE, systemic lupus erythematosus; TB, tuberculosis

2. Describe characteristic signs and symptoms of rheumatoid arthritis (RA).

People with RA develop pain, swelling, and morning stiffness in the small joints of the hands and feet (sparing the distal interphalangeal joints [DIPs]), wrists, and knees. Ankles, elbows and eventually the shoulders, hips, neck, and TMJ joints may become involved with more severe disease. Rheumatoid factor (RF), anti-CCP antibody, and elevated markers of inflammation (sedimentation rate or C-reactive protein) may be present. A duration of at least 6 weeks makes the diagnosis more likely.

3. Who gets RA?

RA affects approximately 1% of the population worldwide. It may begin at any age, but there is a peak of onset in women of childbearing years and a second peak in elderly men and women. Genetic influences are important, with concordant rates of arthritis at 15% in monozygotic twins. More severe disease is associated with HLA-DR 4 subtypes.

4. Why is it important to diagnose RA?

- RA is a potentially destructive arthritis. It has been clearly shown that outcomes are much improved in terms of joint preservation and function with early initiation of treatment.
- RA is a systemic disease associated with increased mortality. Mortality rates are more than doubled, especially for MI and pneumonia.
- Aggressive therapy, even with its attendant risks, has been shown to prolong life.
5. What x-ray changes are typical of rheumatoid arthritis?
   - Soft tissue swelling is noted first.
   - Periarticular osteopenia follows.
   - Erosions may occur at the joint margin.
   - Joint space narrowing, malalignment, and progressive osteopenia occur over time.

6. Describe joint pathology in RA.
   Chronic changes include thickening and edema of the synovial lining of the affected joints. The underlying connective tissue cells become activated by many inflammatory mediators and invade and destroy cartilage and bone at the margins of the joints. This is called pannus formation.

7. List the most common hand and wrist deformities associated with rheumatoid arthritis.
   - Subluxation and ulnar deviation at metacarpophalangeal (MCPs)
   - Swan neck deformity (flexion at DIP, extension at proximal interphalangeal [PIP])
   - Boutonnière deformity (extension at DIP, flexion at PIP)
   - Flexion, radial deviation, and subluxation at wrist
   - Extensor tendon rupture at wrist

8. How do children present with juvenile inflammatory arthritis?
   Different patterns of involvement have been identified and are listed here. By definition, onset is before the age of 16.
   - Systemic onset with fevers, serositis, rash, arthritis, and other organ involvement
   - Oligoarticular (most common presentation) with four or fewer joints involved at onset
   - Polyarticular onset, behaves like adult RA
   - Psoriatic arthritis
   - Enthesitis-related
     Synovitis in children is typically not severely painful. Limping, activity modification, or noted swelling in joint(s) are more likely to bring a child to medical attention. A very painful swollen joint or joints should raise the concern of leukemia in a child.

9. What labs help in the diagnosis of RA?
   RF is an antibody, most often an IgM antibody directed against IgG antibodies, that precipitates immune complex formation. It is found in approximately 80% of patients with RA at some point in their illness. RF is associated with nodule formation, extraarticular disease, and more severe joint disease. RF is not diagnostic of RA, because it is present in many chronic diseases (low specificity). Higher specificity for RA (approximately 98%) is found by testing for anticyclic citrullinated peptide antibodies (anti-CCP antibody)—antibodies directed against specifically modified proteins found in the rheumatoid synovium. Anti-CCP antibody is also associated with more aggressive joint disease.

10. Does RA affect the spine?
    Rheumatoid arthritis is a disease of the synovial tissue, so discs are not involved. There is synovial tissue anterior and posterior to the odontoid process of C2. Inflammation there can cause erosion of the dens or loosening of the transverse ligament that holds the dens in place. Subluxation here can cause compression of the cervical spinal cord. The spine is otherwise not clinically involved in RA.

11. What are the possible medical emergencies associated with RA that physical therapists should keep in mind?
    - Cervical myelopathy from subluxation at C1-C2 can occur, usually in a patient with long-standing, polyarticular, and deforming RA. Even minor trauma may precipitate symptoms. Patients may have paresthesias, weakness, and hyperreflexia.
    - Infection is a significant risk, especially with immunosuppressive drugs. Consider especially with monoarticular joint flares.
    - Rarely, in the age of aggressive medical therapy, rheumatoid vasculitis can be seen with mononeuritis multiplex (multiple unrelated peripheral nerve deficits), skin lesions, and internal organ involvement.
    - Painful red eye may relate to inflammation in a variety of structures of the eye, some vision-threatening. Urgent ophthalmologic evaluation is indicated.

12. What are signs and symptoms of carpal tunnel syndrome?
    Numbness and tingling are noted in the first three fingers of the hand. With more prolonged and severe disease, there is a loss of muscle mass in the hypothenar area.
13. Is carpal tunnel associated with systemic disease?  
Although carpal tunnel syndrome is often associated with mechanical overuse and osteoarthritis, systemic diseases should be considered, especially with bilateral disease. Diabetes, RA, gout, and hypothyroidism are common secondary causes.

14. What is systemic lupus erythematosus (SLE)?  
SLE is a multisystem inflammatory disease that may cause fever, fatigue, rash, blood count abnormalities, renal disease, serositis, lung disease, nervous system changes, joint pain, and other problems.

15. Is lupus diagnosed by the presence of antinuclear antibodies (ANA)?  
ANA are present in 99% of patients with SLE, but false-positive results are very common (up to 30%–40% false positive, especially with low-titer ANA). Lupus is a clinical diagnosis.

16. List musculoskeletal problems that patients with SLE can develop.  
- Arthralgia and arthritis  
- Osteonecrosis  
- Tendinitis and tendon rupture  
- Fibromyalgia  
- Steroid myopathy  
- Polymyositis

17. Describe typical lupus arthritis.  
- Arthralgies are most common, without visible joint swelling.  
- When inflammation is present, it often involves the small joints of the hands, similar to the pattern in RA.  
- The arthritis is not erosive, although joint deformities may be seen (eg, Jaccoud’s arthropathy, with swan neck deformities).

18. Name the seronegative arthropathies.  
- Ankylosing spondylitis  
- Reactive arthritis (previously called Reiter’s syndrome)  
- Psoriatic arthritis  
- Enteropathic arthritis associated with inflammatory bowel disease

19. List the clinical features that the seronegative arthropathies share.  
- Enthesitis (inflammation at sites of insertion of tendons or ligaments into bone)  
- Sacroiliitis and other axial skeletal involvement  
- Asymmetric, peripheral pauciarticular inflammatory arthritis  
- Extraarticular disease involving the gastrointestinal or genitourinary systems, skin, and eye  
- Association with HLA-B27 (in patients with spondylitis)

20. List clinical features of psoriatic arthritis.  
- Psoriatic skin lesions (nail changes are common)  
- Asymmetric peripheral arthritis with DIP involvement  
- Sausage digits and other tendinitis  
- Occasional spondylitis and sacroiliitis  
- Occasional arthritis deformans with telescoping of digits

21. What is reactive arthritis?  
Reactive arthritis is a seronegative arthritis that is triggered by infection, typically by *Chlamydia*, *Shigella*, or *Yersinia*. The classic triad of arthritis, conjunctivitis, and urethritis is seen in a minority of cases.

22. How does the back pain of ankylosing spondylitis differ from mechanical back pain clinically?  
<table>
<thead>
<tr>
<th></th>
<th>Ankylosing Spondylitis</th>
<th>Mechanical Back Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset</td>
<td>Late teens to 20s</td>
<td>Any age (more common in older age)</td>
</tr>
<tr>
<td>Timing of onset</td>
<td>Insidious, nontraumatic</td>
<td>Often sudden, traumatic</td>
</tr>
<tr>
<td>Pain with rest</td>
<td>Increased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Pain with activity</td>
<td>Decreased</td>
<td>Increased</td>
</tr>
<tr>
<td>Stiffness</td>
<td>+++</td>
<td>±, usually &lt;15 min</td>
</tr>
</tbody>
</table>
23. What treatments are available for ankylosing spondylitis?
- Education and exercise are still very important.
- Extension exercises three times daily (swimming recommended), attention to erect posture, and sleeping without a pillow to prevent kyphosis are recommended.
- Nonsteroidal antiinflammatory drugs (NSAIDs) can help relieve pain and stiffness.
- Tumor necrosis factor (TNF) inhibitors have been shown to cause clinical improvement in the axial skeleton.
- Methotrexate and sulfasalazine improve inflammation in peripheral joints but are not helpful for spinal disease.

24. Describe x-ray changes in ankylosing spondylitis.
- Erosion, pseudowidening, sclerosis, and ultimately fusion of sacroiliac joints
- Squaring of vertebrae with shiny corners
- Syndesmophyte formation (ossification of the outer layer of the intervertebral disc), leading to bamboo spine
- Fusion of apophyseal joints

25. Are there red flags for a PT treating a patient with ankylosing spondylitis?
Persistent localized back pain after a fall should raise a concern of fracture with pseudoarthrosis. Bone density is typically low because of a lack of normal biomechanical forces across the vertebrae. If much of the spine is fused, there can be a great deal of movement at the fracture site, preventing normal healing.

26. What causes gout?
Gout is caused by the accumulation of uric acid crystals in synovial joints. Polymorphonuclear leukocytes are attracted to the joint, try to engulf the crystals, and release digestive enzymes and proinflammatory mediators causing severe pain, swelling, and warmth in affected joints.

27. What causes pseudogout?
Calcium pyrophosphate crystals initiate inflammation.

28. How can the crystal types in gout and pseudogout be distinguished?
Examination of synovial fluid under a polarizing microscope helps differentiate gout from pseudogout: uric acid crystals are needle-like and negatively birefringent, whereas pseudogout crystals are rod-shaped and positively birefringent.

29. Describe a typical episode of acute gout.
Acute gout episodes typically begin with the sudden onset of severe burning pain, often in the middle of the night, usually involving the first metatarsophalangeal (MTP) joint. The pain may be so severe that even the weight of the bed sheets may be unbearable. The joint appears red, swollen, and hot to the touch. Episodes usually resolve within 7 to 10 days. They may be precipitated by alcohol consumption, trauma, surgery, or immobilization.

30. What joints other than the first MTP may be affected in gout?
Any joint in the body can be affected, but the knee, ankle, midfoot, wrist, and hand are commonly affected. Tophi, or collections of uric acid crystals, are seen as painless lumps in chronic gout, often at the olecranon, fingers, toes, or Achilles.

31. How is acute gout treated?
Cold packs may be helpful, but no other modalities or exercise is recommended. NSAIDs are the mainstay of treatment; however, local injection or oral administration of corticosteroids is another form of treatment. Colchicine is also sometimes used.

32. Can gout be diagnosed by an elevated serum uric acid level?
No. Of patients with elevated serum uric acid levels, <20% develop gout and 30% of patients do not have elevated uric acid levels at the time of a joint flare-up. The diagnosis must be made by examining joint fluid.

33. How does pseudogout differ from gout?
Calcium pyrophosphate deposition disease can present similarly to gout (pseudogout) but also may present similarly to RA (pseudo-RA) or as aggressive osteoarthritis (OA). Calcium pyrophosphate deposits (chondrocalcinosis) often can be seen on radiographs as opacities in the knee joint space or in the triangular fibrocartilage of the wrist. Definitive diagnosis requires examination of joint fluid. Aspiration of fluid often is adequate to relieve symptoms. Local steroid injection or NSAIDs are used.
34. What is the differential diagnosis of a single red, hot joint?
Infection is the most dangerous condition associated with this diagnosis and must immediately be ruled out by testing a sample of synovial fluid. Other diagnoses include acute gout, hemarthrosis with or without trauma, pseudogout, RA, seronegative arthropathy, and other less common etiologies (eg, tumor or pigmented villonodular synovitis).

35. Describe the clinical signs of infected total joint prostheses.
With acute infection, wound dehiscence or drainage may be seen along with classic signs of inflammation (pain, redness, swelling, and heat). Later, pain and loosening of the joint may be the only signs. If acute infection is suspected, patients should be referred back to their orthopedist for immediate evaluation.

36. How are infected total joint arthroplasties treated?
If detected early, infected joint arthroplasties may be salvaged with aggressive lavage and intravenous antibiotics. If not caught early or if gram-negative bacteria are present, the joint must be removed and often cannot be replaced until after extensive antibiotic treatment.

37. How common is OA?
OA is the most common type of arthritis. Prevalence increases with age, and it has been estimated to affect >80% of individuals >75 years of age.

38. What are common types of inflammatory muscle disease?
- Polymyositis is an autoimmune disease manifested by symmetric weakness in the proximal muscles of the upper and lower extremities.
- Dermatomyositis also causes proximal weakness and inflammation of the skin.
- Inclusion body myositis causes asymmetric weakness, especially in the quadriceps, wrists, and finger flexors.

39. Is it safe to exercise with myositis?
Exercise has proven to be safe in patients with stable active and inactive muscle disease. Patients studied were found to be less likely to suffer from weakness because of their disuse.

BIBLIOGRAPHY

CHAPTER 6 QUESTIONS
1. What is the most accurate way to diagnose gout?
   a. elevated uric acid
   b. joint aspiration
   c. physical exam
   d. radiographic appearance
2. What laboratory study is most helpful in diagnosing rheumatoid arthritis?
   a. uric acid
   b. Anti-CCP
   c. ANA
   d. HLA-B27
3. Which x-ray finding is not typical of rheumatoid arthritis?
   a. Soft tissue swelling
   b. Periarticular osteopenia
   c. Erosions at the joint margin
   d. Syndesmophyte formation (ossification of the outer layer of the intervertebral disc), leading to bamboo spine
1. Define Virchow’s triad. Virchow’s triad represents the risk factors for the development of a deep venous thrombosis (DVT).

1. Endothelial injury—change to the vascular wall that serves as a potent thrombogenic influence. It may be caused directly by trauma (including surgical) or indirectly by hematoma formation or thermal injury.
2. Alteration in blood flow—arterial turbulence or venous stasis contributes to the development of thrombi. Stasis occurs during the time spent on the operating table and postoperatively because of immobilization or impaired ambulation.
3. Hypercoagulability—alteration in the blood coagulation mechanism that predisposes one to thrombosis. A transient hypercoagulable state may exist as a part of the normal host response to surgery.

2. List states that are associated with hypercoagulability.

**GENETIC**
- Antithrombin C deficiency
- Protein C deficiency
- Protein S deficiency
- Factor V Leiden deficiency
- Prothrombin G20210A mutation

**ACQUIRED**
- Postoperative
- Malignancy
- Postpartum
- Congestive heart failure
- Prolonged bed rest or immobilization
- Advanced age
Severe trauma
Nephrotic syndrome
Cancer
Obesity
Oral contraceptives
Prior thromboembolism

3. What is the most common inherited thrombophilia?
Factor V Leiden is the most prevalent inherited thrombophilia, accounting for approximately 20% of patients with DVT. It is present in 3% to 5% of people of northern European descent. The risk of thromboembolism is increased by a factor of 7 in persons who are heterozygous for the Leiden mutation and 80 times for persons who are homozygous for the mutation.

4. In the general population, how frequent does DVT occur?
In the general population, DVT occurs at a rate of approximately 1 in 1000 people.

5. How common are genetic factors in association with hypercoagulability?
Approximately 20% to 30% of patients with DVT have a predisposing genetic factor.

6. When do venous thrombi develop?
- DVT may begin during the surgical procedure.
- Patients may present with signs and symptoms of DVT 24 to 48 hours postoperatively.
- The risk of late postoperative DVT is recognized to continue for 3 months.

7. Describe the incidence of DVT after total joint arthroplasty.
Patients who undergo total hip arthroplasty or total knee arthroplasty are at high risk for DVT. If no prophylaxis is used, DVT occurs in 40% to 80% of these patients. Thromboembolic prophylaxis, early mobilization, and modern surgical techniques have reduced the incidence of fatal pulmonary embolism to 0.18%. Despite prophylaxis, venous thromboembolism remains the most common reason for emergency department readmission and death after a total joint arthroplasty.

8. Does the type of anesthetic used during surgery affect the incidence of DVT?
Regional epidural anesthesia has been associated with a reduction in overall, proximal, and distal DVT. Epidural anesthesia may reduce the overall incidence of DVT by 40% to 50%. Hypotensive anesthesia may also be beneficial.

9. List the clinical signs and symptoms of DVT.
- Calf pain
- Engorged veins
- Swelling
- Edema
- Calf cramping
- Low-grade fever
- Warmth
- Palpable cord along the course of the involved vein
- Erythema
- Pain along the course of the involved vein

10. Is DVT easily clinically diagnosed?
No. DVT may be difficult to diagnose on the basis of physical examination. In one study, the diagnosis was confirmed with diagnostic studies in less than half of those suspected of having a DVT. Most venous thrombi are clinically silent. A clinician may not rely on physical examination findings alone to diagnose a DVT.

11. What is Homans’ sign?
Homans’ sign is calf pain with forced passive foot dorsiflexion; it is a physical examination finding suggestive of DVT.

12. List differential diagnoses of DVT.
- Muscle strain
- Nerve compression syndromes
- Cellulitis
- Lymphedema
- Superficial thrombophlebitis
13. Name the most dreaded complication from DVT.
Pulmonary embolism (PE) is the most feared complication.

14. Describe the signs and symptoms of PE.
PE may be the first clinical sign of a DVT. The most common signs of PE are tachycardia, low oxygen saturation, and shortness of breath. However, the clinical presentation of PE is notoriously unreliable. The clinical signs of PE are nonspecific, and, as with DVT, diagnostic studies are needed to confirm the diagnosis.

A classic presentation of pulmonary embolism consists of pleuritic chest pain and dyspnea (40%). Patients also may present with cough, diaphoresis, apprehension, altered mental status, hemoptysis, tachypnea, tachycardia (most common finding, 85%), rales, fever, bulging neck veins (30%), and a pleural friction rub. In one study, nearly 40% of patients who had a DVT, but no symptoms of pulmonary embolism, had evidence of pulmonary embolism on diagnostic studies. Massive pulmonary embolism may present as syncope or sudden death. Two thirds of patients who suffer a fatal pulmonary embolus do so within 30 minutes of becoming symptomatic.

15. What are the electrocardiogram (ECG) findings of pulmonary embolism?
Typical ECG findings include ST segment depression or T wave inversion, right axis deviation, or right bundle branch block. The classic ECG pattern S1Q3T3 is rare.

16. What long-term complications are associated with DVT?
Chronic venous insufficiency secondary to venous dilation and valvular incompetence is a typical long-term DVT complication. At 5 years post-DVT, symptoms may include:
- Night pain (45%)
- Pigmentation changes (50%)
- Pain with prolonged standing (39%)
- Venous ulceration (7%)
- Edema (52%)

17. Discuss the modalities that are available to prevent the formation of a DVT.
- Heparin—may be given subcutaneously in the perioperative period. It may be given as a fixed dosage (5000 units every 8 to 12 hours) or as an adjusted low dosage (3500 units every 8 hours; then adjust the dose to desired anticoagulation).
- Low-molecular-weight heparin (LMWH)—typically given at a fixed dosage without the need for outpatient monitoring. However, it may be associated with a slightly increased incidence of postoperative bleeding and wound problems.
- Fondaparinux—a selective factor Xa inhibitor.
- Warfarin (Coumadin)—may take several days to reach therapeutic levels, and patients often are placed on heparin or LMWH until the warfarin is therapeutic.
- Aspirin—the benefit for patients after joint arthroplasty is not conclusively proven. Aspirin has been proven to be a safe drug, but more studies are needed to prove its efficacy for prevention of thromboembolism. Aspirin has been found to be effective in decreasing DVT when combined with exercise and graded stockings or leg pumps.
- Dextran—should be used cautiously. The additional fluid volume may result in heart failure in patients with low cardiac reserve. A decrease in renal function also may occur from excessive diuresis after administration of dextran.
- Mechanical—a variety of mechanical modalities exist. External pneumatic compression devices decrease the risk of DVT without bleeding risk by decreasing venous stasis and stimulating fibrinolytic activity. Calf and thigh sleeves exist, as well as pneumatic foot pumps. The devices should not be used on patients who have an acute DVT or lower extremity fracture. Compression stockings also may help prevent venous thrombosis.

18. What actions should a therapist take if a DVT is suspected?
The therapist should hold therapeutic intervention and immediately inform the physician. The patient should be non-weight-bearing on the affected lower extremity until he or she is evaluated by the physician. Diagnostic tests may be ordered by the physician to confirm the suspicion. Historically, patients
with DVT were admitted to the hospital and placed on bed rest. Today, mobilization may begin as early as 24 hours after anticoagulation therapy has been initiated. Early mobilization may help prevent long-term complications.

19. Discuss the sensitivity and specificity of diagnostic tests for DVT.
   - Duplex ultrasound—the screening test of choice for initial evaluation of patients with suspected DVT. Color flow Doppler imaging improves the ability to detect a clot. In patients with asymptomatic DVT, the sensitivity and specificity have been found to be 89% and 100%, respectively. When used as a screening tool in asymptomatic patients, the sensitivity and specificity are 62% and 97%, respectively. Duplex ultrasound imaging is highly operator dependent, and these values vary widely among institutions. It is less sensitive for detecting calf vein thrombi than those located more proximally.
   - Venography—the gold standard for the diagnosis of DVT in the calf and thigh, with sensitivity and specificity almost 100%. The procedure is not an ideal screening test because of cost and potential morbidity related to the test. The detection of pelvic thrombi is poor, unless direct femoral vein puncture is performed.
   - Impedance plethysmography—poor sensitivity (30%) for large thigh thrombi. Plethysmography is even less sensitive for small thigh and calf thrombi.
   - ¹²⁵I-Fibrinogen scanning—90% accurate in detecting calf vein DVT. It may be falsely positive in the thigh after total hip arthroplasty in which there is fibrin present at the surgical site.
   - Magnetic resonance imaging—also may be used to image DVT, particularly in the pelvis (100% sensitivity, 95% specificity), where it is more sensitive than venography. Magnetic resonance imaging is equally sensitive for detection of DVT in the thigh but inferior in the calf (87% sensitivity and 97% specificity).
   - D-dimer—a blood assay that can significantly reduce the need for emergent venous Doppler examinations. It has a sensitivity of 100%, specificity of 49%, positive predictive value of 22%, and negative predictive value of 100%.

20. If the presence of a DVT is confirmed, what treatments are available?
    Anticoagulation is the treatment of choice for venous thromboembolism.
    - Heparin or LMWH is initiated to prevent propagation and promote stabilization of the clot.
    - Warfarin therapy is commonly used for maintenance anticoagulation. Therapy is often continued for at least 3 months for DVT caused by surgery or other transient risk factors.

21. What are the mechanisms of action of heparin, LMWH, warfarin, aspirin, and fondaparinux?
    - Heparin—produces its major anticoagulant effect by inactivating thrombin and activated factor X (factor Xa)
    - LMWH—derived from heparin by chemical or enzymatic depolymerization to yield fragments approximately one third the size of heparin. LMWH acts similarly to heparin, but compared with the unfractionated form, LMWH has a greater ratio of antifactor Xa/antifactor IIa activity, greater bioavailability, and longer duration of action.
    - Warfarin—acts by inhibiting the synthesis of vitamin K-dependent clotting factors, which include factors II, VII, IX, and X, and the anticoagulant proteins C and S
    - Aspirin—inhibits platelet cyclooxygenase, a key enzyme in thromboxane A2 (TXA2) generation. TXA2 triggers reactions that lead to platelet activation and aggregation.
    - Fondaparinux—antithrombotic effect is as a result of antithrombin III (ATIII)-mediated selective inhibition of factor Xa. By selectively binding to ATIII, fondaparinux sodium potentiates (about 300 times) the innate neutralization of factor Xa by ATIII. Neutralization of factor Xa interrupts the blood coagulation cascade.

22. Define PTT, PT, and INR.
    - PTT (partial thromboplastin time)—used to monitor anticoagulation if patient is taking heparin
    - PT (prothrombin time)—used to monitor anticoagulation if the patient is taking warfarin
    - INR (international normalized ratio)—represents measured PT adjusted by reference thromboplastin so that all laboratories have a universal result of patient PT; usually kept between 2 and 3 for treatment or prevention of DVT
BIBLIOGRAPHY


CHAPTER 7 QUESTIONS

1. What is the initial test of choice for diagnosis of DVT?
   a. MRI
   b. Venography
   c. duplex ultrasound
   d. d-dimer

2. What drug is not used for the treatment of confirmed DVT?
   a. Heparin
   b. Coumadin
   c. Aspirin
   d. Arixtra

3. Which drug does not match its mechanism of action?
   a. Heparin - inactivating thrombin and activated factor X (factor Xa)
   b. Warfarin - inhibiting the synthesis of vitamin K-dependent clotting factors
   c. Aspirin - inhibits platelet cyclooxygenase
   d. Fondaparinux – inhibits bound and free thrombin
1. At what depth have tissue temperature changes been recorded after treatment with superficial ice?

Ice application is reported to lower tissue temperature in the skin, subcutaneous tissue, and muscle, depending on the amount of subcutaneous tissue (adipose), type of cold application, and length of time treated. Measurements of decreased temperature have been recorded at a 4-cm depth. Patients with little subcutaneous tissue showed more significant cooling with a much shorter treatment time.

2. Which method is more effective in lowering tissue temperature: ice massage or ice pack?

Both are effective. A 5-minute ice massage treatment in the lower extremity decreased skin temperature by 20 °C, subcutaneous tissue by 15 °C, and muscle temperature at a depth of 2 cm by 5 °C and at a depth of 4 cm by 4 °C. Zemke et al., measuring at an average depth of 1.7 cm, found that a 15-minute ice massage treatment of a 4-cm² area created an intramuscular temperature drop of >4 °C, reaching its lowest temperature at 17.9 minutes after the initiation of treatment. Zemke et al. also found that an ice pack treatment produced an intramuscular temperature drop of >2 °C and had its maximum effect at 28.2 minutes. The ice pack and ice massage resulted in the same minimum skin temperature of 29.67 °C. The extent of the temperature change seems to relate more to the length of application and the amount of subcutaneous adipose tissue. Clinical considerations include the size and location of the affected area, time allotted for ice application, and patient preference. Ice massage may produce its maximum effect sooner than an ice pack; however, if a large area is to be treated, an ice pack may be more efficient.

3. What is the effect of ice application on metabolic rate?

Lower tissue temperatures produce a decrease in metabolic rate and subsequently a decrease in demand for oxygen. This decreased need for oxygen serves to limit further injury, particularly in the case of acute tissue damage, when the blood supply and oxygen delivery are impaired, resulting in hypoxia.

4. What is the physiologic effect of cold application on the muscle spindle?

Cold-induced lower tissue temperature raises the threshold of activation of the muscle spindle, rendering it less excitable.

5. How may the physiologic effect of cold application be successful in reducing a muscle spasm or cramp?

A decrease in muscle tension is produced by the less excitable muscle spindle that is not altered by active or passive stretching exercises, which means that an ice pack can be employed successfully during a passive or active stretch of a muscle that is in spasm.

6. Describe the effect of therapeutic ice on local blood flow.

Maximum vasoconstriction occurs at tissue temperatures of 15 °C (59 °F). Normal skin temperature is 31 °C to 33 °C. The superficial vasculature has a sympathetic innervation that produces vasoconstriction when stimulated. The neurotransmitters for this system are norepinephrine and epinephrine. Norepinephrine secretion and epinephrine secretion are stimulated by exposure to ice and are secreted into the blood vessels, resulting in vasoconstriction. If the tissue temperature drops below 15 °C, vasodilation occurs as a result of paralysis of the musculature, which provides vasoconstriction or a conduction block of the sympathetic nervous system. Vasoconstriction can lead to vasodilation if ice application is such that a tissue temperature <15 °C is reached. If vasodilation results, there is no definite consensus regarding the overall effect on the blood flow. A decrease in
the amount of blood lost was reported in patients who showed lower joint temperatures; this would seem to indicate that the overall blood flow remains decreased. Intramuscular temperature recordings have shown a range drop of 1.5 °C in the calf to a 17.9 °C drop in the bicep. Neither of these temperature ranges should bring the muscle tissue temperature to <15 °C and should not produce vasodilation within the deeper or target tissues.

7. At what temperature does local tissue damage occur with ice application?
   Although the core body temperature is 98.6 °F, the shell temperature (temperature in the extremities) can vary depending on exposure to the environment. Frostbite occurs when the extremities or face has been exposed to cold such that there is a drop in shell temperature, resulting in freezing of the tissue. Tissue freezing occurs as ice crystals form in the extracellular areas, causing fluid to be drawn out of cells. The earliest or precursor stage of frostbite begins with tissue temperatures of 37 °F to 50 °F (3 °C to 10 °C). Zemke et al. indicated that tissue temperatures of 19 °C to 25 °C after ice treatment had no adverse effect and that consistent tissue damage does not occur until tissue temperature declines to —10 °C. Cold-induced vasodilation occurs at temperatures <15 °C, reaching a maximum at tissue temperatures of 0 °C (32 °F).

8. What is the ideal tissue temperature to achieve the optimal physiologic effects of cryotherapy?
   Optimal physiologic effects from cryotherapy are achieved at tissue temperatures of 15 °C to 25 °C.

9. How long do tissue blood flow and tissue temperature remain decreased after application of an ice pack?
   Forearm blood flow has been shown to return to normal gradually over a 35-minute period after a 20-minute ice pack treatment. A 15-minute ice pack treatment has been shown to produce a maximum intramuscular cooling effect at 28.2 ± 12.5 minutes, and a 15-minute ice massage has been reported to produce a maximum intramuscular cooling effect at 17.9 ± 2.4 minutes from the start of treatment. Zemke et al. indicate that, for a 15-minute treatment, the maximum cooling effect does not occur until the ice treatment is completed. Myrer et al. report that tissue rewarming begins at 5 minutes after ice pack or cold whirlpool (10 °C) treatment; however, the intramuscular temperature remains decreased relative to pretreatment temperatures for up to 50 minutes post treatment.

10. Which form of cold treatment is the most effective at relieving postoperative pain and swelling?
   There does not seem to be a consensus in the literature. Dervin et al. found no difference in pain level or total wound drainage between postacromioclavicular ligament reconstruction patients treated with a cryotherapy cuff device (Cryocuff) using cold water and those treated with room temperature water. Previous researchers reported a greater decrease in pain in those treated with a Cryocuff than in those treated with an ice pack. It is possible that, because of the postoperative dressing, the tissue temperature is not decreased to an effective level to produce analgesia or to decrease swelling in some cases.
   Crushed ice was compared with continuous flow cold therapy in patellar tendon graft anterior cruciate ligament (ACL) reconstructions. Patients receiving continuous flow cold therapy demonstrated a statistically significant decrease in pain, a decrease in pain medication usage, and an increase in range of motion 1 week after surgery compared with the patients receiving crushed ice.

11. Explain the impact of cold application on the diabetic patient.
   Caution should be exercised when using modalities in the diabetic patient. Non–insulin-dependent diabetes mellitus (NIDDM) patients exposed to cold-water immersion have been found to have a significantly reduced capacity to recover skin temperature compared with healthy controls. There did not appear to be a correlation between the severity of the disease process and the length of recovery time required. Underlying pathology to explain the slow recovery is probably related to sympathetic nervous system involvement and associated peripheral vascular disease. This needs to be considered in addition to the possibility of peripheral polyneuropathy resulting in impaired touch and hot/cold temperature sensation.

12. Should ice be used in the treatment of a subacute or chronic injury?
   Ice may be used for pain relief or decreases in muscle guarding or spasm, which may allow the therapist to achieve other objectives such as joint mobilization, stretching, or strengthening exercises.
13. What is the hunting response?
The hunting response is proposed to occur as a mechanism by which the body responds to extreme cold by vasodilation that occurs secondary to the extreme cold temperature. This vasodilation is proposed to last for 4 to 6 minutes and to be followed by vasoconstriction lasting 15 to 30 minutes. Recent studies have not been able to demonstrate this cycle. Cold-induced vasodilation has been shown to occur at tissue temperatures <15 °C, and some researchers recommend treatment duration of no greater than 20 minutes to avoid the peripheral vasodilation effect. The maximum temperature effect may not be achieved because recent studies indicate that ice pack treatment may not reach its maximum effect until nearly 39 minutes.

14. What is hyperbaric gaseous cryotherapy, and what are the proposed benefits?
Gaseous cryotherapy is based on a projection of CO2 microcrystals that are, under high pressure, sprayed onto the skin. It is designed to decrease skin temperature rapidly and to a greater extent than more traditional cold modalities. Skin temperature is rapidly decreased with gaseous cryotherapy triggering systemic cutaneous vasoconstriction. It is applied for a short time. However, it can potentially cause burns if not applied correctly. The proposed physiologic effects are pain inhibition through reduction in sodium pump activity, activation of the vasomotor reflex, and reduction in muscle tone.

15. What skin temperature can be achieved with hyperbaric cryotherapy?
In a study of normal male volunteers hyperbaric gaseous cryotherapy applied to the hand produced a significant decrease in skin temperature in the dorsal hand from 32.5 °C ± 0.6 °C to 13.0 °C ± 0.7 °C. This drop in surface temperature was greater than with the traditional ice pack. An ice pack applied to the same area also produced a significantly reduced skin temperature from 32.5 °C ± 0.6 °C to 13.0 °C ± 0.7 °C.

16. At what depth have tissue temperature changes been recorded after treatment with superficial heat?
Tissue temperature changes have been recorded at 1- to 2-cm depth. This may reach all desired tissues in the hand; however, in other areas of the body, subcutaneous tissue may prevent adequate heating of the desired structures. Superficial heat is proposed to affect deeper structures by conduction heating.

17. What is the desired therapeutic tissue temperature produced by heat?
Therapeutic heating effects are achieved when a tissue temperature of 41 °C to 45 °C is reached. When tissue temperatures are >45 °C, tissue damage can occur. Much greater temperatures than can be achieved with superficial heat (60 °C to 65 °C) have been proposed to provide a breakdown and structural change in the collagen fiber, resulting in tissue shrinkage. This tissue shrinkage may be useful in the treatment of capsular laxity or instability of the shoulder.

18. What is the oxygen-hemoglobin dissociation curve?
At rest, tissues require approximately 5 ml of oxygen from each 100 ml of blood traveling through the area. At the level of the lung where oxygen is transferred into the bloodstream, the PO2 is normally 104 mm Hg. This PO2 facilitates the association of oxygen to hemoglobin. At the level of the tissues, oxygen needs to be dissociated from the hemoglobin to allow it to be delivered. The PO2 at the level of the tissues needs to be <40 mm Hg to allow this dissociation to occur.

19. What does a shift in the oxygen-hemoglobin dissociation curve to either the right or the left signify?
- A shift to the right is called the Bohr effect, producing an enhanced dissociation of oxygen from hemoglobin and improving delivery of oxygen to the tissues from the bloodstream.
- A shift to the left produces an enhanced association of oxygen to hemoglobin, enhancing the delivery of oxygen from the alveolus to the blood and improving the oxygen saturation level.

20. Explain the mechanism by which heat reduces muscle spasm or cramp.
Either type of heating modality—superficial or deep—has been reported to decrease muscle tone. The physiologic mechanism for this effect may be caused by the decrease in firing rates of the afferent fibers in the muscle spindle when heat is applied. Heat also lowers the threshold for activation of the muscle spindle afferent fibers. This makes the spindle more excitable when movement is applied to the body part and results in increased muscle tension if the heat is applied during a passive or active
stretching treatment. For example, application of a hot pack to a muscle in spasm, during a passive stretch technique, may result in increased muscle tension. Superficial heat can help to decrease spasm but works better if the muscle is heated while at rest.

21. **Describe the effect of heat on a tight or shortened muscle, capsule, or tendon during stretching.**

Application of a heating modality before or during stretching may yield a benefit resulting from increased extensibility of collagen fibers in the associated supporting structures and tendons as well as decreased firing rates of the efferent muscle spindle fibers. In an environment of connective tissue healing, the immature collagen bonds can be degraded by heat. This allows the tissue to be stretched more effectively. A 25% increase in potential elongation of mature connective tissue is noted if the temperature of the collagen tissue reaches 40 °C. The therapist needs also to consider the possibility of increased excitability of the muscle spindle during passive and active stretching.

22. **What is the effect of heat application on local blood flow?**

The application of heat to the skin results in increased local blood flow as a consequence of vasodilation. This increase in blood flow increases the delivery of oxygen, nutrients, and metabolites to the area.

23. **Describe the physiologic effect of heat on muscle performance during exercise.**

- During strenuous exercise, there is an increase in blood flow to the muscle of up to 25 times that which occurs at rest, which is important to provide adequate oxygen to the area. Much of the increase in blood flow is because of vasodilation instigated by increases in muscle metabolism.
- Although during the actual contraction there is a decrease in blood flow from a wringing effect, resulting in the compression of blood vessels, muscle heating also occurs because much of the energy that helps muscle function is directed into the production of heat within the muscle.
- Treatment of an area with superficial heat can affect tissues directly at 1 to 2 cm in depth, and so it is suggested to heat deeper than 1 to 2 cm by compression and conduction.
- If a relatively superficial muscle is heated, such as in the forearm or hand, the result is increased muscle metabolism and blood flow secondary to vasodilation. This in turn allows for an increased supply of oxygen, which may be beneficial as a warm-up before initiating exercise with a patient. Heating the area has a similar, but less dramatic, effect on muscle metabolism and blood flow as activity does, and together there is an additive effect.
- Heat has been reported to produce a decrease in muscle strength for the first 30 minutes after treatment. Heat treatment after exercise may prove to be even more beneficial because it can have the effect of continued elevation in muscle metabolism and blood flow, providing greater levels of oxygen to the tissue during the period of recovery from activity.

24. **What is the effect of heat and ice on nerve conduction velocity?**

Heat results in increases in local nerve conduction, whereas the lower tissue temperatures resulting from ice treatment produce a relative slowing of nerve conduction. Cryotherapy applications do reduce sensory nerve conduction velocity by up to 22.6 m/s and motor NCV by up to 8.3 m/s when using cold water immersion. Significant but less dramatic decreases in motor and sensory nerve conduction velocity also can be achieved with ice massage and ice pack treatments. In addition, skin temperature values need to be monitored when performing nerve conduction studies, because a cool extremity may produce nerve conduction values that appear to be pathologic but are the result of lower skin temperatures.

25. **How do the superficial heat and ice modalities act to reduce pain?**

Heat and ice serve to stimulate thermoreceptors, which transmit the message proximally to the dorsal horn and may act to inhibit transmission of the painful stimulus by the gate control theory. Sluka et al. report that arthritic rats treated with ice had a delayed pain response, which indicated some effect of ice application on pain response. They also treated the arthritic rats with heat and found no change in the pain response but did notice a decrease in muscle guarding with heat application.

26. **Are home heat wraps effective in treating low back pain?**

A group of researchers examined this topic using prospective, randomized, single-blinded placebo-controlled clinical trials. The first study involved 219 subjects between 18 and 55 years of age with acute, nonspecific low back pain. Treatment was for three consecutive nights, and outcome measures were
taken for an additional 2 days. There was a significant difference in pain relief and decreases in muscle stiffness and disability, as well as increases in flexibility, compared with the placebo over all 5 days of outcome measurement.

27. Are low-level heating wraps effective when used in the extremities?  
A level I study did investigate the use of continuous low-level heat wraps in common wrist and hand conditions to include carpal tunnel syndrome, osteoarthritis, sprains, and strains. Low-level heat wraps were found to effectively improve pain, stiffness, and strength.

28. Is a contrast bath effective at reducing pain and edema?  
There is some evidence that the baths can elevate superficial skin temperature and superficial blood flow in the hand. These same researchers could not identify a relationship between the physiologic effects of contrast baths and hand function. A volumetric study of the effect of contrast baths on hand volume in CTS patients did not find any significant impact on edema either before or after carpal tunnel release.

29. Should an athlete return to performance immediately after local treatment with cryotherapy?  
A systematic review of 35 studies identified mixed results regarding muscle strength and performance immediately after local cryotherapy applications. However, the majority of studies evaluating whole body exercise demonstrated a decrement in athletic performance to include vertical jump, sprint, and agility even when cryotherapy was applied only to one specific body part.

30. How is hand dexterity affected by cold?  
Cryotherapy applications to the hand and forearm diminish hand dexterity and throwing accuracy immediately after treatment. Lower skin temperature associated with local cooling of the forearm and hand is also correlated with decreases in hand dexterity and strength.

BIBLIOGRAPHY


**CHAPTER 8 QUESTIONS**

1. Vasoconstriction can lead to vasodilation if a tissue temperature below ______ °C is reached.
   a. 15
   b. 20
   c. 25
   d. 30

2. A 15-minute ice pack treatment has been shown to produce a maximum intramuscular cooling effect for up to ______ minutes.
   a. 10
   b. 20
   c. 30
   d. 40

3. A student athlete is being treated with ice for knee pain. What is the most appropriate timing of the ice application to maximize athletic performance based on the evidence?
   a. Treat with ice pack, which is removed 15 minutes before athletic participation.
   b. Treat with ice pack, which is removed immediately before athletic participation.
   c. Wear an ice pack during athletic participation.
   d. Treat with ice pack immediately following athletic participation.
1. Define cellular membrane potentials.

   All living cells are electrically charged or polarized, the inside of the cell being relatively negative in
   charge compared with the outside of the cell. The polarization is a result of the unequal distribution of
   ions on either side of the cell membrane. This polarity can be measured as a difference in electrical
   potential between the inside and the outside of the cell and is referred to as the membrane potential.
   A change in the membrane potential is referred to as an action potential and is the basis for the
   transmission of a nerve impulse. Nerve cells are specialized in detecting changes in their surroundings.
   If a change in their surroundings reaches a certain intensity or threshold, it can disturb the membrane’s
   resting state and trigger a nerve impulse.

   ![Diagram of Nerve Fiber](image)

2. Define refractory period.

   Immediately after a nerve impulse is triggered, an ordinary stimulus is not able to generate another
   impulse. This brief period is termed the refractory period. The refractory period consists of two phases—
   the absolute refractory period and the relative refractory period. The absolute refractory period lasts about
   1/2500 of a second and is followed by the relative refractory period. During the relative refractory
   period, a higher intensity stimulus can trigger an impulse.

3. What is saltatory, or jumping, conduction?

   Saltatory (ie, jumping) conduction of a nerve impulse occurs in myelinated nerve axons because
   myelin is an excellent insulator with a high resistance to current flow. Because myelin does not cover
   the nodes of Ranvier, current flows from one node of Ranvier to the next. The action potentials do not
   travel along the entire length of the axon; consequently, the nerve impulses can travel much faster in
   myelinated axons compared with unmyelinated axons. This jumping of nerve impulses also is much
   more efficient from a metabolic and physiologic standpoint. Fewer sodium and potassium ions are
   necessary to cross the cell membrane during the nerve impulse, and as a result, resting potentials
   are reestablished at a much faster rate, while conserving metabolic energy.
4. What are the average conduction velocities for myelinated and unmyelinated nerve fibers?
   - Myelinated: $\approx 130$ m/sec
   - Unmyelinated: $\approx 0.5$ m/sec

**PHYSICS OF ELECTRICAL FORCES**

5. What is an electrical current?
   Current—the natural drifting of ions that occurs within all matter—is defined as the directed flow of free electrons from one place to another. The unit of current is the ampere (A), which is the amount of electrical charge flowing past a specified circuit point per unit of time. The drifting is somewhat random and involves free electrons, positive ions, and negative ions.

6. Clinically, therapeutic intensities should not exceed what amperage?
   Typical therapeutic intensities should not exceed 80 to 100 mA.

7. What is electromotive force?
   The rate of current flow depends on a source of free electrons, positive ions, materials that allow the electrons to flow, and the electromotive force that concentrates electrons in one place. The volt (V) is the International System of Units measure of electrical potential and electromotive force, whereas voltage is the driving force of the electrons. One V is the electromotive force required to move 1 A of current through a resistance of 1 ohm ($\Omega$).

8. What role does voltage play in nerve cell membrane depolarization?
   For a nerve cell membrane to depolarize, an adequate number of electrons must be forced to move through conductive tissues. Given that likes repel and opposites attract, a high concentration of electrons flows to an area of low concentration. The greater the difference in concentration, the greater the potential for electron flow.

9. How does Ohm’s law express the relationship between current (I), voltage (V), and resistance (R)?
   \[ V = IR \]
   \[ I = V/R \]
   \[ R = V/I \]
   Therefore:
   - When resistance decreases, current increases
   - When resistance increases, current decreases
   - When voltage decreases, current decreases
   - When voltage increases, current increases
   - When voltage is zero, current is zero

10. What properties of a material tend to make it resist electrical currents?
    Conductors have low resistance, whereas insulators have high resistance. The actual resistance of a material is determined by the following formula:
    \[ R = P \times \text{Length of the material}/\text{Cross section}, \text{where } R = \text{resistance and } P = \text{resistivity} \]
    Therefore:
    - Greater cross-sectional area = decreased resistance
    - Greater temperature = decreased resistance = increased conductivity
    - Longer resistor = increased resistance

11. What factors typically alter skin impedance?

    | Increases Skin Impedance | Decreases Skin Impedance |
    |--------------------------|--------------------------|
    | Cooler skin temperature  | Increasing electrode surface |
    | Electrode type/surface factors | Removing excess hair |
    | Hair and oil present     | Warming skin |
    | Increased skin dryness   | Washing skin |
    | Increased skin thickness | |
12. What criteria are used to describe direct current (DC)?

DC is the flow of electrons in one direction for >1 second. A current is considered DC if it meets the following criteria:
- Flow of electrons is unidirectional
- Polarity is constant
- Current produces a twitch response only at the time of make (when the circuit is closed)
- Membrane is hyperpolarized as long as the current is on
- Duration of current flow is >1 second

13. Direct currents produce polar effects. What polar effects are produced by the anode and the cathode?

<table>
<thead>
<tr>
<th>Positive (Anode)</th>
<th>Negative (Cathode)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperpolarizes nerve fibers</td>
<td>Depolarizes nerve fibers</td>
</tr>
<tr>
<td>Repels bases</td>
<td>Attracts bases</td>
</tr>
<tr>
<td>Hardens tissues</td>
<td>Softens tissues</td>
</tr>
<tr>
<td>Stops hemorrhage</td>
<td>Increases hemorrhage</td>
</tr>
<tr>
<td>Sedates, calms</td>
<td>Stimulates</td>
</tr>
<tr>
<td>Reduces pain in acute situations</td>
<td>Reduces pain in chronic situations</td>
</tr>
</tbody>
</table>

14. What are the criteria used to describe alternating current (AC)?

AC is characterized by sine wave modulation and has a constantly fluctuating voltage and a symmetric pattern. A current is termed AC if it meets the following criteria:
- Magnitude of flow of electrons changes
- Direction of flow reverses
- No polar effects
15. List the typical frequencies (ranges of currents, if applicable) used in therapeutic applications.

<table>
<thead>
<tr>
<th>Frequency (Hz)</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Direct current</td>
</tr>
<tr>
<td>0–1000</td>
<td>Low frequency</td>
</tr>
<tr>
<td>1000–100,000</td>
<td>Medium frequency</td>
</tr>
<tr>
<td>&gt;100,000</td>
<td>High frequency</td>
</tr>
</tbody>
</table>

16. Does medium-frequency stimulation (MFS) differ from low-frequency stimulation in terms of skin resistance (capacitive impedance)?

Yes. When electrical current passes through cutaneous tissues, by surface electrodes, an opposition to the flow of current is encountered. When electrical currents are introduced into the body, ions accumulate at tissue interfaces, and cell membranes create a charge that opposes the applied voltage. This opposing voltage is referred to as reactance or capacitive impedance. The capacitive impedance can be calculated using the following formula:

\[
Z = \frac{1}{C(F) \times 2\pi \times f(\text{Hz})}
\]

where \( Z \) = capacitive impedance, \( C \) = polarization capacitance of tissues in farads (constant), and \( F \) = frequency of current. This formula shows that capacitance impedance decreases as the frequency increases.

17. Describe the key attributes of interferential currents.

With an interferential current (IFC), two separate current generators produce electrical currents that vary in relation to one another in amplitude or frequency, or both. Where these two distinct currents meet in the tissue, an electrical interference pattern is created based on the summation or the subtraction of the respective amplitudes or frequencies. With a sinusoidal wave pattern, when oscillations from two unlike frequencies or amplitudes are out of phase and blend (heterodyne), they produce the interference effect for which this modality was given its name. The typical depiction of the interference pattern is that which may be produced in homogeneous tissues, which would differ in human tissues. With IFC, the patient perceives the resulting signal or beat signal produced by the heterodyned alternating current as amplitude-modulated electrical pulses.
The beat signal often is described as being comparable to low-frequency pulse rates. For example, a 4100-Hz frequency and a 4200-Hz frequency could produce a constant beat frequency of 100 Hz. The phase duration of the delivered current can be easily calculated as follows:

\[
\text{Frequency} = \frac{1}{2 \times \text{Phase Duration}}
\]

**WAVEFORM CHARACTERISTICS**

18. Draw and label the following waveform characteristics: (1) pulse duration, (2) phase duration, and (3) amplitude.

19. What is the typical nomenclature and the appropriate units of measurement used to describe waveform characteristics?
   - Amplitude = intensity or millamp (mA)
   - Frequency = pulse rate, pulses per second (pps) or Hertz (Hz)
   - Phase duration = pulse width or microseconds (μs)

20. Discuss the practical and clinical implications for frequency, phase duration, and amplitude.
    Frequency contributes to the type of contraction, as well as theorized opiate-mediated effects:

<table>
<thead>
<tr>
<th>Frequency (Hz)</th>
<th>Nonfatiguing tetanic contraction</th>
<th>Fatiguing tetanic contraction</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–70</td>
<td>Released</td>
<td>β-Endorphins</td>
</tr>
<tr>
<td>100–1000</td>
<td>Enkephalins</td>
<td>Serotonin</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pulse Rate (Hz)</th>
<th>Released</th>
<th>Carryover</th>
</tr>
</thead>
<tbody>
<tr>
<td>40–150 (110–120)</td>
<td>Enkephalins</td>
<td>Short</td>
</tr>
<tr>
<td>15–100 (40–60)</td>
<td>Serotonin</td>
<td>Longer</td>
</tr>
<tr>
<td>1–4</td>
<td>β-Endorphins</td>
<td>Longest</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Type of Contraction</th>
<th>Released</th>
</tr>
</thead>
<tbody>
<tr>
<td>Twitch contraction</td>
<td></td>
</tr>
<tr>
<td>Tetanic contraction</td>
<td></td>
</tr>
</tbody>
</table>

Phase duration contributes to the comfort of the stimulation, the amount of chemical change that occurs in the tissues, and nerve discrimination. A duration of 50 to 100 μs typically is used for sensory stimulation, and 200 to 300 μs is typically used for motor stimulation.
Amplitude is best described by the following characteristics:
- Is less discriminatory than phase duration and pulse rate
- Greater intensity yields greater depths of penetration (generally speaking)
- Low intensity used for sensory stimulation
- High intensity used for motor stimulation

21. What is the clinical relevance of the pulse characteristics that are labeled in the diagram?
- Intrapulse interval—used to increase patient comfort
- Interpulse interval—needed to ensure the absolute refractory period
- Interburst interval—used with some protocols as a form of modulation

22. Define rise time, fall time, and duty cycle.
- Rise time—the time that it takes the wave to travel from zero to its peak amplitude
- Fall time—the time that it takes the wave to travel from its peak amplitude to zero
- Duty cycle—the relative proportion of time between the stimulation period and the rest period

23. Describe the key attributes of high-volt current and the unique characteristics of high-volt units.
High-volt galvanic currents are unique because they are not grouped with alternating or direct currents. The typical high-volt current stimulator produces a twin-peak monophasic waveform. Because the waveform is fixed and small in duration, two peaks are required to depolarize nerve cells. High-volt current stimulators are constant voltage units capable of delivering amplitudes $>100$ V. They also have a high peak current; however, the average current is only 50% of the peak current.

High-volt units typically have two electrode leads—one active and one dispersive—with the active electrode being much smaller than the dispersive electrode. A variety of hand applicators
and probes are available for the high-volt unit. A polarity switch typically is present and can be used to set the polarity of the active electrode.

![Depiction of high-volt current](image)

### 24. Discuss how high-volt currents differ from direct currents.

<table>
<thead>
<tr>
<th>High Voltage</th>
<th>Direct Current</th>
</tr>
</thead>
<tbody>
<tr>
<td>Used to excite peripheral nerves</td>
<td>Useless in exciting peripheral nerves</td>
</tr>
<tr>
<td>Useless in exciting denervated tissues</td>
<td>Used to excite denervated tissues</td>
</tr>
<tr>
<td>Creates minimal to no thermal reaction under electrodes</td>
<td>Creates thermal and chemical reactions under electrodes</td>
</tr>
<tr>
<td>Ineffective current for iontophoresis</td>
<td>Effective current for iontophoresis</td>
</tr>
<tr>
<td>Affects superficial and deep tissues</td>
<td>Affects superficial tissues</td>
</tr>
<tr>
<td>Useful in discriminating between sensory, motor, and painful stimulation</td>
<td>Discrimination is almost impossible and stimulation is usually uncomfortable</td>
</tr>
<tr>
<td>Used to resolve many clinical presentations and pathologies</td>
<td>Restricted benefit to a limited number of clinical presentations and pathologies</td>
</tr>
</tbody>
</table>

### ELECTRODES AND ELECTRODE PLACEMENT

25. What is the relationship between interelectrode distance and depth of penetration? Current travels through areas of least resistance; electrodes placed at greater distances from each other should be expected to provide deeper penetration, provided that all other treatment parameters and factors remain constant.

26. Name three common electrode placement strategies for neuromuscular electrical stimulation (NMES).
   1. Unipolar method: the active electrode is placed on the motor point, and the dispersive electrode is placed on some other point such as the nerve trunk.
   2. Bipolar method: two electrodes of equal size are placed along the length of the muscle belly. Usually the active electrode is placed over the motor point.
   3. Quadripolar method: four electrodes of equal size are used. This application is typically reserved for interferential currents in which two electrodes from each channel crisscross the treatment region.

### STIMULATION OF HEALTHY AND DENERVATED TISSUES

27. List electrically excitable and nonexcitable tissues.

<table>
<thead>
<tr>
<th>Excitable Tissues</th>
<th>Nonexcitable Tissues</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal organ cells</td>
<td>Bone</td>
</tr>
<tr>
<td>Autonomic motor fibers</td>
<td>Blood cells</td>
</tr>
<tr>
<td>Cardiac muscle fibers</td>
<td>Cartilage</td>
</tr>
<tr>
<td>Cells that produce glandular secretion</td>
<td>Collagen</td>
</tr>
<tr>
<td>Nerve axons of all types</td>
<td>Extracellular fluid</td>
</tr>
<tr>
<td>Nerve cells of all types</td>
<td>Ligaments</td>
</tr>
<tr>
<td>Voluntary motor fibers</td>
<td>Tendon</td>
</tr>
</tbody>
</table>

According to Pflüger’s law, healthy muscle contracts with less current if stimulated by the cathode compared with stimulation by the anode. When stimulating a muscle with a direct current, the cathode should be the active electrode because the amount of current required to acquire a muscle contraction is less with the active cathode than with the anode:

\[ CCC > ACC > AOC > COC \]

where \( CCC \) = cathode closing current, \( ACC \) = anode closing current, \( AOC \) = anode opening current, \( COC \) = cathode opening current, closing = starting the current, and opening = stopping the current.

29. What is accommodation?

Accommodation is the increased threshold of excitable tissue when a slowly rising stimulus is used. Both nerve and muscle tissues are capable of accommodating an electrical stimulus; nerve tissue accommodates more rapidly than muscle tissue. Understanding the process of accommodation is important when stimulating healthy muscle by the motor axon because the electrical stimulus must be applied somewhat rapidly to avoid accommodation.

30. What is the strength-duration curve?

The strength-duration curve describes the relationship between the strength of the stimulus (intensity) and the duration of the stimulus (on time) required to reach a specified level of activation. By varying the intensity and duration of an electrical stimulus, it is possible to plot a strength-duration curve. The strength-duration curve gives a graphic representation of the excitability of nerve and muscle tissues. Although the strength-duration curves are comparable for healthy nerve and muscle tissues, they are different from denervated nerve and muscle tissues. As a result, we are clinically able to stimulate healthy, innervated muscles with a stimulus of adequate amplitude and of short duration. It also is shown by this curve that greater amplitudes of stimulus and longer durations are necessary to stimulate denervated muscles effectively.

---

**TEST**

<table>
<thead>
<tr>
<th>NORMAL</th>
<th>DENERVATING</th>
<th>DENERVATED</th>
<th>REINNERVATING</th>
<th>REINNERVATED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronaxy</td>
<td>&lt; 1 msec</td>
<td>begins to rise</td>
<td>30 to 50 msec</td>
<td>begins to decrease approaches normal</td>
</tr>
<tr>
<td>Strength Duration Curve</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reaction of Degeneration</td>
<td>AC = DC</td>
<td>DC &gt; AC</td>
<td>DC only</td>
<td>AC begins</td>
</tr>
<tr>
<td>Nerve Conduction</td>
<td>40 to 60 m/sec</td>
<td>No conduction after 3 days</td>
<td>No conduction</td>
<td>Conduction increases</td>
</tr>
</tbody>
</table>
31. What are common contraindications and precautions for electrotherapy application?

**CONTRAINDICATIONS (ABSOLUTE AND RELATIVE)**
- Cardiac pacemaker of synchronous or demand type
- Patients prone to seizures
- Placement of electrodes across or around the heart
- Placement of electrodes over a pregnant uterus, especially during the first trimester (this is controversial, and delivery itself presents with relative precautions)
- Placement of electrodes over an area suspected of arterial or venous thrombosis or thrombophlebitis
- Placement of electrodes over the pharyngeal area
- Placement of electrodes over protruding metal
- Placement of electrodes over the carotid sinus

**PRECAUTIONS**
- Allergies to tapes and gels
- Areas of absent or decreased sensation
- Electrically sensitive patients
- Patients with cardiac disease
- Patients with severe hypotension or hypertension
- Placement of the electrode over an area with significant adipose tissue
- Placement of the electrode over damaged skin (with the exception of certain tissue healing protocols)
- Placement of the electrode over or near the stellate ganglion
- Placement of the electrode over the temporal and orbital region
- Patients who are unable to communicate clearly

**APPLICATION**

32. List common indications for electrical stimulation.
- Edema management
- Maintaining and improving range of motion
- Muscle strengthening
- Neuromuscular facilitation and reeducation
- Orthotic substitution
- Pain management
- Reduction of muscle spasm
- Temporary reduction of spasticity

33. Is there a difference between the use of NMES or voluntary exercise or the use of combined NMES and voluntary exercise in terms of muscle strength?
Yes. There is evidence showing that NMES combined with voluntary exercise may accelerate gains in quadriceps muscle strength and activation greater than voluntary exercise alone after total knee arthroplasty. For example, Stevens and colleagues showed that the addition of ten 10-second NMES-elicited quadriceps contractions to treatment sessions significantly improved quadriceps strength, with the most dramatic improvement noted in the first 3 weeks of treatment.

34. Outline a suitable protocol for neuromuscular facilitation and reeducation including purpose, rationale, indications, parameters, and special considerations.
1. **Purpose**—To barrage the central nervous system (CNS) with appropriate sensory information
2. **Rationale**—By supplying the proper sensory input of what a muscle contraction or limb movement feels like, and visual information about the appearance of the action, electrical stimulation can enhance a motor response.
3. **Indications**—Any patient for whom a motor- and sensory-augmented muscle response would assist in better performance of his or her own voluntary actions
4. **Parameters**—Pulse duration, 100 to 200 μs; pulse rate, 35 to 50 Hz; intensity, to a tolerable motor level up to 3+/5; ramp, 1 to 3 sec up/down; on/off, 1:1 ratio set or hand-held switch; treatment time, 5 to 30 min, 1 to 3 times/day, 3 to 7 days/week, 1 to 2 weeks
5. **Special considerations**—Facilitation and reeducation require active participation by the patient and may be limited by patient tolerance, cooperation, and attention span.
35. When is NMES indicated after knee surgery and immobilization?
- Prevention of muscle atrophy associated with prolonged immobilization
- Prevention of decreases in muscle strength
- Prevention of decreases in muscle mass

36. Is there a difference between the use of high-intensity electrical stimulators and low-intensity or battery-powered stimulators with regard to quadriceps femoris muscle force production in the early phases of anterior cruciate ligament (ACL) rehabilitation?
Yes. Studies more frequently support the use of high-intensity electrical stimulation but do not consistently support the use of low-intensity or battery-powered stimulation when the desired objective is the recovery of quadriceps femoris muscle force production. For example, a study by Snyder-Mackler and colleagues found that training contraction intensity is positively correlated with quadriceps femoris muscle recovery, with an apparent threshold training contraction intensity of 10% of the maximal voluntary contraction of the uninvolved quadriceps femoris muscle.

37. Outline a suitable protocol for muscle strengthening in terms of purpose, rationale, indications, parameters, and special considerations.
1. Purpose—to increase muscle strength, encourage muscle hypertrophy, and facilitate normal motor response
2. Rationale—electrical stimulation can be used to help patients achieve a volitional contraction sufficient to increase strength and prevent disuse atrophy if they are unable to do so on their own.
3. Indications—any patient in need of increasing girth and strength of an atrophied muscle
4. Parameters—pulse duration, 200 to 300 μs; pulse rate, 35 to 80 Hz; intensity, motor, 60% ± maximal voluntary contraction (MVC); ramp, 1 to 5 sec up/down, as tolerated; on/off, 1:5 ratio; treatment time, activity specific, 10 to 20 repetitions, 3 to 5 days/week; 2 to 3 weeks
5. Special considerations—this program should be used with patients with sufficient innervation to make muscle strengthening practical. It is important to avoid muscle fatigue with this type of stimulation.

38. Is microcurrent electrical nerve stimulation (MENS) effective in the treatment of temporomandibular joint pain?
No. There is no clear evidence supporting the use of MENS in the treatment of temporomandibular joint pain; further research is necessary.

39. What are the benefits and limitations of NMES after ACL reconstruction?
Benefits identified in literature include reduced postsurgical muscle atrophy, increased muscle torque values, and significantly greater strength gains in patients receiving NMES and exercise, compared with exercise alone. However, studies investigating the long-term benefits of NMES, as well as the effect of NMES on functional outcomes, are inconsistent.

40. Is NMES more effective for strength training after ACL reconstruction when performed against isometric resistance?
Yes. There is evidence showing that the strength training effect is decreased when NMES is applied without isometric resistance. However, use of NMES without resistance is considered to be an acceptable alternative when clinicians do not have access to a dynamometer or for patients who do not tolerate NMES-induced contractions against isometric resistance.

41. Should the presence or absence of a knee extensor lag be a criterion for using or not using NMES after ACL reconstruction?
No. No strong relationship has been found between knee extensor lag and treatment outcomes after use of NMES. Data indicate that NMES is beneficial regardless of whether or not an extensor lag is present.

42. Is there a relationship between the number of NMES training sessions per week and strength outcomes?
Yes. For example, three training sessions per week for 4 weeks have been shown to be effective for strength gains compared with two training sessions per week for 4 weeks.

43. Is NMES effective in the treatment of elderly patients with knee osteoarthritis?
Yes. There is evidence supporting the use of NMES for improving voluntary muscle activation and quadriceps muscle strength. However, the benefits of using NMES for improving functional outcomes in this patient population are inconsistent.
44. Is there a relationship between muscle contraction strength or fatigue and type of waveform used with electrical stimulation?

Yes. There is evidence showing that monophasic and biphasic waveforms generate greater torque and are less fatiguing than polyphasic waveforms.

45. What are the suitable parameters and rationale for conventional, low-rate, and brief intense transcutaneous electrical nerve stimulation (TENS)?

<table>
<thead>
<tr>
<th></th>
<th>Conventional</th>
<th>Low Rate</th>
<th>Brief Intense</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indications</td>
<td>Acute, superficial pain, and/or first time application</td>
<td>Subacute to chronic pain</td>
<td>Before or in conjunction with other interventions</td>
</tr>
<tr>
<td>Phase duration</td>
<td>60–100 µs</td>
<td>200–400 µs</td>
<td>&gt;250 µs</td>
</tr>
<tr>
<td>Pulse rate</td>
<td>80–125 Hz</td>
<td>1–4 Hz</td>
<td>125 Hz (can be varied)</td>
</tr>
<tr>
<td>Intensity</td>
<td>Sensory just below motor</td>
<td>High sensory to slight twitch contraction</td>
<td>High sensory to tolerance to twitch contraction</td>
</tr>
<tr>
<td>Treatment duration</td>
<td>15–30 minutes</td>
<td>30–45 min</td>
<td>10–15 min</td>
</tr>
<tr>
<td>Onset of relief</td>
<td>10–20 min</td>
<td>20–40 min</td>
<td>1–10 min</td>
</tr>
<tr>
<td>Carryover</td>
<td>30 min to 2 hours</td>
<td>Several hours to a day</td>
<td>Short (&lt;30 min)</td>
</tr>
<tr>
<td>Rationale</td>
<td>Gate theory</td>
<td>Gate theory</td>
<td>Gate theory</td>
</tr>
<tr>
<td></td>
<td>Opiate mediated</td>
<td>Opiate mediated</td>
<td>Opiate mediated</td>
</tr>
<tr>
<td></td>
<td>Counterirritant</td>
<td>Counterirritant</td>
<td>Counterirritant</td>
</tr>
<tr>
<td></td>
<td>Placebo</td>
<td>Placebo</td>
<td>Placebo</td>
</tr>
</tbody>
</table>

46. Are there differences between the effects of low-frequency transcutaneous electrical nerve stimulation (TENS) and interferential current (IFC) in the management of pain?

Yes. Decreased skin resistance encountered by IFC makes it more effective for the delivery of total current to tissues, as well as for the treatment of deeper and larger treatment areas, compared with TENS. Quadrupolar (vector-field) IFC application should also be more effective for targeting and treating deeper tissues.

47. Discuss important considerations and treatment parameters for maintaining joint range of motion.

Protocols should typically begin with simple one-plane joint movements, use antigravity starting positions with a rest between movements, and progress to antigravity positions without a rest between movements (ie, flexion-rest-extension-rest > flexion-extension-flexion) as tolerated. Reasonable treatment parameters are as follows: intensity, to a tolerable motor level up to 3 +/5; frequency, 35 to 50 Hz; phase duration, 100 to 200 µs; ramp, 4 to 5 sec progressing to 3 sec; on/off, as required to achieve desired range of motion; treatment time, 30 min/day, 50 to 100 repetitions, as needed.

48. Discuss key considerations and treatment parameters for edema control.

Muscular activity is an important aspect of lymphatic and venous flow. The contraction of skeletal muscles by electrical stimulation can produce a muscle contraction capable of aiding lymphatic and venous flow. The intervention can be enhanced further by combining it with other forms of management, such as elevation, cryotherapy, rest, and compression. Muscle pumping protocols are valuable for pain modulation. Reasonable stimulation parameters should focus on producing a nonfatiguing muscle contraction: pulse rate, 4 to 10 Hz; phase duration, 300 µs; waveform, biphasic or high volt; polarity, not applicable with this protocol; intensity, visible contraction of muscles in the area where edema is noted, 1/5 to 3/5; time of treatment, 30 min, two to three times per day as part of a home program; 1 to 2 weeks; electrode placement, muscle bulk of an involved muscle, region, and/or an involved joint. This protocol is typically used in conjunction with cryotherapy and elevation of the affected area.

49. Can electromyographic biofeedback aid in the recovery of quadriceps femoris muscle function after ACL reconstruction?

Yes. For example, findings from studies by Draper and Ballard suggest that (1) biofeedback is more effective than electrical stimulation in promoting recovery of peak torque, (2) biofeedback and electrical stimulation are comparable in terms of recovery of active knee extension, and (3) biofeedback combined with muscle strengthening exercises facilitates a more rapid recovery of quadriceps femoris peak torque after ACL reconstruction compared with electrical stimulation alone.
Pthomegroup

ELECTROTHERAPY

BIBLIOGRAPHY


**CHAPTER 9 QUESTIONS**

1. An otherwise healthy 66-year-old man is referred to an outpatient physical therapy clinic after being diagnosed by his doctor as having osteoarthritis of the right knee. His major symptoms are muscle weakness and pain with ambulation. Which of the following is the most appropriate rationale for using neuromuscular electrical stimulation (NMES) for muscle strengthening with this patient?
   a. To improve activities of daily living and for long-term functional outcomes
   b. To improve quadriceps femoris metabolism and blood flow
   c. To improve quadriceps femoris peak torque values
   d. To improve voluntary muscle activation and quadriceps muscle strength

2. A 24-year-old female visits a physical therapist 5 days after ACL reconstruction. Which of the following are the most appropriate parameters for her first electrical stimulation intervention?
   a. Amplitude = motor (tolerable tetanic), frequency = 50 pps, and phase duration = 200 μs
   b. Amplitude = motor (twitch contraction), frequency = 10 pps, and phase duration = 50 μs
   c. Amplitude = motor (60% MVC), frequency = 80 pps, and phase duration = 400 μs
   d. Amplitude = motor (80% MVC), frequency = 100 pps, and phase duration = 300 μs

3. Select the maximum voluntary isometric contraction (MVIC) that is necessary for strengthening healthy muscle tissue.
   a. 10% of MVIC
   b. 25% of MVIC
   c. 50% of MVIC
   d. 70% of MVIC
IONTOPHORESIS, ULTRASOUND, PHONOPHORESIS, AND LASER AND LIGHT THERAPY

F.D. Pociask, PT, PhD, OCS, and T.M. Fleck, MPT, OCS

CHAPTER 10

IONTOPHORESIS

1. Are iontophoresis and phonophoresis interchangeable clinically?

No. Ions are introduced with iontophoresis, whereas molecules are introduced by the ultrasound waves. Furthermore, because sound waves are not electrical in nature, no ionization takes place.

2. Describe Leduc’s classic experiment.

In 1908 Leduc showed that ionic medication could penetrate intact skin and produce local and systemic effects in animals. Two rabbits were placed in series in the same direct current circuit so that the current had to pass through both rabbits to complete the circuit. The electrical current entered into the first rabbit by a positive electrode soaked in strychnine sulfate and exited the rabbit by a negative electrode soaked in water. The current then entered the second rabbit by an anode soaked in water and exited by a cathode soaked in potassium cyanide. When a current of 40 to 50 mA was used, the first rabbit exhibited tetanic convulsions secondary to the introduction of the strychnine ion, and the second rabbit died quickly, secondary to cyanide poisoning. When the animals were replaced and the flow of current was reversed, they were not harmed because the strychnine ion was not repelled by the positive pole and the cyanide was not repelled by the negative pole.

3. Describe the potato experiment.

Two electrodes were implanted at opposite ends of a potato, and a potassium iodine solution was placed in a depression that was made in the central-top portion of the potato. Direct current (DC) attracted the iodine anion toward the positive pole, and the free iodine formed blue starch iodine.

4. Define direct current.

Direct current is the flow of electrons in one direction for >1 second. A current is termed a direct current if:

- The flow of electrons is unidirectional
- The polarity is constant
- The current produces a twitch response only at the time of make
- The membrane is hyperpolarized as long as the current is on
- The duration of current flow is >1 second

With iontophoresis, the current is on for the duration of the treatment.

5. List some commonly used ionic solutions and their proposed indications.

<table>
<thead>
<tr>
<th>Ionic Solution</th>
<th>Indications</th>
<th>Polarity</th>
<th>Solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetic acid</td>
<td>Calcium deposits</td>
<td>Negative</td>
<td>2%–5%</td>
</tr>
<tr>
<td>Dexamethasone sodium phosphate</td>
<td>Inflammatory conditions</td>
<td>Negative</td>
<td>4 mg/mL</td>
</tr>
<tr>
<td>Lidocaine hydrochloride</td>
<td>Skin anesthesia</td>
<td>Positive</td>
<td>4%–5%</td>
</tr>
<tr>
<td>Potassium iodide</td>
<td>Scar tissue</td>
<td>Negative</td>
<td>5%–10%</td>
</tr>
<tr>
<td>Water</td>
<td>Hyperhidrosis</td>
<td>Alternate</td>
<td>100%</td>
</tr>
<tr>
<td>Zinc oxide</td>
<td>Ulcers, antiseptic</td>
<td>Positive</td>
<td>20%</td>
</tr>
</tbody>
</table>
6. Why are the effects of iontophoresis often longer lasting than those of phonophoresis?
Ions are introduced into the superficial tissues, where circulation is limited, giving the ions time to be absorbed and used. Phonophoretically introduced molecules are delivered to deeper layers, where vascularization is more abundant, leading to early transport out of the area before effective breakdown and reuse are possible.

7. Does increasing the concentration of the drug increase the amount delivered to the target tissue?
No. Although concentrations vary based on the applied ion (eg, dexamethasone sodium phosphate [DexNa2PO3] at 0.04% and sodium salicylate [NaSal] at 2%), higher concentrations have not been shown to be more effective.

8. Are there concerns with using direct current?
Yes. Intact skin cannot tolerate a current density >1 mA/cm².

9. Ion transfer depends on what factors?
- The concentration of ions in solution
- The current density of the active electrode
- The duration of current flow

10. Why do burns occur with iontophoresis?
Most burns are caused by poor technique, which can be greatly negated by the use of quality, commercial products. Some common to less common conditions that can produce burns are the following:
- Poor skin-electrode interfaces
- Improperly sized or placed electrodes
- Intensity too high
- Electrodes too small or too dry
- Improper size differential or placement distance between anode and cathode
- Wrong polarity
- Velcro straps or adhesive tapes too tight
- Use of current other than continuous DC

11. Where should the iontophoresis electrodes be placed?
The active electrode containing the ion that is to be repelled is placed over the treatment tissues, and the depressive electrode is placed about 18 inches away to encourage a greater depth of penetration. Because electrodes and units typically come with specific instructions, it is wise to read both sets of instructions before attempting the procedure.

12. What are advantages of iontophoresis compared with injection?
- No carrier fluids required
- Reduced risk of infection secondary to noninvasive application
- Relatively painless for most patients
- Able to deliver antiinflammatory medication locally without the gastrointestinal side effects associated with oral ingestion or the systemic effects noted with injection

13. What are the disadvantages of iontophoresis?
- Numerous treatments may be required to obtain results.
- Depth of penetration is limited to approximately 8 to 10 mm (depth of penetration has been reported at up to 2 cm).
- Electrodes are costly.
- Risks of polar effects and skin damage
- Setup and application are time consuming.

14. Is corticosteroid delivery by iontophoresis an effective treatment for lateral epicondylitis?
Research suggests that iontophoresis is an effective alternative to local injection of dexamethasone for patients with lateral epicondylitis with the potential for fewer side effects.

15. What is the preferred treatment duration for iontophoresis in the treatment of medial or lateral epicondylitis?
Research suggests that administration of dexamethasone sodium phosphate for six treatments over 10 days or less has had better results than administration over a longer period.
16. How many serial iontophoresis treatments are safe?
   One to six treatments of dexamethasone are considered safe when administered alone.

17. Does the magnitude of iontophoresis current determine the depth of penetration?
   No. Evidence suggests that diffusion, rather than magnitude of current, determines depth of drug penetration. Comparable doses delivered at low-magnitude currents over several hours may be more effective than those delivered by higher magnitude currents for 10 to 30 minutes.

18. Do buffered electrodes stabilize skin pH under the cathode?
   The literature suggests that when iontophoresis is properly delivered at 20 to 40 mA/min, pH changes with or without a buffer are not significantly different. In contrast, when iontophoresis is delivered at 80 mA/min, significant changes in pH are stabilized by the addition of buffers.

19. Is there evidence showing the benefits of iontophoresis with specific pathologies?
   Studies have conflicting findings regarding the benefits of iontophoresis. There is limited support for its use with acute epicondylitis, TMJ, carpal tunnel syndrome, Achilles tendinitis, and plantar fasciitis using dexamethasone sodium phosphate. The use of iontophoresis for adhesive capsulitis and calcific tendinitis of the shoulder is not supported at this time.

ULTRASOUND

20. How is ultrasound generated, and what is a piezoelectric effect?
   The natural quartz or synthetic crystal housed within the sound head, classified as a piezoelectric material, will mechanically respond or deform when subjected to alternating current (AC) by expanding and contracting at the same frequency at which the current changes polarity. When the crystal expands, it compresses the material in front of it, and when it contracts, it rarefies the material in front of it. This process is described as a piezoelectric effect.

21. What is the beam nonuniformity ratio (BNR)?
   BNR is the measure of the variability of the ultrasound wave intensity produced by the machine. If the machine is set at 1.5 W, BNR is the range of possible intensities actually delivered by the machine. The lower the ratio, the more uniform the machine output, resulting in a more uniform treatment. A higher ratio, 8 W, for example, means that when the machine is set at 1 W, it could deliver in the range of 1 to 8 W.

22. What is the effective radiating area (ERA) of a transducer?
   ERA is the effective radiating area that corresponds to the part of the sound head that produces the sound wave. The ERA should be close to the size of the sound head or transducer. If it is smaller than the sound head, it may be misleading when treating the patient. The recommended treatment area is only two to three times the ERA.

23. What are nonthermal and thermal ranges of therapeutic ultrasound?
   Intensities between 0.1 and 0.3 W/cm² are considered nonthermal, and intensities above approximately 0.3 W/cm² are considered thermal.

24. What are the reported and theorized nonthermal effects of ultrasound?
   - Increased cell membrane permeability
   - Increased vascular permeability
   - Increased blood flow in chronically ischemic tissue
   - Collagen synthesis
   - Phagocytosis
   - Promotes tissue regeneration
   - Breaks down scar tissue in acute injuries
   - Kills bacteria and viruses in chronic situations

25. What are the reported and theorized thermal effects of ultrasound?
   - Preferentially heats collagen-rich tissues
   - Increased tissue elasticity of collagen-rich tissue
   - Increased blood flow
   - Increased pain threshold
   - Decreased muscle spasm
   - Decreased pain and joint stiffness
   - Mild inflammatory response
26. How does ultrasound frequency relate to depth of penetration?
Increasing the frequency of ultrasound causes a decrease in its depth of penetration and concentration of the ultrasound energy in the superficial tissues. For example, the approximate depth of penetration at 1 MHz is 2 to 5 cm, and the approximate depth of penetration at 3 MHz is 1 to 2 cm.

27. Does the ultrasound transducer speed affect the intramuscular tissue temperature?
It has been reported that the speed of the ultrasound transducer over the treatment area does not affect the underlying tissue temperature as long as the ultrasound treatment is applied within a set treatment area of two times the size of the transducer. Speeds of 2 to 3, 4 to 5, and 7 to 8 cm/sec were studied.

28. Will tissue temperature increases in human muscle vary between pulsed and continuous ultrasound application when administered at equivalent temporal average intensities?
The literature suggests that equivalent temporal average intensities will produce similar increases in intramuscular tissue temperature. For example, 3 MHz at a 50% duty cycle and an intensity of 1.0 W/cm² over a 10-minute period produced similar heating compared with 3 MHz at a 100% duty cycle and an intensity of 0.5 W/cm² over a 10-minute treatment.

29. Is a metal implant an absolute contraindication for the use of ultrasound?
No. However, caution should be exercised because ultrasound is contraindicated over plastic implants and joint cement, which are often components of a total joint replacement.

30. Is ultrasound effective in treating calcific tendinitis of the shoulder?
Yes. It has been suggested that ultrasound treatment helps resolve calcifications and is associated with short-term improvements in pain and quality of life. In a study by Ebenbichler and colleagues, patients received 24 15-minute sessions of 25% pulsed ultrasound (0.89 MHz at 2.5 W/cm²) over a 6-week period. After 6 weeks of treatment, calcifications resolved in 19% of patients and decreased by at least 50% in 28% of patients (compared with 0 and 10% in those receiving sham ultrasound). At the 9-month follow-up, calcifications resolved in 42% of patients and improved in 23% of patients receiving ultrasound (compared with 8% and 12% in those receiving sham ultrasound).

31. Is there evidence supporting the use of static ultrasound application over conventional ultrasound application?
Studies have shown that the use of high power pain threshold ultrasound over myofascial trigger points is effective at improving pain level, pressure pain threshold, and neck pain disability scores. A study by Majlesi and Unalan in 2004 specifically showed improvements in pain and active cervical spine ROM with this static technique applied to patients with acute upper trapezius trigger points.

32. Is there evidence supporting the use of low-intensity pulsed ultrasound?
Research suggests that low-intensity pulsed ultrasound is effective in promoting tendon healing and reducing inflammation (ie, acute tendinitis). Animal models have also shown that this technique may attenuate the progression of cartilage degeneration and increase articular cartilage formation in arthritic conditions. More research is needed to support these effects in humans.

33. Is ultrasound effective in treating carpal tunnel syndrome?
A study by Ebenbichler and colleagues suggests that ultrasound may be effective in reducing pain and improving electromyographic variables (motor distal latency and nerve conduction velocity) in patients with carpal tunnel syndrome. In this study, 20 sessions of ultrasound treatment were performed over a 6-week period (1 MHz, 1.0 W/cm², pulsed mode 1:4, 15 minutes per session).

34. Is there sufficient support for the use of ultrasound in a physical therapy treatment program?
Based on a review of randomized controlled trials (RCTs) published between 1975 and 1999 in which ultrasound was used for patient treatment, it was suggested that there is little evidence to support the use of active therapeutic ultrasound versus placebo ultrasound. It was also noted that 25 out of the 35 studies reviewed were methodologically inaccurate, and the 10 remaining studies had significant variability in dosages used and patient problems treated; further research is required to answer this question.
ULTRASOUND AND PHONOPHORESIS

35. How does phonophoresis work?
It was once thought that ultrasound exerted pressure on the drug, driving it through the skin. However, ultrasound exerts only minimal pressure. Another explanation is that ultrasound changes the permeability of the stratum corneum (the most superficial skin layer) through thermal and nonthermal effects. Ultrasound performed before the application of a drug to the skin has been found to increase drug penetration, supporting this theory.

36. When performing phonophoresis, what dosage is preferred?
In several animal studies Griffin and colleagues demonstrated that ultrasound allowed cortisone to penetrate paravertebral muscles and nerves under a variety of treatment dosages (eg, 1.0 W/cm² for 5 minutes, 3.0 W/cm² for 5 minutes, 0.3 W/cm² for 17 minutes, and 0.1 W/cm² for 51 minutes, with frequencies that ranged from 0.09–3.6 MHz). Griffin’s work demonstrated the greatest penetration with higher intensities at shorter durations and with lower intensities at longer durations. Results favored lower intensities at longer durations in terms of greatest delivery of cortisone to muscles and nerves. Clinically, modest intensities at longer durations using a nonstationary sound mode of application within carefully constrained areas of treatment are recommended for patient comfort and to prevent tissue damage.

37. When performing phonophoresis, what concentrations of hydrocortisone are most effective?
A study by Kleinkort and Wood suggests that treatments using 10% hydrocortisone are more effective than those using 1% hydrocortisone for relieving pain associated with tendinitis or bursitis.

38. How many serial phonophoresis treatments are safe?
Once a drug passes through the skin, it is circulated through the body and can become systemic; this is also true in the case of phonophoresis.

39. What are examples of drugs that can be administered by phonophoresis?
The following drugs have been identified as phonophoretic agents: dexamethasone (0.4% ointment), hydrocortisone (0.5%–1.0% ointment), iodine (10% ointment), lidocaine (5% ointment), magnesium sulfate (2% ointment), salicylates (10% trolamine salicylate or 3% sodium salicylate ointment), and zinc oxide (20% ointment).

40. Provide an example of a topical nonsteroidal antiinflammatory drug (NSAID) that may be administered by phonophoresis.
Fastum gel (ketoprofen 2.5%) has been shown to be an effective phonophoretic agent. Phonophoretic application of this drug appears to be superior to topical application. In a study by Cagnie and colleagues, the concentration of ketoprofen in synovial tissue was significantly greater in the groups receiving phonophoresis with either continuous (1 MHz at 1.5 W/cm²) or pulsed (20%) ultrasound than in the group receiving only topical application.

41. What is the most efficiently transmitted topical antiinflammatory media used in phonophoresis?
Fluocinonide 0.05% (Lidex) gel and methyl salicylate 15% (Thera-Gesic) cream transmit ultrasound the best—97% relative to water.

42. Is phonophoresis effective in treating lateral epicondylitis?
A study by Baskurt and colleagues suggests that phonophoresis of naproxen (10%) may be equally as effective as iontophoresis of naproxen (10%) in reducing pain and improving grip strength in patients with lateral epicondylitis.

LASER AND LIGHT THERAPY

43. What is laser and light therapy?
Laser (ie, light amplification by stimulated emission of radiation) and light therapy is electromagnetic energy that falls within or close to the visible range of the electrometric spectrum (eg, typically between 600 and 1300 nm). More specifically, laser and light therapy uses laser diodes, light emitting
diodes (LEDs), and super-luminous diodes (SLDs) to treat human tissues at various depths of penetration. Most present-day applicators include a combination of laser, LED, and SLD photodiodes in order to offer a broader array of treatment options.

In terms of key differences, laser diodes produce directional light composed of a single wavelength that is most suitable for treating small areas at various depths of penetration as determined by the wavelength of the applicator. LEDs produce diffuse light most suitable for treating larger areas and superficial tissues, and SLDs produce light that is most suitable for treating intermediate sized areas and superficial to moderately deep tissues as determined by the wavelength of the applicator.

44. What is the relationship between wavelength and depth of penetration?
Light with longer wavelengths will penetrate deeper than light with shorter wavelengths. For example, near infrared (700 and 1100 nm) is more suitable for deeper tissues (3–4 cm deep), and red light (600 and 700 nm) is more suitable for treating superficial tissues (0.5–1 cm deep).

45. What are the proposed benefits, strengths, and limitations of laser and light therapy?
Proposed beneficial effects of laser and light therapy include promotion of ATP production, collagen synthesis, microcirculation, and tissue healing (ie, including, reduced wound healing times), pain reduction, and inhibition of bacterial growth across a variety of conditions (eg, osteoarthritis, carpal tunnel syndrome, chronic low back pain, radiculopathy, chronic Achilles tendinopathy, and diabetic peripheral neuropathy).

Notwithstanding a number of studies reporting positive, mixed, and negative effects, laser and light therapy remain controversial in mainstream physical therapy practice and medicine, as the mechanisms underlying identified positive effects are not entirely understood. Equally, rationale for selecting treatment parameters and dosage (eg, type of diode or diodes, wavelength, power density, pulse structure, treatment time, and treatment duration) lack agreement; further research is needed.

46. What is multiwave locked system (MLS) laser therapy?
MLS laser therapy combines continuous and pulsed laser light emissions in order to act on multiple cellular sites and different cellular complexes at the same time. Continuous emissions are said to promote ATP production and microcirculation and modify the inflammatory response. Pulsed emissions are said to reduce pain by affecting superficial nociceptors and afferent nerve fibers. As with more traditional laser, LED, and SLD modalities, the underlying effects on human tissues are not entirely clear, research findings are mixed, and further inquiry is needed.

47. Is the use of MLS laser therapy supported by the literature?
A very limited number of studies have reported positive MLS laser outcomes. For example, a study by Kedzierski et al. in 2012 showed better analgesic effects using MLS laser versus low-frequency TENS in patients with knee osteoarthritis. At present, more evidence is required to recommend the use of MLS laser therapy as an effective therapeutic modality.

BIBLIOGRAPHY
1. Which of the following statements regarding iontophoresis is true?

a. Increasing the drug concentration increases the amount delivered to the tissue.

b. The active and passive electrodes should be placed about 8 inches apart.
c. The depth of penetration is limited to approximately 8 to 10 mm.
d. The magnitude of iontophoresis current determines the depth of penetration.

2. Which of the following ultrasound parameters should be administered to achieve thermal effects in superficial tissues?
   a. Frequency = 1.0 MHz, Intensity = 0.3 w/cm², Treatment area = two times the ERA
   b. Frequency = 1.0 MHz, Intensity = 1.0 w/cm², Treatment area = three times the ERA
   c. Frequency = 3.0 MHz, Intensity = 0.5 w/cm², Treatment area = four times the ERA
   d. Frequency = 3.0 MHz, Intensity = 1.5 w/cm², Treatment area = two times the ERA

3. The effective radiating area of the sound head is:
   a. Equal to the frequency of the sound head
   b. Larger than the size of the sound head
   c. Smaller than the size of the sound head
   d. The same size as the sound head
1. What is stress relaxation?
Stress relaxation is a physical property of viscoelastic structures, such as a muscle tendon unit (MTU). If an MTU is elongated to a specific length and held in that position, the internal tension within the MTU decreases with the passage of time. Clinically, this is what occurs during a static stretch of an MTU.

2. Define creep.
Creep occurs when an MTU is elongated to a specific length and then allowed to continue to elongate as stress relaxation occurs. Clinically, this is what occurs when a therapist performs a stretch in which joint range is increased during the stretch repetition. Creep is partially responsible for the immediate increase in joint range of motion (ROM) during a stretch repetition.

3. When stretching a muscle joint complex, what structures are influenced?
- Joint capsule
- Ligaments
- Nerves
- Vessels
- Skin
- MTU

4. What is ballistic (dynamic) stretching?
Ballistic stretching places the muscle joint complex at or near its limit of available motion and then cyclically loads the muscle joint complex (bouncing motion at the end ROM). The rate and amplitude of the stretch are variable. Ballistic muscle stretching is indicated for preconditioning a muscle joint complex for activities such as sprinting, high jump, or other events that depend on the elastic energy in an MTU to enhance the performance of a particular movement pattern.

5. Define static stretching.
Static stretching is a technique that places a muscle joint complex in a specific ROM until a stretch is perceived. The position is held for a specific period of time and repeated as necessary to increase joint ROM.

6. Describe some commonly used proprioceptive neuromuscular facilitation (or active inhibition) stretching techniques.
- Hold-relax—the muscle to be stretched is placed in a lengthened but comfortable starting position. The patient is instructed to contract the target muscle for approximately 5 to 10 seconds. After the 10-second contraction, the patient is instructed to relax the target muscle completely as the therapist passively increases joint ROM. This is repeated for a specific number of repetitions. Intensity of the stretch is limited by the patient.
- Hold-relax-antagonist contraction—the muscle to be stretched is placed in a lengthened but comfortable starting position. The patient is instructed to contract the target muscle for approximately 5 to 10 seconds. After the 10-second contraction, the patient is instructed to relax and then contract the muscle opposite (reciprocally inhibiting the target muscle) the target muscle, actively increasing joint ROM. Intensity of the stretch is limited by the patient.
- Antagonist contraction—the muscle to be stretched is placed in a lengthened but comfortable starting position. The patient is instructed to contract the muscle opposite (reciprocally inhibiting the target muscle) the target muscle, actively increasing joint ROM. Intensity of the stretch is limited by the patient.
7. **What is the optimal number of static stretch repetitions?**
   The optimal number of stretch repetitions is four. Boyce and Brosky in 2008 found that passive stretching beyond five repetitions results in insignificant gains in hamstring length and that the greatest increase in range of motion occurs during the first stretch repetition.

8. **What is the optimal amount of time that a static stretch should be held?**
   The literature reports stretching durations from 6 seconds to 60 minutes. When looking at immediate increases in range of motion, the literature recommends (on average) stretch times between 15 and 60 seconds. Overwhelmingly the literature reports that prolonged stretching times impair performance. Additionally, it has been found that shorter stretch times <30 seconds result in the least performance impairments and stretch times of 6 seconds (repeated 6 times) can improve ROM while significantly lessening the negative impairment effects of static stretching. Thus stretch durations between 6 to 30 seconds are advocated.

9. **What is the optimal intensity of a static stretch?**
   Stretching to the point of discomfort (POD) is a common practice and is often believed to yield the greatest results. The vast majority of the literature has found that stretching to the POD results in decreased performance measures (decreased force production, jump height, and balance). Literature examining stretch intensities under a person's POD reports results of improved ROM and has been found to have less negative effects on performance.

10. **How often must static stretching be performed to maintain gains experienced during a static stretch session?**
    Bohannon found that stretch gains lasted 24 hours after a stretching session of the hamstrings. Zito reported no lasting effect of two 15-second passive stretches of the ankle plantar flexors after a 24-hour period. Clinically, this suggests that stretching should be performed at least every 24 hours.

11. **If an individual statically stretches on a regular basis, how long will the gains be retained?**
    According to Zebas, after a 6-week regimen of stretching, gains realized during that period were retained for a minimum of 2 weeks and in some subjects a maximum of 4 weeks.

12. **Does static muscle stretching alter performance?**
    According to a recent review the majority of the literature surrounding performance measures such as (force production, isokinetic power, and vertical jump) are impaired with static stretching. Impairments caused by static stretching can last upward of 2 hours in some instances. It should be noted that in some instances static stretching can improve performance of activities that require slower submaximal force production such as jogging and submaximal running or in jumping and hopping activities with longer contact times. Additionally, shorter stretch durations (<30 seconds) have less negative effects on dynamic activities. Finally, it is recommended that static stretching should be avoided in activities that require high-speed rapid movements or when explosive/reactive forces are required.

13. **Does dynamic stretching alter performance?**
    Yes. Dynamic stretching is preferred to static stretching when preparing for physical activity. According to the literature surrounding dynamic stretching, it has been shown that dynamic stretching enhances athletic performance and in some cases improves ROM similar to static stretching. Dynamic stretching activities should be carried out at frequency of 50 to 100 beats per minute. Dynamic stretching of at least three stretch repetitions of 30 seconds’ duration per muscle group is advocated. A 10-minute dynamic warm-up consisting of dynamic stretching, light aerobic activity, skipping, and hopping is best to prepare for physical activity.

14. **Does static stretching decrease the chance of injury?**
    Several studies have found that static stretching alone does not reduce injury. However, a dynamic warm-up that consists of stretching, strengthening, balance training, sport specific drills, and landing drills carried out for at least 3 months reduces injury.

15. **Does static stretching decrease muscle soreness?**
    Stretching focused on the reduction of delayed-onset muscle soreness (DOMS) after exercise has not been found effective at reducing pain. Some reports in the literature say that stretching can reduce DOMS; however, it is not statistically significant.
16. Is static stretching effective at reducing the effects of spasticity?
A systematic review by Bovend’Erdt, et al. in 2008 found the effects of stretching on spasticity to be inconclusive because of a lack of quality research in this area.

17. Does static stretching reduce joint contracture?
The exact cause of joint contracture is unknown, however, it is generally agreed upon that neurologic and nonneurologic factors contribute to the formation of joint contractures. A 2010 Cochrane Review concluded that for persons suffering with neurologic or nonneurologic joint contracture, stretching did not have clinically important immediate, short-term, or long-term effects on joint mobility. Additionally, it was found that pain; spasticity, activity limitation, participation restriction, or quality of life did not improve when stretching was employed for joint contracture.

18. Should a muscle joint complex be warmed up to optimize the effects of a stretch?
Not necessarily. Logically, it seems that increasing the tissue temperature before stretching would increase viscoelastic properties of the soft tissue surrounding a muscle joint complex; however, research has shown that stretching with or without a warm-up yields similar results.

19. What stretching technique results in the greatest flexibility gain?
According to a recent systematic review, static stretching seems superior to dynamic stretching as it relates to increasing joint ROM (however, dynamic stretching has been found to increase ROM).

20. Does age influence the extensibility of muscle and tendon?
It does appear that with increasing age the extensibility of the muscle tendon unit decreases (related directly to the calf muscle tendon unit). This is important with regard to normal ambulation, balance, and fall prevention in the older adult. A flexibility program directed toward the calf musculature appears to be a logical prevention program for the older adult.

21. Does stretching the gastrocnemius muscle in subtalar supination result in greater ankle dorsiflexion range of motion?
It is often theorized that stretching the gastrocnemius muscle in a subtalar neutral position will result in increased gastrocnemius muscle length because the totality of the stretch will be directed more specifically toward the target muscle (gastrocnemius) rather than the stretch force being dissipated across the midtarsal and subtalar joints. The literature suggests that there is no significant difference in the dorsiflexion ROM gains between individuals who stretched while maintaining the subtalar joint in supination versus pronation.

22. Does stretching alter joint position sense?
A brief stretching regimen of three stretches held for 30 seconds had no effect on knee joint position sense.

23. Is stretching effective at reducing neck pain?
According to the neck pain clinical practice guidelines published by the Orthopedic Section of the American Physical Therapy Association in 2008, only limited literature is available regarding the use of stretching and neck pain. Compared with manual therapy of the cervical spine, stretching of the cervical spine has been found to be equally effective. Stretching of the suboccipitals, scalenes, levator scapulae, upper trapezius, and pectoralis major and minor muscle groups should be considered in patients with neck pain.

24. Is stretching effective at reducing hamstring injuries?
According to a 2012 Cochrane Review, there is conflicting evidence to suggest that stretching the hamstrings will reduce hamstring injury. There is evidence to suggest that stretching after hamstring injury and exercise can reduce time to return to full activity.

25. Is stretching effective at reducing patellofemoral pain syndrome (anterior knee pain)?
According to a recent review, the most effective manner to treat patients with patellofemoral pain syndrome is a combined physical therapy program, including strength training of the quadriceps and hip abductors and stretching of the quadriceps muscle group.

26. Is stretching effective at reducing heel pain?
Stretching has been found to be no more effective than taping and bracing. The main pain-relieving benefits of stretching occur in the first 2 weeks to 4 months after the onset of stretching. Specific plantar fascia stretches may provide better short-term results versus Achilles stretching. Recommendations for frequency and number of repetitions are two to three times per day with a sustained hold of 15 to 30 seconds to as long as 3 minutes.
**CHAPTER 11 QUESTIONS**

1. When looking at acute increases in range of motion when performing static stretching, what is the optimal number of stretch repetitions?
   a. 1
   b. 2
   c. 3
   d. 4

2. Which of the following is true regarding static or dynamic stretching?
   a. Stretching to the point of discomfort results in the greatest increases in range of motion.
   b. Stretching to the point of discomfort has no effect on performance impairment.
   c. **Stretching has no effect on delayed onset muscle soreness.**
   d. Stretching is effective at reducing spasticity.

3. Which of the following is not true regarding stretching?
   a. Static stretches held for <30 seconds do not impair performance.
   b. **Dynamic stretching reduces and impairs performance.**
   c. Static stretching is effective a reducing joint contracture.
   d. Specific plantar fascia stretches are advocated for in the treatment of plantar/heel pain.
1. **What is manual therapy?**

Manual therapy, as described by the International Federation of Orthopedic Manual Therapists, is the use of skilled hand movements performed by physical therapists, chiropractors, or other health professionals to improve tissue extensibility, increase range of motion, induce relaxation, mobilize or manipulate soft tissue and joints, modulate pain, and reduce soft tissue swelling, inflammation, or restriction. Hands-on procedures such as mobilization, manipulation, massage, stretching, and deep pressure are all components of manual therapy.

2. **When is manual therapy treatment indicated?**

Manual therapy is used to treat motion impairments that cause pain and decreased range of motion. Joint-specific techniques are indicated when the motion impairment is caused by loss of the normal joint motion as a result of a reversible joint hypomobility. Manual therapy is typically contraindicated when the motion impairment is caused by excessive joint mobility. Motion impairment caused by weakened or shortened muscles is often an indication to use soft tissue techniques. Once pain has been reduced and joint mobility improved with the application of manual therapy, it is easier for a patient to regain normal movement patterns and restore maximal function. Current research has shown that manual therapy, when combined with therapeutic exercise, provides a beneficial outcome for patients. Therefore manual therapy is a technique to be used in combination with exercise during the episode of care.

3. **What is joint play?**

Mennell defined joint play as “a movement that cannot be produced by the action of voluntary muscles.” Joint play movements include distraction, compression, roll, glide, and spin. Loss of joint play frequently impairs range of motion. Manual therapy techniques use joint play movements for treating joint impairments.

4. **Is manual therapy always passive?**

Much of manual therapy is passive. However, some manual therapy techniques use the patient’s muscle contraction to assist or augment the treatment applied by the therapist. In these cases, the patient’s participation is an expected, extra force that enhances the technique. Manual therapy occurs in response to existing extrinsic forces (the therapist or gravity force) or intrinsic forces (patient’s muscle contraction or breathing) acting on the patient’s body.

5. **Describe the basic types of manipulations.**

Manipulation consists of techniques utilizing skilled passive movements to joints and/or soft tissues that are applied at varying speeds and amplitudes. Thrust manipulation employs high-velocity, low-amplitude therapeutic movement within or at the end range of motion of a joint, whereas nonthrust manipulation uses all of the same principles for soft tissue and joint impairments without the thrust component. Following are more specific definitions of various types of manipulations.

- **Joint manipulation (thrust)—**a localized, single passive movement using a high-velocity, low-amplitude thrust to bring the joint beyond its physiologic barrier. The result is distraction or translation of the joint surfaces. It does not exceed the anatomic barrier.
- **Joint mobilization (nonthrust)—**uses repetitive passive movements to return full range of motion and decrease pain. It moves joints within the physiologic ROM and uses three types of motion application: graded oscillation, progressive loading, and sustained loading.
- **Muscle energy/Osteopathic approach (nonthrust)—**uses patient’s active muscle contraction after joint is passively taken to restrictive motion. It is indicated when the limiting factor to motion is the neuromuscular system. The osteopathic approach uses postisometric relaxation principles and also employs thrust manipulation when deemed necessary.
6. What is a physiologic and anatomic barrier?
- A physiologic barrier is a point at which voluntary range of motion in a joint is limited by soft tissue tension. This is sometimes referred to as the end-feel of the joint. When the joint reaches the physiologic barrier, further motion toward the anatomic barrier can be induced.
- An anatomic barrier is a point at which passive range of motion of a joint is limited by bone contour, soft tissues (especially ligaments), or both. The anatomic barrier serves as the final limit to motion in an articulation. Movement beyond the anatomic barrier causes tissue damage.

7. Define direct and indirect manual therapy techniques.
- Direct technique—movement and force are in the direction of the motion restriction. Direct technique allows maximal restoration of movement; however, it may be painful when pain and muscle guarding are present.
- Indirect technique—movement and force are not both in the direction of the motion restriction. This technique is indicated in acute stages.

8. What is the difference between general and specific manual therapy techniques?
- General technique—the force is transmitted to a number of joints that have been determined to be hypomobile. A disadvantage of the general technique is that it can increase motion in an unstable joint not previously detected.
- Specific technique—the force is localized to one joint; therefore force transmission is minimized through the uninvolved joints.

9. Is there evidence that specific thrust manipulation techniques are delivered accurately to the targeted segment?
No. Studies have compared the target location of the technique with the location of the joints that actually produced an audible pop (cavitation) in response to manipulation therapy. It has been reported that spinal manipulation is accurate about half of the time. However, part of this accuracy was because of most procedures being associated with multiple audible joint cavitations, and in most cases, at least one audible cavitation emanated from the target joints. Therefore it seems that the clinical success of spinal manipulation is not dependent on the accurate delivery of that therapy to a specific targeted spinal joint.

10. What is the pop?
Popping of the joint frequently accompanies a manipulative thrust. The crack noise or joint cavitation is the result of generation or collapse of a gaseous bubble in the synovial fluid. Cineradiographic studies reported increased joint space and carbon dioxide gas production/breakdown after thrust manipulation. Because carbon dioxide is a gas with higher miscibility within the synovial fluid, this increase has been suggested to be the mechanism responsible for an increase in range of motion in the joint after manipulation. It has also been hypothesized that the cavitation would initiate certain reflex relaxation of the periarticular musculature. After the manipulation, the joint takes approximately 15 minutes to rearrange the gas particles and allow for another cavitation sound. Some people believe that if there is no noise after a thrust manipulation, that nothing has happened; this belief is incorrect. Studies have suggested no relationship between the occurrence of an audible pop during joint manipulation and improvement in pain, ROM, and disability in patients with nonradicular low back pain.

11. Describe the grading systems for joint mobilization.
Different grading systems exist for joint mobilization. Two of the most widely used grading systems are proposed by Maitland and Kaltenborn.

The Maitland system is based on joint oscillation techniques and has five different grades as follows:
- Grade 1—slow, small-amplitude movements performed at the beginning of the range
- Grade 2—slow, large-amplitude movements that do not reach the resistance or limit of the range
- Grade 3—slow, large-amplitude movements performed to the limit of the range
- Grade 4—slow, small-amplitude movements performed at the limit of the range
- Grade 5—small-amplitude, high-velocity movements (thrust) performed beyond the pathologic limitation of the range

Grades 1 and 2 are used mainly to reduce pain, grades 3 and 4 are used primarily to increase mobility, and grade 5 is used for the thrust technique and is indicated when resistance limits movement in the absence of pain in that direction.
The Kaltenborn system is based on sustained hold gliding techniques and traction. Kaltenborn has three grades of traction and two grades of gliding as follows:

**Traction**
- Grade 1—traction force that nullifies the compression forces acting on a joint, as a result of musculature tension, cohesive forces, and atmospheric pressure
- Grade 2—slack is taken up in the tissues surrounding the joint.
- Grade 3—beyond the slack, traction force is applied so that tissues crossing the joint are stretched.

**Gliding**
- Grade 2—translatoric gliding occurs until slack in joint is taken up and tightened.
- Grade 3—after slack is taken up and more force is applied, the tissues (capsular) crossing the joint are stretched.

* There is no grade 1 glide according to Kaltenborn.

12. Is there evidence that manual therapy is effective in the treatment of spinal conditions?

Yes. There is an extensive body of evidence showing the effectiveness of manual therapy for cervical, thoracic, and lumbar spine conditions.

**LOW BACK PAIN**
- Results have been mixed, with some showing spinal manipulation techniques (SMT) to be an effective intervention and others showing it to be ineffective. These results become clearer when patients are placed in subgroups based on specific evaluative findings. Based on updated criteria, patients who are likely to benefit from SMT have no symptoms distal to the knee, a recent onset of symptoms (<16 days), low Fear Avoidance Belief Questionnaire—Work (FABQW) scores (<19), hypomobility of the lumbar spine, and no movement restriction in hip internal rotation ROM (>35 degrees for at least one hip). Those patients with four or more of these factors present were more likely to improve, with those receiving manipulation experiencing greater relief from pain.
- SMT, when combined with exercise, has been found to be effective. A high-quality randomized clinical trial investigated the effect of adding exercise classes, spinal manipulation, or manipulation followed by exercise to “best care” in general practice for patients complaining of back pain. This study reported that although all groups improved over time, manipulation followed by exercise achieved the most significant benefits, followed by the spinal manipulation group, and last by the exercise group.
- Current clinical practice guidelines recommend the use of thrust procedures in those with acute low back pain to “reduce pain and disability in patients with mobility deficits.” It is also suggested that SMT may be beneficial for patients with subacute and chronic pain. These recommendations are based on strong evidence.

**THORACIC PAIN**
- SMT is frequently used in the thoracic spine as an intervention for cervical pain, shoulder pain, and even elbow pain. There is limited evidence regarding the use of SMT of the thoracic spine for thoracic pain. Several small randomized clinical trials (RCTs) have been completed, suggesting SMT directed at the thoracic spine is superior to placebo or no treatment. Additional studies have evaluated the effectiveness of SMT on segmental stiffness and general versus targeted techniques. SMT had small to no statistical effect on stiffness, but the targeted segments showed the greatest change in overall stiffness compared with general SMT.

**NECK PAIN**
- Evidence suggests manual therapy directed to the neck, particularly when combined with exercise, is an effective intervention for patients with mechanical neck pain with no radicular symptoms. One systematic review reported that spinal manipulation and/or mobilization is superior to general practitioner management for short-term pain reduction in patients with chronic neck pain. There is moderate evidence that mobilization is superior to physical therapy and family physician care. There is no evidence to support the use of thrust manipulation versus nonthrust manipulation (mobilization) for patients with neck pain.
- Manual therapy intervention directed to the thoracic region, instead of the cervical spine, has been shown to cause an immediate decrease in pain and increase in neck range of motion. It is theorized that biomechanical relationships between the cervical spine and thoracic spine make it possible for disturbances in joint mobility in the thoracic spine to contribute to movement restrictions and pain in the cervical region. Additionally, it appears that targeted supine thoracic
manipulation is superior to general seated thoracic manipulation, showing increases in cervical range of motion and decreases in pain.

- Current clinical practice guidelines recommend the use of SMT directed at the cervical spine, both thrust and nonthrust, for neck pain and headache. Similar to the lumbar spine, the effect of cervical manipulation is increased with the addition of exercise. This recommendation is based on strong evidence.
- Current clinical practice guidelines recommend the use of SMT, specifically thrust manipulation directed at the thoracic spine, for neck pain and neck related arm pain. This recommendation is based on weak evidence.

13. Is there evidence that manual therapy is effective in treating cervicogenic headache?
Systematic reviews suggest that thrust and nonthrust manipulation is effective for patients with cervicogenic headache. A more recent trial of patients with cervicogenic headaches compared a control group to groups receiving cervical thrust and nonthrust manipulation, strengthening of the deep neck flexor and scapular muscles, and a combined manual therapy and exercise group. The results showed significant reductions in headache symptoms in all treatment groups versus the control group. At 7- and 12-week follow-up visits, the combined exercise and manual therapy group showed some advantages over the other groups. Another RCT evaluated the effectiveness of manual therapy versus usual care at a general practitioner’s office. The authors found that mobilizations to the cervical and thoracic spine, combined with exercise and postural correction, reduced headache frequency and intensity. Eighty-one percent of the participants in the manual therapy group experienced at least a 50% decline compared with only 40% in the usual care group.

14. Is there evidence that manual therapy is effective in treating conditions of the extremities?

**HIP JOINT**
- A recent randomized trial compared manual therapy (thrust and nonthrust manipulations of the hip joint) with an exercise therapy program in patients with osteoarthritis of the hip. Success rates after 5 weeks were 81% in the manual therapy group and 50% in the exercise group. Furthermore, patients in the manual therapy group had significantly better outcomes on pain, stiffness, hip function, and range of motion.
- Current clinical practice guidelines recommend the use of manual therapy for patients with mild hip osteoarthritis. Manual therapy should be used to decrease pain and increase mobility of the joint. This recommendation is based on moderate evidence. Current clinical practice guidelines advocate, in the absence of contraindication, the use of manual therapy for patients with nonarthritic hip pain. Manual therapy can include nonthrust manipulation for capsular restriction and soft tissue techniques to treat muscular and fascial impairments. This recommendation is based on expert opinion.

**KNEE JOINT**
- One study compared a group who received manual therapy combined with exercise to a placebo group. Subjects in the manual therapy group received joint mobilization techniques to the lumbopelvic region, hip, knee, and/or ankle, depending on whether they exhibited pain or reduced mobility. The manual therapy plus exercise group showed improvements in pain, stiffness, and function. The control group did not change. Yet the combination of manual therapy and exercise resulted in positive effects.
- A systematic review in 2011 found evidence for the use of manual therapy in patient with knee osteoarthritis (OA), but the heterogeneity of the studies prohibited meta-analysis and a definitive conclusion when combining manual therapy and exercise. An RCT in 2013 showed equivocal results for manual therapy and exercise in terms of outcomes. A cost analysis done alongside showed manual therapy was cost effective and cost saving compared with exercise and usual care for knee OA.

**SHOULDER JOINT**
- Manual therapy used alone or combined with exercise has shown to be effective in the treatment of patients with shoulder problems. One trial studied the effectiveness of manipulative therapy for the shoulder girdle in addition to usual medical care. At 12 and 52 weeks after treatment, the manipulation group reported better rates of full recovery. A consistent between-group difference in severity of the shoulder pain, disability, and general health favored manipulative therapy.
- An RCT compared a group of patients with shoulder impingement syndrome who performed supervised flexibility and strengthening exercises with a group who performed that same exercise...
program plus received manual physical therapy treatment. They reported significantly more improvement in pain and function in the exercise plus manual therapy group.

- Current clinical practice guidelines for adhesive capsulitis report studies showing a beneficial effect from manual therapy, but there is little evidence to establish its efficacy versus other interventions. The recommendation, based on weak evidence, is that joint mobilization be targeted to the glenohumeral and be used to decrease pain and increase range of motion. A recent RCT compared manual therapy with corticosteroid injections for shoulder impingement. Both groups demonstrated approximately 50% improvement in outcome scores, but the corticosteroid group required additional visits to their primary care physician, additional injections, and utilized additional physical therapy resources. As with other equivocal outcomes, manual therapy proved to be superior in terms of efficiency and cost.

- Finally, there has been evidence presented that the use of thoracic manipulation may benefit those patients with subacromial impingement. Though follow-up was only 48 hours after, thoracic manipulation provided statistically significant decreases in patients’ reported pain and disability.

**ELBOW JOINT**

- Limited evidence indicates that mobilization with movement may help reduce painful movements and improve grip strength in patients with lateral epicondylalgia.
- A randomized trial of mobilization with movement (MWT) and exercise versus corticosteroid injections versus wait and see showed those treated with MWT and exercise had decreased pain and improved function. MWT was not superior to injections at the 52-week follow-up, but those who received MWT sought significantly less treatment than those who received injections.
- A systematic review showed short- and long-term benefits from mobilization with movement in patients with lateral epicondylalgia. There is, however, limited evidence to support one particular technique over another.

15. Is there evidence that manual therapy is effective for other conditions?
Less rigorous studies indicate that the use of manual therapy techniques may help decrease pain in patients with temporomandibular joint osteoarthrosis and in patients with fibromyalgia. There is also some indication that manual therapy may have positive effects on cervical radiculopathy, cervicogenic dizziness, carpal tunnel syndrome, thoracic outlet syndrome, and cubital tunnel syndrome. Few studies that have dealt with manipulation effectiveness used muscle energy or soft tissue techniques.

16. What are the expected side effects of spinal manipulation?
Reactions after spinal manipulation are very common in clinical practice. A recent study reported that approximately 61% of patients complain of at least one postmanipulative reaction. The most common side effects are stiffness (20%), local discomfort (15%), headache (12%), radiating discomfort (12%), fatigue (12%), muscle spasms (6%), dizziness (4%), and nausea (3%). Most reactions begin within 4 hours and generally disappear within 24 hours after treatment. Women are more likely to report side effects than men.

17. Is there any evidence to support the use of craniosacral therapy?
Although research exists reporting the presence of cranial bone motion, there are no rigorous studies to support craniosacral therapy as an effective therapeutic intervention.

18. Does manual therapy affect the visceral organs?
There is some emerging evidence to support a relationship between organ manipulation and mobilization and decreased pain in specific areas of the body. Some patients report improvement in their gastrointestinal discomfort or in constipation after thoracic or lumbar manipulation. Joint dysfunction facilitates the corresponding spinal cord segment, which can excite any of the neural elements arising from that segment, causing adverse visceral symptoms. There is a belief that when joint lesion is addressed, it may suppress or attenuate visceral complaints. To date, however, little evidence exists to validate the side effects of manual therapy for visceral problems.

19. Can manual therapy straighten a spinal deformity?
When there are structural spinal deformities, such as scoliosis or hyperkyphosis, manipulation cannot straighten the curves.

20. Can manual therapy restore spinal curvatures?
When there is a temporary loss of spinal curvature, such as in a lateral lumbopelvic list or in a straightened cervical spine because of muscle spasm, nonaggressive manipulative techniques can be used to decrease spasm and increase movement.
21. How does manual therapy help to increase range of motion and decrease pain and disability?

The specific in vivo effects of manual therapy are not known, but suggested theories include:

- Manual therapy moves or frees the mechanical impediment (loose body, disc material, synovial fringe, or meniscoid entrapment) to joint movement, permitting movement and halting nociceptive input and associated reflex muscle spasm.
- Improvement in range of motion helps to relieve pain that is the direct result of such hypomobility.
- Manual therapy stretches or ruptures periarticular scar tissues.
- Manual therapy may improve nerve conductivity and circulation by means of increasing the space where nerves and blood vessels exit or cross.
- Manual therapy improves muscle function and decreases stress on bones and ligaments by improving the distribution of joint forces and levers.
- Manual therapy initiates a number of neurophysiologic effects that can be linked to the favorable outcomes seen when treating musculoskeletal issues.

22. Should joint hypomobility be treated in the absence of symptoms?

No. Despite the fact that some clinicians advocate a prophylactic treatment for joint hypomobility, there is no evidence that this approach prevents dysfunction.

23. What is end-feel and how is it classified?

End-feel is a type of resistance felt by an examiner at a joint end range of a passive range of motion. Its assessment is used to guide diagnosis and treatment. End-feels can be normal or pathologic, depending on the movement they accompany at a particular joint and where in the range of movement they are felt. Pathologic end-feels are muscle spasms, sensation of soft end-feel, springy rebound, and severe pain without any motion restriction (empty end-feel).

Typical end feels:

- Bone to bone—abrupt stop to the movement that is felt when two hard surfaces meet (eg, passive extension of the elbow)
- Capsular—feeling of immediate stop of movement with some give (eg, end range of shoulder flexion)
- Tissue approximation—limb segment cannot be moved farther because the soft tissues surrounding the joint cannot be further compressed (eg, end range of knee flexion)
- Empty—patient complains of severe pain from the movement without the examiner perceiving an increase in resistance to the movement; indicates acute inflammation or extraarticular lesions
- Springy block—rebound is felt at the end of the range; results from displacement of an intraarticular structure
- Spasm—feeling of a muscle coming actively into play during the passive movement; indicates the presence of an acute or subacute condition
- Soft—results from soft tissue approximation or soft tissue stretching (eg, resistance felt at the end range of knee flexion)

24. What are the general contraindications to manual therapy?

- Fracture
- Instability of the target joint
- Infectious arthritis
- Tumors
- Joint ankylosis
- Acute inflammatory disorders
- Lack of diagnosed joint lesion
- Presence of pathologic end-feel

25. List specific contraindications for thrust manipulation.

- Cranial nerve signs or symptoms and dizziness of unknown origin (specific for cervical spine)
- Sacroperineal numbness or loss of bowel and bladder control (specific for lumbar spine)
- Painful movements in all joint directions or just one degree of movement free of pain and restriction
- Bilateral or multisegmental neurologic signs or symptoms
- Paralysis in nonperipheral nerve distribution
- Hyperreflexia or positive pathologic reflexes
- Presence of emotional disorders
- Patient taking anticoagulant medication or steroidal medication for a long period
26. Describe the convex-concave rule, and explain how it influences manual therapy.

Though this theory is currently debated, the classic definitions are still applied to manual techniques. When a convex joint surface moves on a fixed concave joint surface, joint rolling and gliding occur in opposite directions. Conversely, when a concave joint surface is moved on a fixed convex joint surface, rolling and gliding occur in the same direction. This rule helps clinicians to decide what direction to apply joint manipulation therapy in. When performing a nonthrust joint manipulation (mobilization), the therapist moves a bone with a convex joint surface in the direction opposite to the restriction, whereas nonthrust manipulation of a concave joint surface is performed in the same direction as the restriction.

27. Describe loose-packed and close-packed positions.

- **Loose-packed position**—resting position in which the joint capsule is most relaxed, the articular surfaces are least congruent, and the greatest amount of joint play is possible. This resting position does not take into account extraarticular structures, such as muscles and fascia. Most joint manipulations are performed in this position and are progressed to the close-packed position.

- **Close-packed position**—the joint capsule and ligaments are tight or at maximal tension. In this position there is maximal contact between the concave and convex articular surfaces, and separation between the articular surfaces by traction forces is difficult.

28. How do the loose-packed and close-packed positions influence manual therapy treatment?

Knowledge of these positions allows clinicians to determine which movement compresses and tightens the joint and which movement distracts and loosens the joint. The loose-packed position is the position used for testing joint play and for starting treatment of restricted joint movement. The close-packed position is used to avoid joint movement. As an example, in order to isolate the mobilizing force to a particular level of the spine, the adjacent vertebral joints are locked in the close-packed position.

29. Define capsular pattern.

Capsular pattern is a limitation of joint movement that occurs in a predictable fashion. Cyriax suggested that these patterns are a result of lesions in the joint capsule or the synovial membrane. It indicates loss of mobility of the entire joint capsule from fibrosis, effusion, or inflammation, which may occur in arthrosis, arthritis, prolonged immobilization, or acute trauma. Joints not controlled by muscles, such as the sacroiliac or tibiofibular joints, do not exhibit a capsular pattern.

30. Compare loose-packed position, close-packed position, and capsular pattern for all joints.

<table>
<thead>
<tr>
<th>Joint</th>
<th>Loose-Packed</th>
<th>Close-Packed</th>
<th>Capsular Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temporomandibular</td>
<td>Mouth slightly open</td>
<td>Teeth clenched</td>
<td>Limited mouth opening</td>
</tr>
<tr>
<td>Cervical spine</td>
<td>Midway between flexion and extension</td>
<td>Maximal extension</td>
<td>Limited in all motion, except flexion</td>
</tr>
<tr>
<td>Steroclavicular</td>
<td>Arm resting by side</td>
<td>Maximal shoulder elevation</td>
<td>Limited full elevation; pain at end ranges</td>
</tr>
<tr>
<td>Acromioclavicular</td>
<td>Arm resting by side</td>
<td>Arm abducted 90°</td>
<td>Limited full elevation; pain at end ranges</td>
</tr>
<tr>
<td>Glenohumeral</td>
<td>55° shoulder abduction, 30° horizontal adduction</td>
<td>Maximal abduction and external rotation</td>
<td>Loss in external rotation &gt; loss in abduction &gt; loss in internal rotation</td>
</tr>
<tr>
<td>Humeroulnar</td>
<td>70° flexion, 10° supination</td>
<td>Full extension and supination</td>
<td>Loss of flexion &gt; loss in extension</td>
</tr>
<tr>
<td>Humeroradial</td>
<td>Extension and supination</td>
<td>90° flexion, 5° supination</td>
<td>Loss of flexion &gt; loss in extension</td>
</tr>
</tbody>
</table>
Radioulnar: proximal 70° flexion, 35° supination, 5° supination, full extension Limited pronation = limited supination
Radioulnar: distal 10° supination 5° supination Limited pronation = limited supination
Radiocarpal Neutral, slight ulnar deviation Full extension, radial deviation Limited flexion = limited extension
Midcarpal Neutral, slight flexion and ulnar deviation Full extension Equal limitation in all directions
Trapeziometacarpal Neutral Full opposition Limited abduction > extension
Carpometacarpal Neutral Full opposition Equal limitation in all directions
Metacarpophalangeal Slight flexion, ulnar deviation Full flexion
Interphalangeal Slight flexion Full extension Limited flexion > extension
Thoracic spine Midway between flexion and extension Maximal extension Side-bending and rotation > extension > flexion
Lumbar spine Midway between flexion and extension Maximal extension Equal limitation of side-bending and rotation; extension > flexion
Hip 30° flexion, 30° abduction, slight external rotation Full extension, abduction, internal rotation Flexion and internal rotation > abduction > external rotation
Tibiofemoral 25° flexion Full extension and external rotation Limited flexion > extension
Talocrural 10° plantar flexion, neutral inversion/eversion Full dorsiflexion Plantar flexion > dorsiflexion
Subtarsal 10° plantar flexion, neutral inversion/eversion Full inversion Limitation in varus
Midtarsal 10° plantar flexion, neutral inversion/eversion Full supination Supination > pronation
Tarsometatarsal Neutral supination and pronation Full supination
Metatarsophalangeal Interphalangeal Neutral Slight flexion Full extension Extension > flexion Limited extension

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CHAPTER 12 QUESTIONS

1. Which of the following manual therapy techniques can be considered harmful when used by inexperienced or untrained practitioners?
   a. Joint manipulation
   b. Joint mobilization
   c. Muscle energy
   d. Soft tissue mobilization

2. During assessment, your patient complains of severe pain during a movement of the shoulder and voluntarily increases his resistance to movement. Which of the following Cyriax-described end-feels best classifies his condition?
   a. Capsular
   b. Springy
   c. Spasm
   d. Empty

3. When subgrouping patients with LBP, which of the following is NOT one of the criteria that predicts success with the application of spinal manipulative therapy?
   a. Hypomobility of the lumbar spine
   b. No symptoms distal to the knee
   c. SLR >90 degrees
   d. An onset of symptoms <16 days

4. There appears to be an interaction effect when SMT is combined with which of the following interventions?
   a. Ultrasound
   b. Electrical stimulation
   c. Exercise
   d. Biopsychosocial interventions
1. Discuss briefly the common approaches to massage.

   Massage techniques differ in origin and the basic premise behind their effectiveness. Classic Western massage was developed in Europe and the United States over the past two centuries. Western massage is based on the Western medical model of disease, with mechanical and neurologic rationales supporting its use as therapy. Contemporary massage and bodywork and Asian bodywork are widely diverse in their rationale, which includes energy balancing, myofascial softening and lengthening, and traditional Chinese medicine and meridian theories.

2. Does massage boost the immune system and reduce inflammation?

   There is a growing body of evidence supporting the positive effects of massage on boosting the immune system and reducing inflammation. Regarding inflammation specifically, Crane et al. in 2012 studied the effects of massage on eleven young males with exercise-induced muscle damage. They analyzed the biopsies acquired from the vastus lateralis at baseline, after 10 minutes of massage, and after a 2.5-hour period of recovery. They concluded that massage was clinically beneficial in reducing inflammation and promoting mitochondrial biogenesis. Relating to the immune system, Ang et al. in 2012 compared the effects of massage therapy versus sham therapy on 120 preterm infants. They concluded that infants who received ≥5 consecutive days of massage demonstrated greater daily weight gain and higher natural killer cytotoxicity. Rapaport et al. in 2010 and 2012 studies compared the effects of Swedish massage therapy versus a light touch control group on oxytocin (OT), arginine-vasopressin (AVP), adrenal corticotropin hormone (ACTH), cortisol (CORT), circulating phenotypic lymphocyte markers, and mitogen-stimulated cytokine function. They concluded that compared with the touch control condition, a single massage session or 5 weeks of Swedish massage stimulated a sustained pattern of increased circulating phenotypic lymphocyte markers and decreased mitogen-stimulated cytokine production and had minimal effect on hypothalamic-pituitary-adrenal function. In contrast, twice-weekly massage produced a different response pattern with increased OT levels, decreased AVP, decreased CORT, minimal effects on circulating lymphocyte phenotypic markers, and a slight increase in mitogen-stimulated interferon-gamma, tumor necrosis factor-alpha, and interleukin (IL)-1β and IL-2 levels, suggesting increased production of proinflammatory cytokines.

3. Does massage improve lymphatic drainage?

   Bass et al. in 2001 investigated the effect of massage on the sensitivity of lymphatic mapping in breast cancer. After the injection of a blue dye and radiocolloid, the patient received a 5-minute massage. They concluded that massage significantly improved the uptake of blue dye by sentinel lymph nodes.

4. Does massage increase tissue temperature?

   Drust et al. in 2003 examined the effects of massage on intramuscular temperature in the vastus lateralis in humans. They concluded that when comparing massage with ultrasound, changes in muscle temperature were significantly higher for massage at 1.5 to 2.5 cm below the skin. They also determined that thigh skin temperatures were significantly higher in massage-treated patients. More recently, Sefton et al. in 2010 demonstrated, through thermography, that massage therapy produced significant increases in temperature over time in the anterior upper chest, posterior neck, upper back, right arm, and the middle back. Additionally, the temperatures remained above baseline levels after 60 minutes.

5. Does massage decrease depression?

   Field et al. in 2004 studied massage therapy effects on depressed pregnant women. The massage therapy group participants received two 20-minute massage therapy sessions by their significant others for 16 weeks of pregnancy starting during the second trimester. By the end of the study, the massage group had higher dopamine and serotonin levels and lower levels of cortisol and norepinephrine.
Field et al. in 2012 compared the effects of yoga, massage therapy, or standard prenatal care on prenatal depression and neonatal outcomes for 84 prenatally depressed women. Following 12 weeks of twice weekly yoga or massage therapy sessions (20 minutes each) both therapy groups, versus the control group, had a greater decrease in depression, anxiety, and back and leg pain. In addition, both groups had greater gestational age and birth weight than the control group. In 2011 Krohn et al. and Poland et al. in 2013 examined the effects of massage on depression in patients with breast cancer or HIV, respectively. Both authors concluded that massage can reduce depression in both of these patient populations.

6. How does massage generate pain relief?
Several mechanisms have been proposed and researched as possible sources of pain relief after massage. One of the oldest theories is that light to moderate mechanical stimulation of cutaneous and subcutaneous tissues results in increased activity in somatosensory neurons, which may inhibit activity in pain-mediating neurons in the spinal cord. This is based on the gate control theory of pain developed by Melzack and Wall in 1965. Another proposed theory is that increased stimulation activation of the descending pain inhibitory system, starting in the periaqueductal gray matter (PAG) and continuing to the dorsal horn of the spinal cord, may reduce pain. In conjunction with this theory is the belief that opioid receptors in the PAG are activated as a result of massage. Last, Lund et al. in 2002 investigated the mechanisms behind the effects of massage on animals. They concluded that long-term pain relief effects of massage may be attributed, at least in part, to the oxytocinergic system and its interaction with the opioid system. Although this mechanism is not well understood, it is theorized that increased endogenous oxytocin may result in greater synthesis of endogenous opioids.

7. Does massage aid in recovery from exercise and competition?
Hemmings et al. in 2000 examined the effects of massage on physiologic restoration, perceived recovery, and repeated sports performance on eight amateur boxers. They concluded that although there were significantly increased perceptions of recovery after massage, there were no significant differences in blood lactate or glucose levels. Another study by Hilbert et al. in 2003 examined the effects of massage on delayed-onset muscle soreness. They determined that massage administered 2 hours after exercise-induced muscle injury to the hamstrings did not improve function but did reduce the intensity of soreness 48 hours post injury. Another study examined the effects of leg massage on recovery from high-intensity cycling exercise. They concluded that massage had no effect on blood lactate concentration, heart rate, and maximum and mean power. They did find that the massage group had a significantly lower fatigue index.

8. Does massage improve muscle flexibility?
Hopper et al. in 2005 examined the effects of classic soft tissue mobilization (STM) and dynamic STM on hamstring length in 45 healthy male subjects. The dynamic STM consisted of classic STM followed by distal to proximal longitudinal strokes performed during passive, active, and eccentric loading of the hamstring. They concluded that dynamic STM significantly increased hamstring flexibility compared with classic STM and controls. In a separate study Hopper et al. in 2005 examined the effects of dynamic STM and classic STM on hamstring length in 50 female field hockey players. In this study, passive straight leg raise and passive knee extension were measured before, after, and 24 hours post intervention. They concluded that both massage techniques significantly improved hamstring length immediately after massage and had no carryover 24 hours post intervention. Huang et al. in 2010 examined the effects of no massage, 10 seconds of massage, and 30 seconds of massage on hip flexion angle, passive leg tension, and electromyography (EMG) in 10 women ages 21 to 23 years old. Treatments were randomized over 3 days. Treatment effects were greatest for the 30 seconds of massage, which resulted in a 7.2% increase in hip angle. There was no significant difference in passive tension or EMG for either the control or the treatment. They concluded that this musculotendinous massage may be used in place of, or in adjunct to, static stretching to improve hamstring flexibility. Finally, Forman et al. in 2014 examined the effects of deep stripping massage strokes (DSMS) in isolation or combined with eccentric resistance on hamstring length in 89 participants. In this study, DSMS consisted of longitudinally directed deep pressure strokes over the hamstring muscles, applied for a duration of 10 seconds, and repeated 15 times with the hamstring relaxed or held in eccentric contraction. They concluded that DSMS + eccentric contraction and DSMS alone resulted in improved flexibility of 10.7% and 6.3%, respectively.

9. Does massage increase blood flow?
Hinds et al. in 2004 examined the effects of massage on limb and skin blood flow after quadriceps exercise. A total of 13 participants performed three 2-minute bouts of concentric quadriceps exercise, followed...
either by two 6-minute bouts of deep effleurage and pêtrissage massage or by a rest period of similar duration. Measures of femoral artery blood flow, skin blood flow, skin temperature, muscle temperature, blood lactate concentration, heart rate, and blood flow were compared. They concluded that skin temperature and skin blood flow were significantly elevated after the application of massage. There were no significant differences between the massage and control groups for the remaining measurements.

10. Does massage decrease blood pressure?
Cambron et al. in 2006 examined the effects of six different massage techniques on 150 participants. Techniques included Swedish, deep tissue, myofascial release, sports, trigger point, and craniosacral. The authors concluded that clients receiving Swedish massage (effleurage and pêtrissage) experienced the greatest reduction in blood pressure, and those who received trigger point therapy and sports massage experienced an increase in blood pressure. Limits to the generalization of the results of this study include a small sample size and lack of control regarding treatment duration and technique standardization.

11. Does massage decrease the frequency of chronic tension headaches?
Quinn et al. in 2002 investigated the effect of massage therapy on chronic nonmigraine headaches. Chronic tension headache sufferers received structured massage therapy treatment to the neck and shoulder muscles. They concluded that headache frequency was significantly reduced within the first week of treatment and continued throughout the study. The duration of headaches also tended to decrease during the massage treatment period. Headache intensity was unaffected by massage.

12. Does massage increase range of motion in patients with cervicogenic headache?
Hopper et al. in 2013 investigated the short term effects of soft tissue massage applied to the neck muscles. Range of motion was measured using the flexion-rotation test immediately preintervention and postintervention after each of the three treatment sessions and 2 weeks following the final treatment. They concluded that range of motion increased significantly from treatment session one to three. Improvement was greatest after the first and second treatments and remained stable after the final treatment to the 2-week follow-up. Limits to the generalization of the results of this study include a small sample size, limited age range of participants, and a lack of a control group.

13. Does massage improve adverse neural tension signs and symptoms?
De-la-Llave-Rincon et al. in 2012 examined the effects of soft tissue mobilization and nerve slider neurodynamic technique on the numeric pain rating score (NPRS) and pain pressure threshold over the median, radial, and ulnar nerves; the C5-C6 zygapophyseal joint; the carpal tunnel; and the tibialis anterior muscle. Participants consisted of 18 women with a clinical and electromyographic diagnosis of carpal tunnel syndrome. Each participant received both treatments at potential entrapment sites of the median nerve. The authors concluded that the combination of soft tissue mobilization and neurodynamic technique decreased pain intensity for up to 1 week post treatment, but did not change pressure pain sensitivity. Saban et al. in 2014 compared the effects of deep massage therapy (DMS) and neural mobilization with a self-stretch program to ultrasound with the same self-stretch program for 69 participants experiencing heel pain. They concluded that both treatment protocols resulted in short-term improvement and that the DMS treatment was significantly more effective than the ultrasound treatment. Krauss et al. in 2012 examined the effects of functional massage, a technique using soft tissue massage and nonpainful joint motion, on the straight leg raise test in 12 asymptomatic young adults. The massage was applied along the course of the sciatic nerve in the posterior thigh and leg. Participants demonstrated an average improvement in ROM of 11.6 degrees immediately following treatment and 5.3 degrees at 1-week follow-up. Limits to this study include the small sample size, lack of a control group, and a limited age range of participants.

14. What is the origin, nature, and purpose of functional massage?
Co-developed by Krauss and Evjenth, functional massage integrates soft tissue massage (in the form of manual soft tissue compression and decompression) and nonpainful joint motion (both angular and translatorial). The goals of functional massage are to: 1) manage musculotendinous and periarticular soft tissue pain and tension and 2) aid in the management of impaired segmental and/or joint motion, impaired muscle function/performance, and impaired neural dynamics caused by, or associated with, musculotendinous and/or periarticular soft tissue pain, and/or tension, and/or gliding restrictions. During functional massage the joint is repeatedly moved so that musculotendinous and/or periarticular soft tissues are lengthened/tensed and shortened/slackened while massage pressure is applied. Functional massage is used to reduce pain and improve mobility. By integration of joint motion and massage it
is theorized that proprioceptors from both the joint and the muscle are stimulated, potentially increasing the treatment’s counterirritation effects at the spinal cord level. In addition, muscle activity during the massage may range from passive to fully active, allowing for a passive intervention to be morphed into an active assistive and/or a fully active motion. Finally, functional massage integrates soft tissue broadening, lengthening, and gliding of the muscles, tendons, fascia, and nerves that occur during normal body function. Indications for functional massage include musculoskeletal pain and/or stiffness and resultant impairment(s) in muscle performance, joint mobility, tendon and neural mobility, and soft tissue edema/swelling. Contraindications for functional massage include severe hypomobility and injuries, medical conditions, and/or medication-related conditions resulting in severe vascular or connective tissue fragility.

15. What is the purpose of Cyriax transverse friction massage? Cyriax transverse friction massage provides movement to the muscle or tendon while inducing traumatic hyperemia in order to stimulate healing.

16. What are the basic principles of transverse friction massage?
- The soft tissue lesion must be properly treated.
- Friction is given across the grain of the soft tissue.
- The therapist’s fingers must move together with the patient’s skin.
- Friction must have sufficient depth and sweep.
- The patient must be comfortable.
- Tendon is put on stretch, whereas muscle is massaged in a relaxed position.

17. Does transverse friction massage induce healing? No well-performed studies have shown histologic support for the promotion of healing of soft tissue with transverse friction massage. Walker examined the use of transverse friction massage on medial collateral ligaments of rabbits and found no difference between massaged and control rabbits. However, the experimentally induced sprain may have been insufficient to promote an inflammatory response. In a recent systematic review, Joseph et al. in 2012 concluded that there is limited evidence supporting the use of deep friction massage (DFM) in combination with Mill’s manipulation in the treatment of elbow tendinopathy and in combination with joint mobilization for the treatment of supraspinatus tendinopathy in the presence of outlet syndrome. There remains a lack of evidence examining the isolated efficacy of DFM.

18. How long should transverse friction massage be performed? The dosing of transverse friction massage is based on the intended or expected outcomes of the massage. If the intention is to decrease pain, then the massage may be performed until the patient reports decreased pain. If the intention is to improve tissue pliability, then the massage may be performed until a palpable change in pliability is noted by the therapist. If the intention is to stimulate a mild inflammation in a tendon that is degenerative but nonpainful, then the massage may be performed until a mild sensation of discomfort is perceived by the patient. Because of the diverse intentions of transverse friction massage, individual treatment doses may vary from a few minutes up to 15 to 20 minutes. The total number of treatment sessions is also dependent on the intended outcomes of the treatment. Changes from friction massage are often noted within one treatment session and at the very least should be noted within two treatment sessions. Failure to achieve results should lead to a careful consideration of the specific treatment parameters (rate, depth, direction, and duration, eg) and of the treatment choice itself. Typically, friction massage is used for up to four to six treatment sessions, with a great deal of variability in the total number of treatment sessions depending on the nature of the specific condition/impairment undergoing treatment.

BIBLIOGRAPHY


**CHAPTER 13 QUESTIONS**

1. Based on the literature which of the following conditions may be improved by the application of massage?
   a. depression
   b. high blood pressure
   c. post exercise fatigue
   d. lymphedema
   e. all of the above may be improved by the application of massage

2. Which of the following are the most likely mechanisms for the reduction of pain associated with massage?
   a. the gate control mechanism
   b. increased activation of the descending pain inhibitory system
   c. increased synthesis of endogenous opioids
   d. all of the above are likely mechanisms for the reduction of pain associated with massage

3. Which of the following outcomes are associated with the massage?
   a. increased tissue temperature
   b. increased myofascial flexibility
   c. reduced inflammation
   d. improved range of motion
   e. all of the above are outcomes associated with massage
1. What are the theoretical effects of spinal traction?

Spinal traction is theorized to have several effects. Among these are distraction or separation of the vertebral bodies, a combination of distraction and gliding of the facet joints, tensing of the ligamentous structures of the spinal segment, widening of the intervertebral foramen, straightening of spinal curves, and stretching of the spinal musculature. There is evidence that a disc protrusion can be reduced and spinal nerve root compression symptoms relieved with the application of relatively high-force spinal traction (approximately 50% of the body weight). Epidurography studies demonstrate temporary reduction in disc protrusions, along with clinical improvement. Onel et al. (1989) used computed tomography (CT) to demonstrate lumbar disc reduction in 21 of 30 patients (70%) and theorized that the reduction was as a result of a suction effect caused by decreased intradiscal pressure. The change in intradiscal pressure caused by traction also has been theorized to positively affect the disc’s nutrition.

2. What are the indications for spinal traction?

Given these theoretical effects, the significant indications are herniated disc or radiculopathy, any condition in which mobilization and stretching of soft tissue are desired, and any condition in which opening the neural foramen is desired.

3. What are the contraindications for spinal traction?

Traction is contraindicated in patients with structural disease secondary to tumor or infection, rheumatoid arthritis, severe vascular compromise, and any condition for which movement is contraindicated. Relative contraindications include acute strains and sprains and inflammatory conditions that may be aggravated by traction. Strong traction applied to patients with spinal joint instability may cause further strain. Traction should be avoided if the patient has had recent spinal fusion. Because spinal fusion techniques and healing rates vary from patient to patient, the surgeon should be consulted before applying traction if the fusion is less than 1 year old. Other relative contraindications may include pregnancy, osteoporosis, hiatal hernia, and claustrophobia.

4. How much force is optimal for cervical traction?

In the cervical spine, Judovich found that 25- to 45-lb forces were necessary to demonstrate a measurable change in the posterior cervical spine structure. Colachis and Strohm demonstrated that a traction force of 30 lb produced separation of the cervical spine and that a 50-lb force produced more separation than a 30-lb force. There is no evidence that midcervical and lower cervical spine separation occurs at forces less than 20 lb.

5. Is cervical traction effective for the treatment of cervical radiculopathy?

One MRI study showed either complete or partial reduction of herniated disc in 21 of 29 patients who received 30-lb seated traction with an inflatable traction device. Honet and Puri provided a progressively more intense cervical traction treatment, depending on severity of symptoms and neurologic findings. Subjects received traction treatment at home, in an outpatient facility, or in the hospital. The percentage of patients with excellent or good outcomes was 92% in the home treatment category, 77% in the outpatient treatment category, and 65% in the hospital treatment category.

6. Is cervical traction effective for treatment of cervicogenic headache?

No clinical trials have been performed using cervical traction to treat cervicogenic headache, but two case studies have suggested that cervicogenic headache can be treated successfully with traction. Using 25- to 30-lb home traction and cervical exercise, Olson reported success with two difficult cases of headache caused by chronic whiplash. The cervical exercise consisted of postural correction and stabilization exercises.
7. What are the important treatment variables for cervical traction?

- Chin halter versus occipital wedges—when traction is provided with a standard head halter with a chin strap, force is transmitted through the chin strap to the teeth, and the temporomandibular joints become weight-bearing structures. A common problem from administering cervical traction is aggravation of the temporomandibular joints because of the force applied at the chin. It is generally advisable to use a cervical traction system that pulls from the occiput, rather than placing pressure on the chin. If the patient has known temporomandibular joint dysfunction, a chin halter should never be used.

- Force—to effectively treat cervical radiculopathy, herniated disc, or other conditions requiring a separation of the intervertebral space, the traction force should be great enough to cause movement at the spinal segment. Based on our experience and the evidence available in the literature, we typically use a force of 25 to 40 lb for the midcervical and lower cervical spine. Less force is necessary when treatment is directed to the upper cervical area.

- Patient position—we recommend the supine position to facilitate patient relaxation, proper force application, and optimal cervical angle. The supine position is favored in the literature. Cervical traction studies show that narrowing of the intervertebral spaces can actually occur during the traction treatment in patients who are unable to relax.

- Cervical angle—cervical traction is performed with the head and neck in some degree of flexion. Some clinicians believe that the greater the angle of flexion, the greater the intervertebral separation in the lower cervical spine. Although it is true that posterior separation does increase with more flexion, anterior separation decreases with flexion. In most cases, clinicians should try to achieve a combination of a posterior and anterior stretch. Thus the ideal traction device will flex the head and neck somewhat but pull at a relatively flat angle. We recommend a 15-degree angle to accomplish this goal.

- Mode (static or intermittent)—the traction mode selected will depend on the disorder being treated and the comfort of the patient. Herniated disc is usually treated more effectively in static mode or with longer hold-rest periods (3- to 5-minute hold, 1-minute rest) in intermittent mode. Joint dysfunction and degenerative disc disease usually respond to shorter hold-rest periods (1- to 2-minute hold, 30-second rest) in intermittent mode.

- Time—when treating herniated disc, the treatment time should be relatively short. As the disc space widens, the intradiscal pressure decreases, causing the herniated disc material to be retracted into the disc space. The decrease in pressure is temporary, however, because eventually the decreased intradiscal pressure will cause fluid to be imbibed into the disc. When pressure equalization occurs, the suction effect on the disc protrusion is lost, and it is possible for patients to experience a sudden increase in pain when traction is released. If the traction time is 8 to 10 minutes, this effect is minimized. For other conditions, a treatment time of up to 20 minutes is often used. As a general rule, the higher the force, the shorter the treatment time. Often the first treatment is only 3 to 5 minutes long. This gives the clinician a chance to determine the patient’s reaction to treatment and plan treatment progression accordingly.

8. How much force is optimal for lumbar traction?

There is consensus in the literature that a force of 40% to 50% of the patient’s body weight is necessary to cause vertebral separation. In one of the earliest lumbar traction studies, Cyriax reported a visible separation between lumbar vertebrae with static traction of 120 lb for 15 minutes. Other studies have reported measurable separation in the lumbar spine at forces ranging from 80 to 200 lb. Judovich advocated a force equal to one half the patient’s body weight on a friction-free surface as the minimum force necessary to produce therapeutic effects in the lumbar spine.

9. Is lumbar traction effective for lumbar radiculopathy?

Epidurography and CT investigations have shown that high-force traction can reduce disc protrusions and relieve spinal nerve root compression symptoms. Despite these findings, lumbar traction is currently out of favor in the literature. Four reviews summarizing lumbar traction studies have concluded that there is no significant benefit for patients treated with lumbar traction compared with a control group. However, wide variations of methods and techniques were described in the studies cited. Some of the studies that showed lumbar traction to be ineffective were performed with low forces. In many of the studies, patient selection criteria were poorly defined. Most studies tended to group all patients with low back pain together and did not distinguish between subgroups or by diagnosis. The only two studies that looked specifically at traction for herniated disc did not use forces generally considered sufficient to separate the intervertebral spaces.
10. What are the most important treatment variables for lumbar traction?

- Force—to effectively treat lumbar radiculopathy, herniated disc, or other conditions requiring a separation of the intervertebral space, the traction force should be great enough to cause movement at the spinal segment. Based on our experience and the evidence available in the literature, we typically use a force of 40% to 50% of the patient’s ideal body weight. Often the first treatment is a little less to ensure patient tolerance.

- Spinal position—the position of the spine during traction is an important treatment variable. In our experience, disc herniation is most effectively treated with the patient lying prone with a normal lordosis. However, this position is not always possible because a patient with an acute herniated disc may not tolerate any position of normal lordosis. If this is the case, the treatment must be given in flexion initially with the goal of gradually working toward neutral lumbar lordosis. Foraminal (lateral) stenosis is usually more effectively treated with the lumbar spine in a flexed (flattened) position initially, with the goal of achieving a neutral lordosis when possible. Soft tissue stiffness/hypomobility and degenerative disc or joint disease may be treated in a neutral position, or some degree of flexion or extension, depending on the goals of treatment. Patient comfort and the patient’s ability to remain relaxed during the treatment are important considerations when choosing the most beneficial position, and no absolute rule applies. Variations of flexion, extension, and lateral bending should be tried to find the most beneficial position for each patient.

- Mode (static or intermittent) and time—see question 7.

11. Does spinal traction change somatosensory evoked potentials (SSEPs)?

SSEP latencies were decreased after cervical traction in patients with radiculopathy and cervical spine. In patients with severe myelopathy, latencies may increase. Traction may improve conduction by improving blood flow to cervical nerve roots.

**BIBLIOGRAPHY**


**CHAPTER 14 QUESTIONS**

1. With regard to the position of the spine during lumbar spinal traction, which of the following is accurate?
   a. The most effective treatment position for a disc herniation is prone with normal lordosis.
   b. There are absolute rules to positioning of the lumbar spine.
   c. Sidebending should not be considered as a positional option for lumbar spinal traction.
   d. The more effective treatment position of foraminal stenosis is extension of the lumbar spine.

2. What is the recommended force to treat a 180-lb man with a cervical radiculopathy?
   a. 5 to 24 lbs
   b. 25 to 40 lbs
   c. 41 to 60 lbs
   d. 61 to 76 lbs

3. Which of the following is true with regard to spinal traction?
   b. A chin halter should never be used on a patient diagnosed with TMJ dysfunction.
   c. Traction is recommended for patients who have recently had a spinal fusion.
   d. Nerve root compression is relieved with low-force spinal traction (less than 45% of body weight).
NORMAL AND PATHOLOGIC GAIT
J.M. Burnfield, PT, PhD

1. What is the average adult walking velocity?
   - On level surfaces, approximately 80 m/min
   - In men, 82 m/min
   - In women, 78 m/min

2. Does walking velocity decline with age?
   Yes. Declines of 3% to 11% in healthy adults >60 years old have been reported.

3. Name contributors to an individual’s walking velocity.
   - Step (or stride) length
   - Cadence

4. What is considered normal stride and step length?
   - Stride length is the distance from ipsilateral heel contact to the next ipsilateral heel contact during gait (ie, right-to-right or left-to-left heel contact). Normal adult stride length averages approximately 1.39 m, with the mean stride length of men (1.48 m) being slightly longer than that of women (1.32 m).
   - Step length is the distance between ipsilateral and contralateral heel contact (eg, right-to-left heel contact) and is on average equal to half of stride length.

5. What is normal cadence?
   Cadence is the number of steps per minute.
   - In adults without pathology, the average is 116 steps/min
   - In women, 121 steps/min
   - In men, 111 steps/min

6. Define gait cycle.
   Gait cycle is a repetitive pattern that extends from heel contact to the next episode of heel contact of the same foot. The gait cycle can be further subdivided into a period of stance, when the limb is in contact with the ground (approximately 60% of the gait cycle), and a period of swing, when the limb is not in contact with the ground (approximately 40% of the gait cycle).

7. Describe the functional tasks associated with normal gait.
   Functionally, each gait cycle can be divided into three tasks:
   1. Weight acceptance
   2. Single limb support
   3. Swing limb advancement
      During weight acceptance, body weight is accepted onto the limb that has just completed swinging forward. The limb must attenuate impact forces arising from the abrupt transfer of body weight while remaining stable and allowing continued forward progression of the body.
      During single limb support, only the stance limb is in contact with the ground, and the limb must remain stable while allowing continued forward progression of the body over the foot.
      Swing limb advancement includes the phase when weight is being transferred from the reference limb to the opposite limb and the entire reference limb swing period. During swing limb advancement, the foot must clear the ground to ensure forward progression.

8. Describe the key motions and muscular activity patterns at the ankle, knee, and hip during weight acceptance.
   At the beginning of weight acceptance, the ankle is positioned in neutral, the knee observationally appears to be fully extended (it is actually in 5 degrees of flexion), and the hip is flexed approximately 20 degrees (relative to vertical) in the sagittal plane. These combined joint positions allow the heel to be the first part of the foot to contact the ground. During weight acceptance, as the foot positions itself flat on the ground, the ankle moves into 5 degrees of plantar flexion, controlled by eccentric activity
of the dorsiflexors. The knee moves into 20 degrees of flexion, controlled by eccentric activity of the quadriceps. The hip remains in 20 degrees of flexion, primarily owing to isometric activity of the single joint hip extensors.

9. Describe the key motions and muscular activity patterns at the ankle, knee, and hip during single limb support.

Movement of the ankle from 5 degrees of plantar flexion to 10 degrees of dorsiflexion is controlled by eccentric activity of the calf. The knee moves from 20 degrees of flexion to what observationally appears to be full extension (actually 5 degrees of flexion by motion analysis), in part as a result of concentric activity of the quadriceps (early single limb support) in combination with passive stability achieved when the ground reaction force vector moves anterior to the knee joint (late single limb support). The hip moves from 20 degrees of flexion to 20 degrees of apparent hyperextension (a combination of full hip extension, anterior pelvic tilt, and backward pelvic rotation), in part as a result of concentric activity of the single joint hip extensors (early single limb support) in combination with passive stability achieved when the ground reaction force vector moves posterior to the hip joint.

10. Describe the key motions and muscular activity patterns at the ankle, knee, and hip during swing limb advancement.

Initially, as the more proximal joints begin to flex, the foot remains in contact with the ground and the ankle moves passively into a position of 15 degrees of plantar flexion. Once the foot lifts from the ground, the ankle moves to neutral dorsiflexion owing to concentric activity of the pretibial muscles. The knee initially moves into 40 degrees of flexion (while the foot is still on the ground) primarily as a result of passive forces. As the foot lifts from the ground, the knee moves into 60 degrees of flexion, owing to concentric activity of knee flexors (biceps femoris short head, gracilis, and sartorius). During late swing limb advancement, the knee fully extends, in part as a result of momentum and quadriceps activity. The hip moves from 20 degrees of apparent hyperextension to 25 degrees of flexion by the middle of swing because of a combination of hip flexor muscle activity and momentum. In late swing, hip flexion decreases to 20 degrees as the hamstrings decelerate further progression of the leg.

11. What factors contribute to shock absorption during weight acceptance?

- Eccentrically controlled knee flexion to 20 degrees allows for dissipation of forces generated by the abrupt transfer of body weight onto the limb.
- Movement of the foot into 5 degrees of eversion functions to unlock the midtarsal joints (talonavicular and calcaneocuboid), creating a more flexible foot that is able to adapt to uneven surfaces. The rate of this motion is controlled by eccentric activity of the tibialis anterior and posterior.

12. What allows for stance stability during single limb support?

- Stability arises primarily from the action of the calf muscles that restrain excess forward collapse of the tibia. As a result, the knee and hip are able to achieve a fully extended position with only minimal muscle activation.
- In late single limb support, a reduction in the amount of subtalar joint eversion functions to lock the midtarsal joints and creates a rigid forefoot over which body weight progresses.

13. What allows for foot clearance during swing limb advancement?

- Early in swing limb advancement, knee flexion to 60 degrees (owing to passive and active factors) assists in clearing the limb.
- As swing limb advancement progresses, hip flexion to 25 degrees, in combination with ankle dorsiflexion to neutral, becomes critical to achieve foot clearance.

14. Name the key factors that are essential to ensure forward progression during the gait cycle.

- Forward progression during weight acceptance results primarily from eccentric activity of the dorsiflexors, which not only lower the foot to the ground but also draw the tibia forward.
- During single limb support, controlled tibial progression resulting from eccentric calf activity allows forward progression without tibial collapse.
- The 20 degrees of apparent hyperextension achieved at the hip contributes to a trailing limb posture that increases step length and forward progression.
- As body weight unloads rapidly from the trailing limb during preswing, elastic recoil energy stored in the Achilles tendon during single limb support is released and the tibia is driven forward.
During swing limb advancement, knee extension and hip flexion to 20 degrees in late swing contribute to forward progression and step length.

15. Describe the role of the heel, ankle, forefoot, and toe “rockers” during gait.
Collectively, the four rockers reflect a combination of joint motions and muscle actions that contribute to the smooth transition of body weight from the heel to the forefoot during stance. The heel rocker occurs during weight acceptance. Eccentric activity of the pretibial muscles lowers the forefoot to the ground and draws the tibia forward, allowing body weight to roll across the heel. Next is the ankle rocker, occurring during the first half of single limb support. The ankle moves from 5 degrees of plantar flexion to slight dorsiflexion. A gradual increase in eccentric calf muscle activity allows the ankle to remain stable as body weight progresses in front of the ankle. The forefoot rocker occurs during the last half of single limb support. A modulated increase in eccentric calf muscle activity permits the ankle to move into 10 degrees of dorsiflexion (without collapsing) and the heel to rise. Body weight smoothly transitions across the forefoot. The toe rocker occurs during preswing. As body weight shifts to the opposite limb, elastic recoil energy stored in the Achilles tendon during single limb support plantar flexes the ankle. The tibia advances forward over the medial forefoot and great toe.

16. What is the functional significance of normal subtalar joint eversion/inversion during the stance phase of gait?
During weight acceptance, subtalar eversion is important for unlocking the midtarsal joints (calcaneocuboid and talonavicular) and creating a more flexible foot that is able to adapt to uneven surfaces. During single limb support, a reduction in the amount of subtalar eversion (motion toward inversion) functions to lock the midtarsal joints, creating a rigid forefoot lever over which the body weight can progress.

17. What effects would a weak tibialis anterior have on gait?
• Foot slap immediately after initial contact (lack of eccentric control)
• Footdrop during swing
• Excessive hip and knee flexion (steppage gait) to clear the toes during swing

18. Describe gait deviations that likely would be evident in a patient with plantar fasciitis or a heel spur.
Patients typically exhibit a forefoot initial contact, avoiding the pressure associated with heel impact during weight acceptance. As the plantar fascia becomes tight with the combination of heel rise and metatarsal-phalangeal joint dorsiflexion during late stance, patients may avoid this posture by prematurely unweighting the limb.

19. What are the consequences of a triple arthrodesis on gait function?
• Loss of subtalar joint motion results in reduced shock absorption during weight acceptance.
• The inability to supinate in terminal stance diminishes the forefoot rocker effect.
• The ability to progress beyond the supporting foot is compromised.
• Stride length is diminished.

20. Describe the effect of calf weakness on ankle function during gait.
Calf weakness results in the inability to control forward advancement of the tibia, causing excessive dorsiflexion during single limb support and a lack of heel rise during late stance. As a result of the inability to control the tibia through eccentric action, the tibia advances faster than the femur, causing knee flexion during stance. The flexed knee posture necessitates activity of the quadriceps muscles, which normally are quiescent during single limb support.

21. Describe the effect of a plantar flexion contracture on ankle function during gait.
A plantar flexion contracture (>15 degrees) results in either a flat-foot or a forefoot-initial contact. This disrupts normal advancement of the tibia and may limit the knee from flexing to dissipate the forces associated with weight acceptance. During single limb support, the primary limitation is the inability to progress over the foot. Because 10 to 15 degrees of ankle dorsiflexion is necessary for normal stance phase function, compensatory mechanisms are necessary. Progression may be augmented through a premature heel rise, forward trunk lean, knee hyperextension, or a combination thereof. The inability to achieve a neutral ankle position during swing also necessitates compensatory movements to ensure foot clearance.
22. What are the characteristics of quadriceps avoidance?
Quadriceps avoidance manifests as reduced knee flexion during weight acceptance. This compensatory strategy results in decreased quadriceps demand and diminished muscular forces acting across the knee.

23. With what orthopedic conditions could quadriceps avoidance be associated?
- Patellofemoral pain
- Anterior cruciate ligament deficiency
- Quadriceps weakness
- Quadriceps inhibition (owing to pain or effusion)

24. Discuss the penalty associated with a knee flexion contracture.
A knee flexion contracture (>20 degrees) results in excessive knee flexion during weight acceptance, during single limb support, and at the end of swing limb advancement. The penalties include altered shock absorption during weight acceptance and instability during single limb support. Excessive knee flexion during stance requires greater amounts of quadriceps activity to support the flexed knee posture, increasing the energy cost of gait. Excess knee flexion at the end of swing limb advancement shortens step length.

25. Name typical compensatory strategies associated with reduced knee flexion range of motion.
Hip hiking or circumduction on the affected side is necessary to clear the foot during swing.

26. What is the penalty associated with reduced knee flexion range of motion?
The muscle activity associated with compensatory strategies increases the energy cost of gait.

27. What is a Trendelenburg gait pattern?
It is a contralateral pelvic drop during single limb support, usually caused by weakness of the ipsilateral hip abductors.

A lateral trunk lean to the same side as the weakness functionally serves to move the body center of mass over the involved hip, reducing the demand on the ipsilateral hip abductors.

29. Discuss the penalty associated with a hip flexion contracture.
A hip flexion contracture results in inadequate hip extension during late stance. Failure to obtain a trailing limb posture during late stance limits forward progression and stride length. To compensate for the lack of hip extension, an anterior pelvic tilt may be employed.

30. Explain the effect of hip extensor weakness on gait function.
Because adequate hip extensor strength is necessary to support the flexed hip posture during weight acceptance, reduced hip flexion is typically observed at initial contact, resulting in a reduced stride length. Also, a posterior trunk lean is commonly seen during weight acceptance as this posture reduces the external hip flexion moment and thus the demand on the hip extensors.

31. How does decreased proprioception influence gait?
Individuals with proprioceptive deficits (secondary to peripheral nerve injury, partial spinal cord injury, or brain lesions) require additional sensory input regarding joint position; typically this can be achieved through a forward trunk lean (to augment visual feedback) or through a more abrupt transfer of weight during the loading response (to augment sensory feedback).

32. How does an ankle fusion alter gait and energy consumption?
Persons who have sustained an ankle fusion often substitute for losses in talocrural joint motion (ie, dorsiflexion) by increasing midfoot and forefoot motion. This permits forward progression over the supporting foot in late stance. Stride length is often reduced, resulting in a slower walking velocity. Gait compensations resulting from an ankle fusion cause individuals to expend a slightly greater amount of energy during walking.
33. What are the energy costs of using various assistive devices (eg, crutches, standard walker, wheeled walker, cane) compared with using no equipment?

<table>
<thead>
<tr>
<th>ASSISTIVE DEVICE</th>
<th>ENERGY COST</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crutches</td>
<td>Energy demand increased 30%–80%, in part because of increased demands placed on arms and shoulder girdle muscles</td>
</tr>
<tr>
<td>Standard walker</td>
<td>Oxygen consumption increased &gt;200%</td>
</tr>
<tr>
<td>Front-wheeled walker</td>
<td>Less impact compared with standard walker</td>
</tr>
<tr>
<td>Cane</td>
<td>No significant contribution</td>
</tr>
</tbody>
</table>

34. How are energy costs of assistive devices affected by the presence of significant gait pathology?
When significant gait pathology is present (eg, excess ankle dorsiflexion and knee flexion secondary to a weak calf), use of an assistive device may lessen the energy demands of ambulation by reducing the demands on lower extremity muscles, allowing achievement of a more normal, energy-efficient gait pattern.

35. How does osteoarthritis of the knee influence gait?
- Patients walk with a slower velocity, owing to reductions in stride length and cadence.
- Many patients are not able to tolerate the demands of loading onto a flexed knee and may purposefully reduce loading response knee flexion.
- Many patients decrease knee flexion during early swing in an effort to limit painful joint movement.

36. How does the energy cost of walking with a total hip fusion compare with that of walking with a total hip arthroplasty?

**TOTAL HIP FUSION**
The average rate of oxygen consumption increases 32% compared with normal values at the same walking speed. Increased energy cost likely results from the compensations required for forward progression during gait (eg, excess lumbar lordosis and an anterior pelvic tilt to enable the fused hip to achieve a trailing limb posture in late stance).

**TOTAL HIP ARTHROPLASTY**
Energy expenditure (1 year postoperatively) is approximately 17% less compared with walking with a fused hip.

37. What influences do various levels of amputation have on walking velocity and energy cost?
- In persons with unilateral amputations, the more proximal the level of amputation (eg, transfemoral versus transtibial) the slower the customary walking speed and the greater the energy cost (milliliters of oxygen per kilogram of body weight per meter of walking) of walking.
- Energy expenditure, heart rate, and oxygen consumption are typically lower during ambulation with a prosthesis compared with ambulation with crutches.

38. What are common gait deviations in a person with a transtibial amputation?
- Limited dorsiflexion during single limb support
- Diminished plantar flexion in preswing
- Forward trunk lean
- Reduced knee flexion during weight acceptance
39. List the pros and cons of using an ankle-foot orthosis (AFO) for the treatment of footdrop.

<table>
<thead>
<tr>
<th>PROS</th>
<th>CONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assists with foot clearance</td>
<td>If AFO is too rigid, then during weight acceptance:</td>
</tr>
<tr>
<td>Reduces need for compensatory maneuvers</td>
<td>- Normal movement of ankle into plantar flexion is disrupted.</td>
</tr>
<tr>
<td></td>
<td>- Heel rocker effect is accentuated, resulting in increased knee flexion and greater quadriceps demand.</td>
</tr>
</tbody>
</table>

40. Compared with traditional nonmicroprocessor-controlled knee units, what impact do microprocessor-controlled knees have on ambulation for individuals with a transfemoral amputation?

Individuals with transfemoral amputations walk faster, more efficiently, and with greater stability when using a microprocessor-controlled knee unit than when using a traditional nonmicroprocessor-controlled prosthetic knee.

41. Describe the effect of hip abductor weakness on pelvis, hip, and knee motion during gait.

Hip abductor weakness results in the inability to control the hip/pelvis in the frontal plane during weight acceptance and single limb support. Common gait deviations associated with hip abductor weakness include a contralateral pelvic drop and/or excessive hip adduction. In addition, the adducted hip may place the knee in a valgus position during stance. A typical compensation for hip abductor weakness is to lean the trunk toward the stance limb to reduce the external frontal plane moment at the hip (compensated Trendelenburg pattern).

BIBLIOGRAPHY


CHAPTER 15 QUESTIONS

1. The range of normal walking velocities for adults is:
   a. 66–70 m/min
   b. 72–76 m/min
   c. 78–82 m/min
   d. 86–90 m/min

2. You observe excessive knee flexion, too much ankle dorsiflexion, and lack of a heel rise during the terminal stance phase of gait. The most likely cause of this combination of deviations is weakness of the:
   a. Plantar flexors
   b. Quadriceps
   c. Hamstrings
   d. Gluteals

3. Common gait deviations associated with weakness of the hip extensors include:
   a. Increased hip flexion during weight acceptance and a forward (anterior) trunk lean
   b. Decreased hip flexion during weight acceptance and a forward (anterior) trunk lean
   c. Increased hip flexion during weight acceptance and a backward (posterior) trunk lean
   d. Decreased hip flexion during weight acceptance and a backward (posterior) trunk lean

4. During stance, common gait deviations associated with hip abductor weakness include:
   a. Ipsilateral trunk lean, contralateral pelvic drop, and ipsilateral knee valgus
   b. Ipsilateral trunk lean, contralateral pelvic drop, and ipsilateral knee varus
   c. Contralateral trunk lean, ipsilateral pelvic drop, and ipsilateral knee varus
   d. Contralateral trunk lean, contralateral pelvic drop, and ipsilateral knee varus

5. The primary muscles contributing to stability during single limb support are the:
   a. Hip extensors
   b. Hip abductors
   c. Knee extensors
   d. Ankle plantar flexors
1. Summarize the properties of common opioid analgesics.

<table>
<thead>
<tr>
<th>GENERIC NAME</th>
<th>TRADE NAME</th>
<th>ADMINISTRATION ROUTES</th>
<th>ONSET OF ANALGESIC ACTION (MIN)</th>
<th>PEAK ANALGESIC EFFECT (MIN)</th>
<th>DURATION OF ANALGESIC ACTION (HR)</th>
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<td>Within 15</td>
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<td></td>
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<td>30–60</td>
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<td>Dilaudid,</td>
<td>Oral</td>
<td>30</td>
<td>30–90</td>
<td>4–5</td>
</tr>
<tr>
<td></td>
<td>Hydrostat</td>
<td>IM</td>
<td>15</td>
<td>30–60</td>
<td>4–5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IV</td>
<td>10–15</td>
<td>15–30</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sub-Q</td>
<td>15</td>
<td>30–90</td>
<td>4–5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rectal</td>
<td>15–30</td>
<td>30–90</td>
<td>4–5</td>
</tr>
<tr>
<td>Levorphanol</td>
<td>Levo-Dromoran</td>
<td>Oral</td>
<td>10–60</td>
<td>90–120</td>
<td>4–5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IV</td>
<td>Unknown</td>
<td>Within 20</td>
<td>4–5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sub-Q</td>
<td>Unknown</td>
<td>60–90</td>
<td>4–5</td>
</tr>
<tr>
<td>Meperidine</td>
<td>Demerol</td>
<td>Oral</td>
<td>15</td>
<td>60</td>
<td>2–4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IM</td>
<td>10–15</td>
<td>30–50</td>
<td>2–4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IV</td>
<td>Immediate</td>
<td>5–7</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sub-Q</td>
<td>10–15</td>
<td>40–60</td>
<td>2–4</td>
</tr>
<tr>
<td>Methadone</td>
<td>Methadose</td>
<td>Oral</td>
<td>30–60</td>
<td>90–120</td>
<td>4–12</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IM</td>
<td>10–20</td>
<td>60–120</td>
<td>4–6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sub-Q</td>
<td>10–20</td>
<td>60–120</td>
<td>4–6</td>
</tr>
<tr>
<td>Morphine</td>
<td>Duramorph,</td>
<td>Oral</td>
<td>Unknown</td>
<td>60</td>
<td>4–5</td>
</tr>
<tr>
<td></td>
<td>MS Contin, many others</td>
<td>IM</td>
<td>10–30</td>
<td>30–60</td>
<td>4–5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IV</td>
<td>Rapid</td>
<td>20</td>
<td>4–5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sub-Q</td>
<td>20</td>
<td>50–90</td>
<td>4–5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Epidural</td>
<td>6–30</td>
<td>60</td>
<td>up to 24</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intrathecal</td>
<td>Rapid</td>
<td>Unknown</td>
<td>up to 24</td>
</tr>
</tbody>
</table>
2. List the common NSAIDs and compare them.

### Common Nonsteroidal Antiinflammatory Drugs

<table>
<thead>
<tr>
<th>GENERIC NAME</th>
<th>TRADE NAME</th>
<th>SPECIFIC COMMENTS—COMPARISON WITH OTHER NSAIDS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>Many trade names</td>
<td>The original NSAID used for analgesic and antiinflammatory effects; also used frequently for antipyretic and anticoagulant effects</td>
</tr>
<tr>
<td>Diclofenac</td>
<td>Voltaren</td>
<td>Substantially more potent than naproxen and several other NSAIDs; may be more selective for the COX-2 isoenzyme than other NSAIDs; adverse side effects occur in 20% of patients</td>
</tr>
<tr>
<td>Diflunisal</td>
<td>Dolobid</td>
<td>Has potency three to four times greater than aspirin in terms of analgesic and antiinflammatory effects but lacks antipyretic activity</td>
</tr>
<tr>
<td>Etodolac</td>
<td>Lodine</td>
<td>Effective as analgesic/antiinflammatory agent with fewer side effects than most NSAIDs; may be more selective for the COX-2 isoenzyme than other NSAIDs; may have gastric-sparing properties</td>
</tr>
<tr>
<td>Fenoprofen</td>
<td>Nalfon</td>
<td>GI side effects fairly common but usually less intense than those occurring with similar doses of aspirin</td>
</tr>
<tr>
<td>Flurbiprofen</td>
<td>Ansaid</td>
<td>Similar to aspirin’s benefits and side effects; also available as topical ophthalmic preparation (Ocufen)</td>
</tr>
</tbody>
</table>

---

### Common Opioid Analgesics (Continued)

<table>
<thead>
<tr>
<th>GENERIC NAME</th>
<th>TRADE NAME</th>
<th>ADMINISTRATION ROUTES</th>
<th>ONSET OF ANALGESIC ACTION (MIN)</th>
<th>PEAK ANALGESIC EFFECT (MIN)</th>
<th>DURATION OF ANALGESIC ACTION (HR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nalbuphine</td>
<td>Nubain</td>
<td>IM</td>
<td>Within 15</td>
<td>60</td>
<td>3–6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IV</td>
<td>2–3</td>
<td>30</td>
<td>3–6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sub-Q</td>
<td>Within 15</td>
<td>Unknown</td>
<td>3–6</td>
</tr>
<tr>
<td>Oxycodeine</td>
<td>OxyContin, Roxicodone</td>
<td>Oral</td>
<td>10–15</td>
<td>60–90</td>
<td>3–6</td>
</tr>
<tr>
<td>Oxymorphone</td>
<td>Opana</td>
<td>Oral</td>
<td>Unknown</td>
<td>Unknown</td>
<td>4–6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IM</td>
<td>10–15</td>
<td>30–90</td>
<td>3–6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IV</td>
<td>5–10</td>
<td>15–30</td>
<td>3–6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sub-Q</td>
<td>10–20</td>
<td>Unknown</td>
<td>3–4</td>
</tr>
<tr>
<td>Pentazocine</td>
<td>Talwin</td>
<td>Oral</td>
<td>15–30</td>
<td>60–90</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IM</td>
<td>15–20</td>
<td>30–60</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IV</td>
<td>2–3</td>
<td>15–30</td>
<td>2–3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sub-Q</td>
<td>15–20</td>
<td>30–60</td>
<td>2–3</td>
</tr>
<tr>
<td>Propoxyphene</td>
<td>Darvon</td>
<td>Oral</td>
<td>15–60</td>
<td>120–180</td>
<td>4–6</td>
</tr>
</tbody>
</table>

*IM, intramuscular; IV, intravenous; Sub-Q, subcutaneous*

(Information adapted with permission from Ciccone CD: Davis’s Drug Guide for Rehabilitation Professionals. Philadelphia, 2013, FA Davis)
3. How do opioid analgesics decrease pain?
Opioids bind to specific neuronal receptors located at synapses in the brain and spinal cord. These synapses are responsible for transmitting painful sensations from the periphery to the brain. Opioid drugs bind to protein receptors on the presynaptic terminal of these synapses and inhibit the release of pain-mediating chemicals, such as substance P. Opioids also bind to receptors on the postsynaptic neuron and cause hyperpolarization, which decreases the excitability of the postsynaptic neuron. These drugs limit the ability of the central nervous system synapses to transmit painful sensations to the brain.

Opioids can also activate descending CNS pathways that suppress pain at the spinal cord level. These pathways originate in the midbrain and travel caudally to the dorsal horn of the spinal cord where they release neurotransmitters, such as norepinephrine and serotonin, onto synapses that normally mediate painful impulses. By suppressing these synapses, afferent pain impulses that enter the dorsal horn are not transmitted rostrally to the pain centers of the brain. Thus opioids appear to exert some of their analgesic effects by activating these descending pathways, thereby suppressing afferent pain impulses reaching the spinal cord.

Common Nonsteroidal Antiinflammatory Drugs (Continued)

<table>
<thead>
<tr>
<th>GENERIC NAME</th>
<th>TRADE NAME</th>
<th>SPECIFIC COMMENTS—COMPARISON WITH OTHER NSAIDS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ibuprofen</td>
<td>Motrin, many others</td>
<td>First nonaspirin NSAID also available in nonprescription form; fewer GI side effects than aspirin but GI effects still occur in 5%–15% of patients</td>
</tr>
<tr>
<td>Indomethacin</td>
<td>Indocin</td>
<td>Relatively high incidence of dose-related side effects; problems occur in 25%–50% of patients</td>
</tr>
<tr>
<td>Ketoprofen</td>
<td>Orudis, Oruvail, others</td>
<td>Similar to aspirin’s benefits and side effects, but has a relatively short half-life (1–2 hrs)</td>
</tr>
<tr>
<td>Ketorolac</td>
<td>Toradol</td>
<td>Can be administered orally or by intramuscular injection; parenteral doses provide postoperative analgesia equivalent to opioids</td>
</tr>
<tr>
<td>Meclofenamate</td>
<td>Meclomen</td>
<td>No apparent advantages or disadvantages compared with aspirin and other NSAIDs</td>
</tr>
<tr>
<td>Meloxicam</td>
<td>Mobic</td>
<td>Relatively fewer gastric side effects than piroxicam; may be more selective for the COX-2 isoenzyme than other NSAIDs</td>
</tr>
<tr>
<td>Nabumetone</td>
<td>Relafen</td>
<td>Effective as analgesic/antiinflammatory agent with fewer side effects than most NSAIDs</td>
</tr>
<tr>
<td>Naproxen</td>
<td>Anaprox, Naprosyn, others</td>
<td>Similar to ibuprofen in terms of benefits and adverse effects</td>
</tr>
<tr>
<td>Oxaprozin</td>
<td>Daypro</td>
<td>Analgesic and antiinflammatory effects similar to aspirin; may produce fewer side effects than other NSAIDs</td>
</tr>
<tr>
<td>Piroxicam</td>
<td>Feldene</td>
<td>Long half-life (45 hr) allows once daily dosing; may be somewhat better tolerated than aspirin</td>
</tr>
<tr>
<td>Sulindac</td>
<td>Clinoril</td>
<td>Relatively little effect on kidneys (renal sparing) but may produce more GI side effects than aspirin</td>
</tr>
<tr>
<td>Tolmetin</td>
<td>Tolectin</td>
<td>Similar to aspirin’s benefits and side effects but must be given frequently (four times daily) because of its short half-life (1 hr)</td>
</tr>
</tbody>
</table>

GI, gastrointestinal
(From Ciccone CD: Pharmacology in Rehabilitation, ed 5, Philadelphia, 2015, FA Davis.)
Finally, opioid drugs may affect neurons outside of the central nervous system. Opioid receptors have been identified on the distal ends of peripheral sensory neurons that transmit pain. Opioid drugs can bind to these peripheral receptors and decrease pain sensation by decreasing the sensitivity of nociceptive nerve endings.

4. Discuss side effects of opioids that can be especially troublesome in patients receiving physical therapy.
Sedation and mood changes (eg, confusion, euphoria, dysphoria) can be bothersome because patients receiving physical therapy may be less able to understand instructions and participate in therapy sessions. Opioid drugs cause respiratory depression because they decrease the sensitivity of the respiratory control center in the brainstem. Although respiratory depression is not especially troublesome at therapeutic doses, this side effect can be serious or fatal if patients overdose on opioid medications. Orthostatic hypotension (a decrease in blood pressure when the patient becomes more upright) may occur during opioid use, and therapists should look for signs of dizziness and syncope, especially during the first 2 to 3 days after a patient begins taking opioid analgesics. Opioids are associated with several gastrointestinal side effects, including nausea, cramps, and vomiting. Constipation may also occur, and this side effect can be a serious problem if these drugs are used for extended periods in people who are susceptible to fecal impaction (eg, people with spinal cord injuries).

5. Does long-term opioid use always result in addiction?
No. Addiction is characterized by tolerance (the need to increase drug dosage progressively to achieve therapeutic effects) and physical dependence (onset of withdrawal when the drug is discontinued suddenly). Although indiscriminate or excessive use of opioids can lead to addiction, tolerance and physical dependence do not necessarily occur when these agents are used appropriately for the treatment of pain. Appropriate use means that the drug dosage matches the patient’s pain as closely as possible. If the dosage is adjusted carefully to meet each patient’s needs, these drugs can be used for extended periods (several weeks to several months) without the patient developing tolerance and physical dependence.

6. Can opioids increase pain in certain patients?
Yes. Certain patients may experience increased pain after taking opioids, a phenomenon known as opioid-induced hyperalgesia (OIH). This phenomenon is not fully understood but seems to occur because opioids activate, rather than suppress, CNS nociceptive pathways in certain patients. In these patients, opioid use can increase the effects of excitatory neurotransmitters such as glutamate on a specific receptor known as the N-methyl-D-aspartate receptor. Activation of this receptor can increase the activity of pain pathways, thus increasing pain perception. It is not clear why OIH occurs in only certain patients, and susceptibility is probably related to genetic factors that predispose these patients to this effect. Nonetheless, clinicians should be aware that pain may not decrease and may actually increase in certain patients when opioids are administered. Pain should therefore be monitored carefully, especially within the first hour or so after the patient receives a dose of the opioid. An increase in pain may represent OIH, and this response should be reported to the physician.

7. List advantages of using a patient-activated electronic drug delivery system, known commonly as patient-controlled analgesia (PCA), to administer opioids.
- Increases patient satisfaction because the patient feels more in control of his or her ability to manage pain
- Provides more consistent pain control while avoiding many of the side effects associated with excessive amounts of opioids

8. Describe the disadvantages of using PCA to administer opioids.
One disadvantage is the inability of some patients to understand fully how to use the PCA device. For example, patients with cognitive problems or unreasonable fear of addiction may not understand that they must activate the PCA device when they feel pain. Other disadvantages include human error in programming the PCA device (the PCA pump can be programmed incorrectly and overdose or underdose the patient) and various technical problems (pump failure, displacement or blockage of intravenous catheters).
9. List the primary effects of NSAIDs.
   - Decreased pain (analgesia)
   - Decreased inflammation (antiinflammatory)
   - Decreased fever (antipyresis)
   - Decreased blood clotting (anticoagulation)

10. How do NSAIDs exert their primary beneficial effects?
    NSAIDs work by inhibiting the synthesis of prostaglandins. Prostaglandins are lipid-like compounds that are synthesized by cells throughout the body. These compounds help regulate normal cell activity, and they are synthesized as a part of the cellular response to injury. Prostaglandins can increase sensitivity to pain, help promote inflammation, raise body temperature during fever, and increase platelet aggregation and platelet-induced clotting. Prostaglandin biosynthesis is catalyzed within the cell by the cyclooxygenase (COX) enzyme. This enzyme transforms a 20-carbon precursor (arachidonic acid) into the first prostaglandin (PGG2). Cells then use PGG2 to form various other prostaglandins depending on their physiologic status and whether or not they are injured. By acting as a potent inhibitor of the COX enzyme, NSAIDs block the production of all prostaglandins in the cell.

11. How do prescription NSAIDs differ from nonprescription (over-the-counter) NSAIDs?
    When used to treat pain and inflammation, prescription NSAIDs do not differ appreciably from an equivalent dose of a nonprescription product. The recommended dosage of nonprescription NSAIDs may be relatively lower than prescription NSAIDs. The major difference between prescription and over-the-counter NSAIDs is their cost; prescription products may be substantially more expensive than their nonprescription counterparts.

12. Discuss potential problems associated with the long-term use of NSAIDs.
    NSAIDs are relatively safe when taken at recommended doses for long periods (eg, several weeks or months). The most common side effect associated with these drugs is gastric irritation. Most NSAIDs inhibit the production of prostaglandins that help protect the gastric mucosa, and loss of these beneficial prostaglandins renders the mucosa vulnerable to damage from gastric acids. This problem can be minimized by taking each dose with food or by administering NSAIDs with other medications (proton pump inhibitors, histamine type-2 receptor blockers) that reduce the effects or secretion of gastric acid. Other potential problems during long-term use include hepatic and renal toxicity. These problems are especially prevalent if other risk factors are present, including preexisting liver and kidney dysfunction, excessive alcohol consumption, and excessive or unnecessary use of other prescription drugs. NSAIDs probably should not be used for extended periods in people who have one or more of these risk factors.

13. Can NSAIDs inhibit healing of bone and soft tissues?
    As indicated in question 10, NSAIDs inhibit prostaglandin biosynthesis. Certain prostaglandins, however, appear to be important during bone healing because these prostaglandins increase the activity of osteoblasts and osteoclasts that promote new bone formation. It follows that NSAIDs could impair bone healing by depriving bone of these important prostaglandins. As indicated in a review by Harder and An, much of the evidence for this detrimental effect has been derived from studies using laboratory animals and in vitro cellular models. Nonetheless, retrospective clinical studies have found a significant relationship between NSAID use and nonunion of the femoral diaphysis; a relationship was also observed in patients who used NSAIDs postoperatively after spinal fusion surgery compared with patients who did not use these drugs. Consequently, many clinicians feel it is prudent to avoid use of NSAIDs immediately after fracture and bone surgery.

    The effects of NSAIDs on soft tissue healing (cartilage, tendons, ligaments, skin) remain unclear. A recent critical review by Chen and Dragoo concluded that short-term (less than 2-week) administration of low-dose traditional NSAIDs or COX-2 selective drugs does not adversely affect soft tissue healing in humans. Laboratory studies have suggested that these drugs may even increase the healing of cartilage, tendons, and ligaments in certain animal models. The effects of NSAIDs, however, on the growth and healing of soft tissues in humans are difficult to determine and likely depend on many other variables such as the exact drug, the dose and timing of treatment, and the type of soft tissue injury. Because of their antiinflammatory and analgesic effects, NSAIDs remain an important and popular treatment for soft tissue injuries and seem relatively safe when used for short periods of time after the injury.

14. What are the COX-2 inhibitors?
    These are drugs that inhibit a specific subtype of the COX enzyme. There are two major subtypes of this enzyme known as COX-1 and COX-2. The COX-2 subtype is produced within various cells that are injured...
or damaged, and the COX-2 enzyme synthesizes prostaglandins associated with pain and inflammation. Drugs that are more selective for the COX-2 enzyme can help control production of prostaglandins that cause pain and inflammation, while sparing the production of beneficial prostaglandins, including the prostaglandins that help protect the stomach lining.

15. Give an example of a COX-2 inhibitor and its benefits.
Celecoxib (Celebrex) may decrease pain and inflammation similar to the traditional NSAIDs, with less chance of causing gastric irritation in some patients. An additional benefit is that this drug does not inhibit platelet function and therefore does not need to be stopped before surgery to prevent bleeding complications. Celecoxib is the only COX-2 inhibitor that is available in the United States at the present time.

16. Are COX-2 inhibitors safe?
COX-2 inhibitors can produce side effects such as headache, abdominal pain, and diarrhea. Moreover, there is concern that these drugs may increase the risk of serious cardiovascular problems, including heart attack and stroke. For this reason, two of the original COX-2 drugs, rofecoxib (Vioxx) and valdecoxib (Bextra), were withdrawn from the market. Research continues to determine whether COX-2 drugs such as celecoxib have an acceptable risk-benefit ratio in certain patients. It seems reasonable that people who are at risk for cardiovascular disease should use these drugs cautiously or perhaps avoid them altogether.

17. How is acetaminophen different from the NSAIDs?

<table>
<thead>
<tr>
<th>Acetaminophen versus NSAIDs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SIMILARITIES</strong></td>
</tr>
<tr>
<td>Analgesic effects</td>
</tr>
<tr>
<td>Antipyretic effects</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

18. Does acetaminophen have any side effects?  
Yes. Liver toxicity is the major side effect, especially if high doses are taken or the patient already has some degree of liver failure.

19. Can analgesics be applied topically or transdermally to decrease pain?  
Certain analgesics can be applied to the skin to treat pain in fairly superficial structures. Trolamine salicylate (an aspirin-like drug) is available in several over-the-counter creams; this drug penetrates the skin and decreases pain in underlying tissues, such as muscle and tendon. Penetration of trolamine and certain other NSAIDs (ketoprofen) can be enhanced by ultrasound (phonophoresis) or by electric current (iontophoresis).

Local anesthetics such as lidocaine can be administered transdermally via a medicated patch that is placed directly over the painful area. Again, the intent is to focus the drug into the tissues directly beneath the patch. Certain opioids, including morphine and fentanyl, can also be administered transdermally. The goal of this administration is to achieve systemic levels that ultimately reach the central nervous system rather than to treat a specific subcutaneous structure or tissue. The use of opioid patches or other transdermal techniques (including iontophoresis) may offer a noninvasive way to provide fairly sustained administration and pain relief with opioid medications.

Several other agents can be applied topically and are often available in over-the-counter products. Some products contain capsaicin, a chemical derived from chili peppers that produces a burning sensation when applied to the skin via creams and lotions. This burning sensation may act initially as a counterirritant to override other painful impulses being transmitted along sensory pathways. Repeated use of capsaicin may also deplete sensory neurons of pain-mediating neurotransmitters, thus decreasing the ability of nociceptive neurons to relay painful information toward the CNS. Topical products can also contain other counterirritants such as menthol, camphor, or a combination of these and other chemicals. These products may offer temporary relief of musculoskeletal pain in some patients. Occasional use is not usually harmful, but excessive use should probably be discouraged.

20. Are medications from other drug categories effective in treating chronic pain?  
Yes. Traditional antidepressants such as nortriptyline (Aventyl, Pamelor) and amitriptyline (Elavil) and some of the newer antidepressants such as paroxetine (Paxil) and venlafaxine (Effexor) are often incorporated in the
analgesic regimen for people with various types of chronic pain such as fibromyalgia and chronic back and neck pain. In some patients, depression may be present along with chronic pain, so it seems reasonable that managing the depression will help provide better outcomes when also trying to manage pain. There is evidence, however, that antidepressants can help improve pain even when a patient is not clinically depressed. Antidepressants prolong the activity of neurotransmitters in the brain such as norepinephrine, dopamine, and serotonin. It follows that their analgesic effects are probably related to their ability to affect these same neurotransmitters, but the exact reason they are effective in treating pain remains to be determined.

Certain antiseizure drugs such as gabapentin (Neurontin) and pregabalin (Lyrica) are also helpful in treating chronic pain, especially neuropathic pain. Again, the reason for their analgesic effects is not clear, but it seems to be related to the ability of these drugs to enhance the inhibitory effects of γ-aminobutyric acid (GABA) throughout the brain. That is, gabapentin and other antiseizure drugs might decrease neuronal excitability in central pain pathways, thereby reducing the sensitivity of neurons involved in pain perception.

21. What are the two primary categories of antiinflammatory medications? NSAIDs and antiinflammatory steroids (glucocorticoids) are the two major types of antiinflammatory medications.

22. List the common glucocorticoids and their antiinflammatory activity.

<table>
<thead>
<tr>
<th>Common Glucocorticoids</th>
<th>ANTIINFLAMMATORY DOSE (MG)*</th>
<th>RELATIVE ANTIINFLAMMATORY ACTIVITY†</th>
</tr>
</thead>
<tbody>
<tr>
<td>GENERIC NAME</td>
<td>TRADE NAME</td>
<td></td>
</tr>
<tr>
<td>SHORT ACTING</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortisone</td>
<td>Cortone</td>
<td>25–300</td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>Cortef, others</td>
<td>20–240</td>
</tr>
<tr>
<td>INTERMEDIATE ACTING</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Methylprednisolone</td>
<td>Medrol, others</td>
<td>2–60</td>
</tr>
<tr>
<td>Prednisolone</td>
<td>Prelone, others</td>
<td>5–60</td>
</tr>
<tr>
<td>Prednisone</td>
<td>Sterapred</td>
<td>5–60</td>
</tr>
<tr>
<td>Triamcinolone</td>
<td>Aristospan, others</td>
<td>8–16</td>
</tr>
<tr>
<td>LONG ACTING</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Betamethasone</td>
<td>Celestone</td>
<td>0.6–7.2</td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>DexPak</td>
<td>0.75–9.0</td>
</tr>
</tbody>
</table>

*Typical daily adult and adolescent dose, administered orally in single or divided doses.
†Antiinflammatory potency relative to hydrocortisone (eg, prednisone is four times more potent than hydrocortisone).
‡Duration of activity related to tissue half-life (ie, short acting, tissue half-life 8–12 hrs; intermediate acting, tissue half-life 18–36 hrs; long acting, tissue half-life 36–54 hrs).
(Data from Klasco RK, editor: USP DI drug information for the health care professional, vol 1, ed 24, Greenwood Village, Colo., 2004, Thompson Healthcare.)

23. How do glucocorticoids decrease inflammation? Glucocorticoids enter the cell, bind to a specific receptor in the cytoplasm, and form a glucocorticoid-receptor complex that moves to the cell’s nucleus. In the nucleus, the drug-receptor complex increases the transcription of genes that code for antiinflammatory proteins (eg, certain interleukins, neutral endopeptidase) while inhibiting genes that code for inflammatory proteins (eg, cytokines, inflammatory enzymes). Glucocorticoids also inhibit directly the function of various cells involved in the inflammatory response, including macrophages, lymphocytes, and eosinophils.

24. How do glucocorticoids compare with NSAIDs in terms of efficacy and safety? Glucocorticoids are generally much more effective in reducing inflammation compared with NSAIDs, but glucocorticoids are not as safe as NSAIDs, and glucocorticoid use can produce several serious side effects when administered systemically for periods of 3 weeks or more.
25. Discuss the serious side effects of glucocorticoids.

Glucocorticoids can cause hypertension, muscle wasting, glucose intolerance, gastric ulcers, and glaucoma. Patients may be more prone to infections because these drugs suppress the immune system. Prolonged glucocorticoid administration causes adrenocortical suppression, in which the adrenal gland stops synthesizing endogenous glucocorticoids (cortisol) because of the negative feedback effect of the drugs on the endocrine system. Because it takes the adrenal gland several days to regain normal function and begin synthesizing cortisol, adrenocortical suppression can be life-threatening if the glucocorticoid drug is suddenly discontinued. Consequently, patients who receive systemic doses of glucocorticoids for extended periods should not discontinue these medications suddenly but should gradually taper off the dosage under medical supervision.

26. Can delivery of antiinflammatory steroids via iontophoresis or phonophoresis cause adrenocortical suppression?

Iontophoresis or phonophoresis, when applied to a single joint or tissue and used at a reasonable frequency (ie, 3 or 4 times each week), does not pose a serious threat for causing adrenocortical suppression.

27. Which side effect of glucocorticoids can be especially troublesome in patients receiving physical therapy?

One of the most troublesome side effects of glucocorticoids is the tendency of these drugs to cause breakdown (catabolism) of muscle, tendon, bone, and other supporting tissues.

28. How can the catabolic side effects of glucocorticoids be managed?

Catabolic side effects can be managed by subjecting muscle and other tissues to resistance exercise. For example, renal transplant patients receiving glucocorticoids to prevent organ rejection were trained using an isokinetic cycle ergometer, and these patients experienced an increase in thigh girth and thigh muscle area of 9% to 44% compared with healthy control subjects. This relative protection against muscle atrophy is variable and depends on the type and intensity of the exercise, the dosage of the glucocorticoid, and the amount of catabolism that may already be present because of high glucocorticoid dosage and prolonged administration. Nonetheless, judicious use of progressive resistance training and other strengthening techniques (eg, walking, aquatic exercise) can be invaluable in minimizing the catabolic side effects.

29. Is there a critical dosage or frequency of administration that contraindicates further intraarticular injections of glucocorticoids?

A given joint should receive no more than four injections within a 12-month period.

30. What are the fluoroquinolones?

They are a group of antibacterial drugs that includes ciprofloxacin (Cipro) and ofloxacin (Floxin). These drugs have a fairly broad antibacterial spectrum and are used frequently to treat urinary tract infections, respiratory tract infections, and other infections caused by gram-negative bacteria.

31. Why are fluoroquinolones potentially harmful to patients with orthopedic conditions?

Some patients experience tendinopathy (pain, tenderness), especially in the Achilles tendon and other large tendons that are subjected to high amounts of stress. The exact reasons for this effect are unclear, but fluoroquinolone-induced tendinopathy can be severe and lead to tendon rupture. Risk factors include advanced age, renal failure, use of glucocorticoids, and a history of tendinopathy caused by these drugs. Therapists should be especially cognizant of tendinitis in patients who are taking these drugs, and any increase in tendon problems should be brought to the attention of the medical staff. Exercise involving the affected tendon should be discontinued until the source of the pain and tenderness can be evaluated.

32. What medications are available to treat skeletal muscle spasms associated with orthopedic impairments (eg, nerve root impingement or direct injury to the muscle)?

Diazepam (Valium) and a diverse group of drugs such as carisoprodol (Soma), cyclobenzaprine (Flexeril), and other centrally acting muscle relaxants are available to treat these conditions. The drugs commonly used to control muscle spasms act on the central nervous system and attempt to reduce excitatory input onto the α-motor neuron. Valium increases the inhibitory effects of GABA in the spinal cord. GABA, an inhibitory neurotransmitter in the central nervous system, tends to decrease neuronal activity, including the activity of the α-motor neuron that activates skeletal muscle. Valium increases
GABA-mediated inhibition of the α-motor neuron, which, in turn, causes decreased muscle activation, with subsequent relaxation of muscles that are in spasm. The actions of other centrally acting muscle relaxants are poorly understood. Carisoprodol, for example, may affect GABA receptors in a manner similar to diazepam, whereas cyclobenzaprine may increase the inhibitory effects of serotonin in the brainstem. On the other hand, all the centrally acting muscle relaxants cause sedation, and it seems likely that any muscle relaxant properties of these drugs are caused by their sedative effects.

33. Discuss the efficacy of the drugs commonly used to treat skeletal muscle spasm. Antispasm drugs typically have been shown to be more effective than placebo in reducing the pain associated with skeletal muscle spasms. These drugs, however, may not be any more effective than simple analgesic medications (eg, NSAIDs, acetaminophen) when treating orthopedic conditions that include spasms. All of the commonly prescribed antispasm drugs cause sedation, and the ability of these drugs to relax skeletal muscle is probably related more to their sedative properties than to a direct effect on muscle spasms. Many practitioners are foregoing use of these muscle relaxants in lieu of pain medications and other nonpharmacologic interventions, including physical therapy.

34. How do antispasm medications differ from drugs used to treat spasticity? Antispasm medications consist primarily of diazepam (Valium) and other drugs that act in the central nervous system and attempt to decrease excitation of the α-motor neuron. Diazepam also can be used to treat spasticity (ie, increased stretch reflex activity secondary to central nervous system lesions). The other traditional antispasm drugs (eg, carisoprodol, cyclobenzaprine) are typically used only for spasms. Antispasticity drugs, including baclofen (Lioresal), tizanidine (Zanaflex), gabapentin (Neurontin), dantrolene (Dantrium), and botulinum toxin (Botox) act at various sites to decrease the hyperexcitability of skeletal muscle. Baclofen, tizanidine, and gabapentin act within the spinal cord to increase inhibition and decrease excitation of the α-motor neuron. Dantrolene acts directly on the skeletal muscle cell and causes relaxation by inhibiting the release of calcium from the sarcoplasmic reticulum. Botulinum toxin can be injected directly into spastic muscles and causes relaxation by inhibiting the release of acetylcholine at the neuromuscular junction. Botulinum toxin can be used to treat severe, chronic muscle spasms in conditions such as torticollis.

35. What are the primary medications used to treat osteoarthritis? Acetaminophen and NSAIDs are the primary medications used in the treatment of osteoarthritis. NSAIDs can be used as an alternative or as a supplement to acetaminophen, especially in more advanced stages of osteoarthritis where some inflammation (synovitis) may occur secondary to other degenerative changes in the joints. Other drugs can be used to help restore joint function and prevent further degeneration. Viscosupplementation involves injection of hyaluronan directly into the joint in an attempt to restore viscosity of the synovial fluid. Another strategy uses over-the-counter dietary supplements, such as glucosamine and chondroitin sulfate, to provide substrates for the formation of healthy articular cartilage and synovial fluid.

36. Is there evidence that dietary supplements (eg, glucosamine, chondroitin) can improve joint function in people with osteoarthritis? There is limited evidence to support the use of dietary supplements containing glucosamine, chondroitin, or a combination of both products. Although some early studies suggested these supplements might decrease joint pain and increase function in osteoarthritis, many of these studies had design or methodologic flaws that limited their interpretation. More recent, high-quality studies and systematic reviews cast doubt on the effects of these supplements in the general population. Nonetheless, it has been suggested that these supplements may help certain patients, such as those who have a high degree of cartilage turnover and might benefit from supplements that provide substrates needed to sustain this turnover. Given the relative safety of these interventions, these nutritional supplements might therefore be worth a trial in certain people with osteoarthritis.

37. Discuss the primary pharmacologic strategies available for treating rheumatoid arthritis.
   - NSAIDs typically are the first drugs used to control pain and inflammation, and these agents often are the cornerstone of treatment throughout the course of the disease.
   - Glucocorticoids are especially effective in controlling inflammation, but these drugs must be used cautiously because of their catabolic properties and other side effects. Glucocorticoids often are used for short periods to help control flare-ups or acute exacerbations of rheumatoid arthritis.
Disease-modifying antirheumatic drugs (DMARDs) include methotrexate (Mexate, Rheumatrex), etanercept (Enbrel), adalimumab (Humira), anakinra (Kinerei), abatacept (Orencia), azathioprine (Imuran), and several other agents. These drugs are grouped together because they can slow or reverse the joint destruction that typifies rheumatoid arthritis. DMARDs seem to work by suppressing the immune response that causes the degenerative changes associated with rheumatoid arthritis. DMARDs tend to be fairly toxic and their use is limited to patients who are able to tolerate long-term administration.

38. Why are local anesthetics used to treat acute and chronic pain?
Local anesthetics (eg, lidocaine, bupivacaine) block transmission of action potentials in nerve axons. This effect occurs because these drugs inhibit sodium channels from opening in the nerve membrane, rendering the membrane inexcitable for a short period. By blocking transmission in sensory axons, local anesthetics prevent painful sensations from reaching the brain. These drugs can be administered in conditions such as reflex sympathetic dystrophy (also known as complex regional pain syndrome) to try to interrupt painful afferent sensations and to decrease efferent sympathetic discharge to the affected extremity. By using a PCA pump and delivery system, local anesthetics can be administered epidurally to the area surrounding the spinal cord. This type of anesthesia can be especially helpful in decreasing severe pain and improving quality of life in conditions such as cancer.

39. How are local anesthetics used to manage pain during and after surgery?
Local anesthetics can be applied near a peripheral nerve to provide regional anesthesia and allow for surgery at a specific body part supplied by that nerve. For example, injecting a local anesthetic near the median nerve can provide anesthesia to the wrist and hand so that carpal tunnel surgery can be performed. Likewise, a local anesthetic can be injected into the epidural or subarachnoid space near the spinal cord to provide anesthesia below the level of injection, thus allowing more extensive surgeries such as hip or knee arthroplasty. Use of local anesthetics can offer advantages over general anesthesia because the local anesthetic does not suppress activity in the brain and other organs, and recovery from the local anesthetic can be quicker and less problematic in older adults and patients with organ dysfunction.

Local anesthetics can also be applied for the first few days after surgery to maintain the anesthetic effect and provide postoperative pain control. This procedure is known as a peripheral nerve block and is done by inserting a small catheter near the peripheral nerve that supplies the surgical area and then steadily administering a small amount of the local anesthetic onto the nerve. For example, a nerve block can be applied to the femoral nerve in the anterior thigh to provide pain control after knee surgeries. Nerve blocks are often very effective in providing postoperative pain control because the patient cannot feel the body part where the surgery was performed. Clinicians must realize, however, that the loss of sensation is often accompanied by a loss of motor function and joint proprioception, thus making it difficult or impossible for the patient to control and stabilize the joint. Extra care must therefore be taken when exercising the joint, and external support (eg, a knee immobilizer) may be needed if the patient needs to bear weight on the joint.

40. How can medications decrease the risk of thromboembolic disease in patients recovering from hip arthroplasty and other surgeries?
Anticoagulant drugs such as heparin and warfarin (Coumadin) are invaluable in maintaining normal hemostasis after surgery. Heparin is a sugar-like molecule that delays blood clotting by decreasing the activity of thrombin, a key component of the clotting mechanism. Heparin acts rapidly but typically must be administered parenterally by intravenous or subcutaneous routes. Warfarin and similar oral anticoagulants are administered by mouth, and these drugs work by decreasing the production of certain clotting factors in the liver. Oral anticoagulants take several days to affect blood clotting because they have a delayed effect on clotting factor biosynthesis. Heparin and oral anticoagulants often are used sequentially to control excessive clotting; drug therapy begins with parenteral administration of heparin but is switched after a few days or weeks to oral anticoagulants (warfarin), which can be administered for several weeks or months to maintain normal coagulation after surgery.

Other strategies to reduce blood clotting and decrease the risk of thromboembolism include drugs that directly inhibit thrombin (eg, dabigatran [Pradaxa], lepirudin [Refludan]), or inhibit the active form of clotting factor X (eg, fondaparinux [Arixtra], rivaroxaban [Xarelto]). These drugs offer an alternative anticoagulant strategy, especially in patients who may not tolerate more traditional agents such as heparin and warfarin.
41. Is aspirin effective in preventing deep venous thrombosis?
Yes. Aspirin exerts anticoagulant effects by inhibiting the production of prostaglandins that cause platelets to aggregate and participate in clot formation. Aspirin can be administered alone or with other anticoagulants (heparin, warfarin), especially in patients who are at high risk for developing deep venous thrombosis.

42. Is ambulation safe for a patient newly diagnosed with deep vein thrombosis (DVT)?
There is no evidence that ambulation increases the risk of pulmonary embolism after an uncomplicated DVT. That is, immediate ambulation seems to be safe provided that the patient does not have a current or recent pulmonary embolism (symptomatic or asymptomatic) or other risk factors that would increase the likelihood of an embolism (eg, malignant cancer, prolonged immobilization, advanced age). An adequate level of anticoagulant therapy using heparin, warfarin, or alternative agents should also be achieved before starting ambulation. Graduated compression stockings should also be considered because there is evidence that proper use of these garments can prevent complications related to DVT.

43. What drugs are contraindicated for upper cervical manipulation?
Anticoagulant drugs such as heparin, warfarin, and traditional NSAIDs (ie, aspirin and other antiplatelet drugs) can increase the risk of vertebral artery damage and bleeding in patients receiving upper cervical manipulation. In patients taking anticoagulant drugs, therapists should avoid using upper cervical manipulation until laboratory tests indicate that the patient’s clotting time is being maintained within normal limits. If these tests indicate relatively normal hemostasis, upper cervical manipulation still must be used cautiously, and the velocity and force of the manipulation must be reduced to decrease the risk of bleeding caused by vertebral artery damage.

44. Discuss medications that are currently available to treat osteoporosis.
- Calcitonin, a hormone normally produced within the body, can be administered to help increase the storage of calcium and phosphate in the bone.
- Estrogen is likewise important in the hormonal control of bone mineral content in women, and estrogen replacement (using patches or oral supplements) can be especially valuable in certain women after menopause.
- Bisphosphonates, including ibandronate (Boniva) and pamidronate (Aredia), may help stabilize bone mineral content by binding directly to calcium in the bone and preventing excessive calcium turnover.
- Calcium supplements can help provide a dietary source of this essential mineral, and vitamin D supplements can increase absorption of calcium and phosphate from the gastrointestinal tract.

45. What is heterotopic ossification?
Heterotopic ossification is the abnormal formation of bone in muscle and other periarticular tissues. This condition is one of the most common complications that occurs in patients recovering from hip arthroplasty and similar surgical procedures.

46. Discuss drugs that are effective in treating heterotopic ossification.
NSAIDs can reduce significantly the prevalence of heterotopic ossification associated with orthopedic surgeries and other conditions (eg, fracture, rheumatoid arthritis). Treatment with NSAIDs has been successful in reducing the prevalence and severity of heterotopic ossification after total hip arthroplasty. These drugs inhibit prostaglandin biosynthesis, and their ability to limit heterotopic ossification undoubtedly is related to a reduction of proinflammatory prostaglandins in periarticular soft tissues. These drugs seem to work best when used prophylactically, and they often are administered a day or so before surgery and continued for 1 to 6 weeks after surgery.

47. Discuss how cardiovascular medications affect exercise responses.
Certain cardiovascular medications blunt the cardiac response to an exercise bout. β-Blockers typically decrease heart rate and myocardial contractility, resulting in a decrease in blood pressure and heart rate at submaximal and maximal workloads. Digitalis increases myocardial contraction force and can increase left ventricular ejection fraction in patients with heart failure. Other cardiovascular drugs, such as diuretics, vasodilators, antiarrhythmics, angiotensin-converting enzyme inhibitors, and calcium channel blockers can have variable effects on exercise responses, depending on the drug and dosage used, the type of cardiac disease, and the presence of comorbidity.
48. List specific concerns for physical therapists regarding cardiac medications and exercise.
   a. Exercise tolerance may improve when the drug is in effect. This is true even for drugs that blunt cardiac function (eg, β-blockers) because the drug may control symptoms of angina and arrhythmias, allowing the patient to exercise longer and at a relatively higher level.
   b. Exercise prescriptions must take into account the medication effects. The prescription should be based on exercise testing that was performed while the drug was acting on the patient. Formulas that estimate exercise intensity based on age, resting heart rate, and other variables may not be accurate because these formulas fail to account for the effect of each medication on these variables.
   c. Therapists should look carefully for medication-related side effects and adverse effects while the patient is exercising. These effects may be latent when the patient is inactive, but exercise may unmask certain side effects, such as arrhythmias and abnormal blood pressure responses.

49. Can lipid-lowering medications cause skeletal muscle damage?
   Lipid-lowering drugs such as the statins (eg, simvastatin [Zocor], atorvastatin [Lipitor]) are generally well tolerated. In susceptible patients, however, they can cause myopathy that is characterized by skeletal muscle pain, weakness, and inflammation (myositis). In severe cases, myopathy can lead to severe muscle damage (rhabdomyolysis) with disintegration of the muscle membrane and release of myoglobin and other muscle proteins into the bloodstream. This situation can lead to renal damage because the kidneys must try to filter and excrete large quantities of muscle protein. Hence, any patient who is taking lipid-lowering drugs and spontaneously develops muscle pain and weakness should be referred to his or her physician immediately to rule out the possibility of drug-induced myopathy.

50. Can physical agents affect drug absorption, distribution, and metabolism?
   Physical agents (eg, heat, cold, and electricity) can have dramatic effects on drug disposition in the body; this is especially true for drugs that are injected into a specific area. Insulin typically is administered through subcutaneous injection into adipose tissue in the trunk or extremities. Insulin is absorbed into the bloodstream more rapidly if heat and other physical interventions (eg, electric stimulation, massage, exercise) are applied to the injection site. Application of cold agents delays insulin absorption.

   Use of physical agents or manual interventions at the site of the injection should be avoided when the rate of absorption must remain constant or the goal is to keep a drug localized in a specific area. Conversely, heat, massage, and exercise could be applied to a certain area of the body with the idea that a systemically administered drug (ie, a drug that is in the bloodstream) might reach the area more easily because of an increase in local blood flow and tissue metabolism. This idea has not been proved conclusively.

BIBLIOGRAPHY


CHAPTER 16 QUESTIONS

1. Which drugs inhibit heterotopic bone formation?
   a. Muscle relaxers
   b. NSAID’s
   c. Acetominophen
   d. Opioids

2. What class of antibiotics is associated with tendonopathies?
   a. Macrolides
   b. Flouroquinolones
   c. Cephalosporins
   d. Aminoglycosides

3. Which is not a side effect of opioid analgesics?
   a. Sedation
   b. Respiratory depression
   c. Orthostatic hypotension
   d. Diarrhea
1. List various nondisease states that can result in an abnormal laboratory test result.
   - Pregnancy
   - Exercise
   - Posture
   - Food intake and nutritional state
   - Drugs, alcohol, and vitamin and dietary supplements
   - Specimen complications (hemolysis, stasis, sampling error, storage, and exposure)
   - Circadian rhythms, diurnal variation
   - Technician error
   - Reference range variations among different laboratories
   - Normal variations based on patient age, gender, race, and body weight

2. What two characteristics are important for diagnostic laboratory testing?
   Sensitivity and specificity. Sensitivity is the percentage of persons with the disease who are correctly identified by the test. Specificity is the percentage of persons without the disease who are correctly excluded by the test. Clinically, these concepts are important for confirming or excluding disease during screening. Ideally, a test should provide a high sensitivity and specificity. Sensitivity \( = \frac{TP}{TP + FN} \) and Specificity \( = \frac{TN}{TN + FP} \). Abbreviations: TP, true positive; TN, true negative; FP, false positive; FN, false negative.

3. Explain the concepts of positive predictive value (PPV) and negative predictive value (NPV).
   PPV is defined as the percentage of persons with a positive test result who actually have the disease. NPV is the percentage of persons with a negative test result who do not have the disease. Predictive value therefore is the probability that a person’s test result (positive or negative) is correct. PPV \( = \frac{TP}{TP + FP} \) and NPV \( = \frac{TN}{TN + FN} \).

4. Where is albumin produced and what are its functions?
   Albumin is synthesized in the liver. Albumin functions to maintain osmotic pressure in the vasculature and also serves as a transport protein. Hypoalbuminemia leads to abnormal distribution of body water. This occurs because of decreased osmotic pressure within the vasculature and resultant tissue edema. Albumin serves to transport various drugs, ions, pigments, bilirubin, and hormones.

5. What is the normal range for serum albumin levels?
   Normal levels are 3.5 to 5.5 g/dL.

6. What conditions result in decreased albumin levels (hypoalbuminemia)?
   - Poor absorption of albumin (malabsorption, malnutrition)
   - Decreased synthesis of albumin (chronic liver disease)
   - Catabolic states (infection, burns, malignancy, chronic inflammation)
   - Increased losses of albumin (hemorrhage, renal disease, protein-losing enteropathies)
   - Albumin dilution

7. Where does alkaline phosphatase originate?
   Liver (cells of the biliary tract), intestine (mucosal cells of the small intestine), placenta (pregnancy), and bone (osteoblasts) are sources of alkaline phosphatase. Biliary obstruction and Paget’s disease (liver and bone) can result in a marked increase in alkaline phosphatase levels compared with intestinal or placental sources.
8. Explain alkaline phosphatase elevation as it relates to bone.

Any bone lesions (such as sarcoma or metastatic lesions) that produce increased osteoblastic activity will result in elevated alkaline phosphatase levels. Normal bone growth in children and adolescents will also result in alkaline phosphatase elevations.

9. What are the two hepatic conditions that result in elevation of alkaline phosphatase concentration?

- Extrahepatic obstruction—obstruction of the large, extrahepatic bile ducts occurs with bile duct stones, strictures, or tumors. This obstructive process of the biliary system can result in significant enzyme elevation.
- Intrahepatic obstruction—processes within the liver parenchyma can also lead to alkaline phosphatase elevation because of interference with bile flow or transport. Examples include leukemia, sarcoidosis, amyloid, malignancy, primary biliary cirrhosis (PBC), and primary sclerosing cholangitis (PSC).

10. How can liver- versus bone-related elevations in alkaline phosphatase levels be differentiated?

Measure 5′-nucleotidase, gammaglutamyl transpeptidase (GGTP), or fractionation of alkaline phosphatase. If nucleotidase or GGTP is elevated, alkaline phosphatase elevations are caused by a liver, not bone, source. Nucleotidase is present in the bile canaliculi of the liver. GGTP is not present in bone or placental tissue; therefore elevations are because of an underlying liver condition.

11. What is the normal range for alkaline phosphatase?

The normal range is 25 to 100 units/L.

12. What are aminotransferases?

Aminotransferases are enzymes involved in liver synthetic function and/or liver injury. Elevations in both aspartate transaminase (AST) and alanine transaminase (ALT) levels occur with liver inflammation, necrosis, or biliary obstruction. These enzymes are found in many other tissues besides the liver. Together with alkaline phosphatase and bilirubin, aminotransferase evaluation can help the clinician determine the pattern or cause of underlying liver disease.

13. What are nonhepatic sources of AST and causes for its elevation?

In addition to liver, AST is found in the heart, kidney, and skeletal muscle. When AST is elevated without elevation of ALT, a nonhepatic source (ie, muscle, heart) should be considered. Examples include 1) skeletal muscle injury from intramuscular injection, muscle trauma with severe/prolonged exercise, polymyositis, and seizure disorder or 2) myocardial damage as seen in acute myocardial infarction. Both of these nonhepatic conditions can result in isolated AST elevation.

14. List the common pancreatic causes for elevated amylase and lipase levels.

- Acute or chronic pancreatitis
- Pancreatic pseudocyst
- Pancreatic trauma

Amylase is the most sensitive test for pancreatitis; lipase is the most specific indicator of pancreatitis. Often the degree of enzyme elevation does not correlate with the severity of disease. Alcohol and gallstones are the most common causes of acute pancreatitis. Chronic pancreatitis is a result of chronic alcohol abuse, hypercalcemia, hyperlipidemia, trauma, or hereditary causes. Any of these conditions can result in elevated amylase/lipase values.

15. List some of the nonpancreatic causes for elevated amylase and lipase levels.

- Salivary gland disorders (amylase)
- Intestinal perforation or ischemia (amylase and lipase)
- Perforated peptic ulcer (amylase and lipase)

16. What are antinuclear antibodies (ANAs)?

ANAs are used to detect the presence of antinucleoprotein factors associated with certain autoimmune diseases. ANAs are γ-globulins that react with the nuclei of various tissues. The ANA test is reported as a pattern and a titer. The presence of a positive result can 1) occur in normal individuals, 2) may not indicate disease, or 3) may indicate persons destined to develop disease. ANA positivity usually requires confirmatory testing with other disease-specific tests—eg, anti–double-stranded DNA (anti-dsDNA), anti-Smith antibody (anti-Sm antibody), or antiscleroderma antibody, depending on the suspected disease.
17. List diseases associated with a positive ANA (conditions associated with the disease or specific laboratory abnormality in parentheses).

- Systemic lupus erythematosus (SLE) (anti-dsDNA, anti-Sm antibody)
- Rheumatoid arthritis (rheumatoid factor [RF], erythrocyte sedimentation rate [ESR])
- Scleroderma/CREST (calcinosis, Raynaud’s, esophageal, sclerodactyly, telangiectasia) (Scl-70 [antitopoisomerase antibodies]/anticentromere)
- Polymyositis
- Drugs (antideoxyribonucleoprotein [anti-DNP])
- Mixed connective tissue disease (antiribonucleoprotein [anti-RNP])
- Sjögren’s syndrome (anti-SSA, anti-SSB [antinuclear antibodies detected in patients with Sjögren’s syndrome])
- Chronic hepatitis
- Tuberculosis

18. What is bilirubin and what are its two forms?
Bilirubin is a byproduct of hemolysis (red blood cell destruction). It is taken up by the liver, conjugated, and secreted into bile. It is eliminated in the stool and urine. Bilirubin exists in a conjugated and an unconjugated form.

19. Why does jaundice occur with hyperbilirubinemia?
Jaundice is yellow discoloration of the skin because of bile deposition in the skin and sclerae. Jaundice can result from abnormal processing of bilirubin, excess bilirubin production, biliary obstruction, or liver damage. Jaundice is clinically evident when the total bilirubin level is >2.5 mg/dL.

20. What conditions are associated with hyperbilirubinemia?
- Liver disease (hepatitis, cirrhosis, biliary obstruction)
- Hereditary disorders (Gilbert syndrome, Dubin-Johnson syndrome, Crigler-Najjar disease)
- Drugs
- Hemolysis

21. What is blood urea nitrogen (BUN)?
BUN is the end product of protein catabolism. BUN is formed in the liver and excreted by the kidneys. Impairment in kidney function, protein intake, and protein catabolism will affect BUN levels. It is used clinically as an estimate of renal function along with serum creatinine levels.

22. What are the causes of elevated BUN levels?
- Inadequate excretion because of kidney disease/impairment
- Urinary obstruction
- Dehydration
- Drugs (aminoglycosides, diuretics)
- Gastrointestinal bleeding
- Decreased renal blood flow (shock, congestive heart failure [CHF], myocardial infarction [MI])

23. What is the normal range for BUN levels?
Typical BUN levels range from 8 to 18 mg/dl.

24. Where is the majority of calcium stored in the body?
Almost 98% to 99% is found in bone; 1% is found in the intracellular/extracellular space.

25. What factors affect serum calcium levels?
- Parathyroid hormone
- Calcitonin
- Vitamin D
- Estrogens and androgens
- Carbohydrates and lactose
  These factors have a wide range of effects on calcium homeostasis (ie, GI tract absorption, renal excretion and reabsorption, and bone calcium mobilization).

26. What conditions are associated with hypercalcemia?
Hyperparathyroidism, malignancy, sarcoidosis, Paget’s disease, vitamin D intoxication, and thiazide diuretics are all causes of hypercalcemia. The two most common causes are hyperparathyroidism and malignancy.
27. What are the signs and symptoms of hypercalcemia?
The phrase “bones, stones, and psychiatric overtones” is often used to remember signs and symptoms of hypercalcemia. Here, bones refer to bone pain, stones to nephrolithiasis, and psychiatric overtones to confusion and altered concentration. Hypercalcemia is defined as a serum calcium concentration >10.5 mg/dl.

28. What are the signs and symptoms of hypocalcemia?
• Neuromuscular irritability: Chvostek’s sign (facial twitch after tapping facial nerve), Trousseau’s sign (carpopedal spasm after inflation of blood pressure cuff), tetany, and paresthesias
• Psychiatric disturbances
• Cardiovascular abnormalities (arrhythmias, CHF)

29. What causes the neuromuscular irritability (tetany) seen with hypocalcemia?
Neuromuscular irritability occurs as a result of the decrease in the excitation threshold of neural tissue, with a resultant increase in excitability, repetitive response to a stimulus, and continued activity of the affected tissue.

30. What is the prothrombin time (PT), and what does its value signify?
Prothrombin time is a measurement of the clotting ability of five plasma coagulation factors (prothrombin, fibrinogen, factor V, factor VII, and factor X). The PT is commonly used for monitoring warfarin therapy (an anticoagulant) and evaluating liver function (liver normally produces clotting factors).

31. How does warfarin function as an anticoagulant?
It interferes with vitamin K–dependent clotting factors (II, V, VII, X). As a result, the PT will increase (or prolong) and coagulation will be delayed.

32. What conditions can lead to an increased PT?
• Anticoagulant use (warfarin)
• Vitamin K deficiency
• Liver disease (with decreased clotting factor production)
• Factor deficiency (II, V, VII, X)

33. What medical therapy requires monitoring of the PTT (partial thromboplastin time)?
Heparin use requires monitoring of the PTT because heparin is involved in the intrinsic clotting pathway. Heparin acts as a cofactor for antithrombin III, and downregulates coagulation. Heparin is used for treatment of pulmonary embolism, prophylaxis of deep vein thrombosis, and treatment of various coronary conditions such as acute MI.

34. What is the INR (international normalized ratio)?
INR = patient PT divided by the mean PT for the laboratory reference range. INR provides a universal result indicative of what the patient’s PT result would have been if measured using the primary World Health Organization International Reference reagent.

35. What components constitute the CBC (complete blood count)?
• Red blood cell (RBC) count
• White blood cell (WBC) count
• Differential white cell count (Diff)
• Platelet (Plt) count
• Hemoglobin (Hgb) level
• Hematocrit (Hct) level
• Red cell indices (MCV, MCH, MCHC)

36. What are the causes of leukocytosis (elevated WBC count)?
Acute infections, hemorrhage, trauma, malignant disease, toxins, drugs, tissue necrosis/inflammation, and leukemia can all contribute to elevated WBC count.

37. Within the differential white cell count (Diff), name the five white blood cell types, their percentages, and what they protect against.
• Neutrophils (58%)—bacterial infections
• Eosinophils (3%)—allergic disorders and parasitic infections
• Basophils (1%)—parasitic infections
• Monocytes (5%)—severe infections
• Lymphocytes (30%)—viral infections
38. List the causes of neutrophilia.
Acute bacterial infections, acute MI, stress, malignancy, and leukemias can all cause neutrophilia.

39. List the causes of neutropenia.
Neutropenia can be caused by viral infections, aplastic anemias, drugs, radiation, and leukemias.

40. List the causes of eosinophilia.
The acronym NAACP is used to remember the causes of eosinophilia, where N = neoplasms, A = allergies, A = Addison’s disease, C = collagen vascular disorders, and P = parasitic infections.

41. What is the ESR (erythrocyte sedimentation rate), and what does its value signify?
ESR is the rate at which erythrocytes precipitate out of unclotted blood in 1 hour. Inflammation, infections, malignancy, and various collagen vascular diseases increase the ESR because they facilitate erythrocyte aggregation. This affects the rate at which erythrocytes precipitate in a tube (increased aggregation/heaviness = increased rate of descent/sedimentation = increased ESR value).

42. What are some common conditions that lead to an increased ESR?
- Infections
- Inflammatory diseases
- Collagen vascular diseases
- Malignancy
- Anemia

43. What are the clinical applications of the ESR?
ESR is a nonspecific index of inflammation. It should not be used as a screening tool in asymptomatic patients. It is indicated in the diagnosis and monitoring of temporal arteritis and polymyalgia rheumatica. It may also be helpful in monitoring therapy in rheumatoid arthritis, Hodgkin’s disease, and other inflammatory disorders. ESR values can increase with age in the normal population and tend to be slightly higher in females. Normal values are 0 to 15 mm/hr (males) and 0 to 20 mm/hr (females).

44. What are the symptoms of hypoglycemia, and what is the most common cause of this condition?
Adrenergic and neuroglycopenic derangements occur as a result of hypoglycemia. Symptoms and signs include weakness, sweating, tremors, tachycardia, headache, confusion, seizure, and coma. By definition, blood glucose levels <50 mg/dl are considered hypoglycemic. The most common cause of hypoglycemia is excessive insulin dosage/administration.

45. What three criteria must be met to diagnose hypoglycemia?
- Presence of symptoms (adrenergic and/or neuroglycopenic)
- Low plasma glucose level in a symptomatic patient
- Relief of symptoms after ingestion of carbohydrates

46. What are the symptoms of hyperglycemia?
The “3 Ps” are used to remember symptoms of hyperglycemia: polydipsia, polyphagia, and polyuria.

47. What are some of the complications of hyperglycemia and long-standing diabetes?
Retinopathy, neuropathy (peripheral and autonomic), nephropathy, and infections are some of the complications.

48. What is the average life span of platelets, and where are they produced?
The life span of platelets is 7 to 10 days. Platelets are produced in the bone marrow from megakaryocytes. Platelets are necessary for blood clotting and contribute to vascular integrity, adhesion, aggregation, and subsequent platelet plug formation.

49. What are the symptoms of thrombocytopenia?
Symptoms include mild to severe hemorrhage, petechiae, purpura, epistaxis, hematuria, bruising, menorrhagia, and gingival bleeding. Platelet counts >50 x 10^9/L are usually adequate to prevent major bleeding. Spontaneous bleeding is not uncommon with counts <10 x 10^9/L.

50. What are the three major causes of thrombocytopenia?
- Splenic sequestration of platelets
- Increased platelet destruction
- Decreased platelet production
Splenic sequestration, or hypersplenism, can result in the pooling of platelets in the spleen and a subsequent decrease in the number of circulating platelets available for clotting.

51. What specific clinical conditions cause thrombocytopenia?
Thrombocytopenia can be caused by idiopathic thrombocytopenic purpura (ITP), anemias (aplastic and hemolytic), massive blood transfusions, pneumonia, infections, drugs, HIV, splenomegaly, disseminated intravascular coagulation (DIC), and thrombotic thrombocytopenic purpura (TTP). Most of these conditions cause platelet injury, platelet consumption, or platelet loss.

52. What is thrombocytosis?
Thrombocytosis is an increased platelet count, defined as >400 × 10^9/L. This can be a primary (essential thrombocythemia), secondary (eg, leukemia, myeloma, polycythemia, splenectomy, hemorrhage, infections, or drugs), or transient process (after exercise, stress, or epinephrine injection). Clinically, thrombocytosis can cause thrombosis or bleeding or can remain asymptomatic.

53. What are some basic facts about potassium?
Potassium (K+) plays a major role in nerve conduction and muscle function. Total body potassium stores are roughly 3500 mEq. About 90% to 95% of potassium is intracellular and functions as a buffer. K+ is the body’s major cation. Approximately 5% to 10% of K+ is extracellular. Routine blood testing measures only the small extracellular portion and not total body potassium. The majority of K+ (90%) is excreted by the kidneys, with the remainder lost in stool and sweat.

54. What factors influence K+ levels?
K+ levels are influenced by acid-base status, hormone status, renal function, gastrointestinal loss, and nutritional status.

55. What are the common causes of hypokalemia?
- Cellular shift (resulting in extracellular to intracellular movement): alkalosis, insulin administration, and β-agonists
- Gastrointestinal loss: diarrhea, vomiting, nasogastric (NG) suction, laxative use, and fistulas
- Renal loss: diuretic use, magnesium deficiency, renal tubular acidosis, and Bartter syndrome
- Sweating, severe burns
- Poor dietary intake, starvation, and licorice

56. What is a normal K+ level?
Normal K+ levels are 3.5 to 5 mEq/L.

57. What are common causes of hyperkalemia?
- Cellular shift (resulting in intracellular to extracellular movement): cell damage (muscle injury, hemolysis, internal bleeding, burns, surgery, and acidosis) causes hyperkalemia by releasing/shifting intracellular K+ into the extracellular space (blood)
- Decreased urinary excretion: renal excretion is the main elimination pathway for potassium; therefore renal failure or decreased urinary K+ excretion results in hyperkalemia
- Increased potassium intake
- Spurious: spurious causes result from hemolyzed specimen, fist clenching during blood draw, and severe thrombocytosis/leukocytosis

58. What are the symptoms of hypokalemia and hyperkalemia?
- Hypokalemia—muscle weakness, paralysis, cardiac arrhythmias, and electrocardiogram (ECG) changes
- Hyperkalemia—weakness, paresthesias, cardiac arrhythmias, and ECG changes

59. What is rheumatoid factor (RF)?
RF is an anti-γ-globulin antibody thought to be directed against the Fc portion of the IgG molecule. A large portion of patients with rheumatoid arthritis (RA) are RF positive, but the role RF plays in RA is uncertain. About 25% of patients with rheumatoid arthritis are RF negative but may become positive later in their disease course. RF is not a screening test for RA. In addition to rheumatoid arthritis, RF can be seen in systemic lupus erythematosus (SLE), chronic inflammatory processes, old age, infections, liver disease, multiple myeloma, sarcoid, and Sjögren’s syndrome.

60. How is RF reported?
RF is reported as a titer. Values greater than 1:80 are significant; values of 1:640 and higher can be seen in rheumatoid arthritis. Higher titers can correlate with disease severity/activity.
61. What is the human leukocyte antigen (HLA) test?
HLAs are major histocompatibility antigens that are found on all nucleated cells and detected most easily on lymphocytes. The HLA complex is located on chromosome 6 and affects immune system functions.

62. What is the purpose of HLA testing?
HLA testing determines the degree of histocompatibility between a donor and recipient when organ transplantation is contemplated. The degree of HLA “matching” between donor and recipient will impact graft survival and rejection.

63. What are other functions of HLA testing?
HLA testing is also used in various rheumatologic disorders. The presence of a certain HLA antigen may be associated with an increased susceptibility to a specific disease, but it does not mandate the development of that disease in the patient.

64. List the disease and corresponding HLA antigen.

<table>
<thead>
<tr>
<th>Disease</th>
<th>HLA Antigen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankylosing spondylitis</td>
<td>B27</td>
</tr>
<tr>
<td>Reiter syndrome</td>
<td>B27</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>B27, Dw2, A3, B18</td>
</tr>
<tr>
<td>Myasthenia gravis</td>
<td>B8</td>
</tr>
<tr>
<td>Psoriasis</td>
<td>A13, B17</td>
</tr>
<tr>
<td>Graves’ disease</td>
<td>B27</td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>Dw4, DR4</td>
</tr>
</tbody>
</table>

65. What percentage of patients with ankylosing spondylitis are HLA-B27 positive?
About 90% of patients with ankylosing spondylitis are HLA-B27 positive.

66. What is C-reactive protein (CRP)?
CRP is a protein that is present in the blood during periods of inflammation (infection, tissue damage). Besides blood, it can be found in peritoneal, pleural, synovial, and pericardial fluid. Diseases such as rheumatoid arthritis, SLE, inflammatory bowel disease, bacterial infection, and malignancy result in increased CRP levels.

67. What is a normal value for CRP?
A normal value for CRP is < 0.8 mg/dl. The presence of CRP can be detected 16 to 24 hours after the inciting inflammatory event.

68. What is the importance of creatine phosphokinase/creatine kinase (CPK/CK)?
CK is an enzyme found in high levels in skeletal muscle (MM), cardiac muscle (MM and MB), and brain tissue (BB). Tissue injury results in CPK enzyme elevations, and the specific isoenzyme (MM, MB, BB) reflects the affected organ or source.

69. List some of the more common causes of CK-MM (skeletal) elevation.
The MM isoenzyme is found in skeletal muscle. Common causes for elevation include rhabdomyolysis, myositis, crush injury/trauma, polymyositis, dermatomyositis, vigorous exercise, muscular dystrophy, seizures, and IM injection.

70. What are the causes of CK-MB elevation?
Myocardial infarction, muscular dystrophy, myocarditis, and cardiac surgery all contribute to CK-MB elevation.

71. What are the causes of CK-BB elevation?
CK-BB elevation can be caused by severe brain injury, hyperthermia, Reye’s syndrome, and uremia.

72. What are the general functions of sodium?
In general, sodium affects acid-base balance, osmotic pressure balance, and nerve transmission. Sodium concentrations are regulated by the renal system, CNS, and endocrine systems acting in concert. Despite wide variations in sodium intake, serum levels are maintained within a narrow therapeutic range. The normal serum sodium level is 135 to 148 mEq/L. Changes in body water and salt balance are determined/monitored by serum sodium levels.
73. What factors play a role in sodium homeostasis?
Renal blood flow, carbonic anhydrase activity, aldosterone, pituitary hormones, renin, and antidiuretic hormone are important in sodium homeostasis.

74. What are the symptoms of hyponatremia?
Manifestations vary with the degree of hyponatremia and the rapidity of onset. Confusion, muscle cramps, lethargy, anorexia, and nausea are seen with moderate hyponatremia or gradual onset of hyponatremia. Severe hyponatremia or rapid onset can lead to seizures or coma.

75. What are the causes of hyponatremia?
There are many causes: 1) hypotonic (isovolemic, hypovolemic, or hypervolemic); 2) isotonic; or 3) hypertonic. Within these categories are renal losses (diuretic use, urinary obstruction), extrarenal losses (vomiting, diarrhea, burns, third spacing), adrenal insufficiency, syndrome of inappropriate antidiuretic hormone (SIADH), water intoxication, renal failure, NSAID use, ACE inhibitor use, CHF, nephrosis, cirrhosis, pseudohyponatremia, and hyperglycemia.

76. How are hyponatremia and hypernatremia similar?
The clinical manifestations (confusion, lethargy, seizures, and coma) relate to the degree of hyponatremia or hypernatremia and the rapidity of onset of the electrolyte disturbance.

77. A patient with low serum sodium levels, tachycardia, hypotension, vomiting, diarrhea, and diuretic use has what form of hyponatremia?
This patient is volume depleted and is suffering from hypovolemic hyponatremia. Treatment is isotonic fluid replacement.

78. A patient with low serum sodium levels, edema, CHF, cirrhosis, and renal failure has what form of hyponatremia?
This patient is volume overloaded and is suffering from hypervolemic hyponatremia. Both sodium and water are increased, but water is increased proportionally more than sodium. Treatment is sodium and water restriction and diuretic therapy.

79. What are the causes of hypernatremia?
- Isovolemic (decreased total body water + normal total body sodium levels): diabetes insipidus, skin loss
- Hypervolemic (increased total body water + marked increase in body sodium levels): iatrogenic administration of high sodium solutions, salt intake
- Hypovolemic (loss of body water > loss of body sodium): renal losses, GI losses, respiratory losses, and skin losses

80. List some normal laboratory values.

<table>
<thead>
<tr>
<th>Test</th>
<th>Low Value</th>
<th>High Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC count</td>
<td>&lt;5000/mm³</td>
<td>&gt;10,000/mm³</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>&lt;55%</td>
<td>&gt;70%</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>&gt;20%</td>
<td>&gt;40%</td>
</tr>
<tr>
<td>Monocytes</td>
<td>&lt;2%</td>
<td>&gt;8%</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>&lt;1%</td>
<td>&gt;4%</td>
</tr>
<tr>
<td>Basophils</td>
<td>&lt;0.5%</td>
<td>&gt;1%</td>
</tr>
<tr>
<td>RBC (male)</td>
<td>&lt;4.7 million/mm³</td>
<td>&gt;6.1 million/mm³</td>
</tr>
<tr>
<td>RBC (female)</td>
<td>&lt;4.2 million/mm³</td>
<td>&gt;5.4 million/mm³</td>
</tr>
<tr>
<td>MCV</td>
<td>&lt;80 mm³</td>
<td>&gt;95 mm³</td>
</tr>
<tr>
<td>MCH</td>
<td>&lt;27 pg</td>
<td>&gt;31 pg</td>
</tr>
<tr>
<td>MCHC</td>
<td>&lt;32 g/dl</td>
<td>&gt;36 g/dl</td>
</tr>
<tr>
<td>Hemoglobin (male)</td>
<td>&lt;14 g/dl</td>
<td>&lt;16 g/dl</td>
</tr>
<tr>
<td>Hemoglobin (female)</td>
<td>&lt;12 g/dl</td>
<td>&lt;14 g/dl</td>
</tr>
<tr>
<td>Hematocrit (male)</td>
<td>&lt;45%</td>
<td>&gt;52%</td>
</tr>
<tr>
<td>Hematocrit (female)</td>
<td>&lt;37%</td>
<td>&gt;47%</td>
</tr>
<tr>
<td>Platelets</td>
<td>&lt;150,000 mm³</td>
<td>&gt;400,000 mm³</td>
</tr>
<tr>
<td>ESR (male)</td>
<td>Up to 15 mm/hr is normal</td>
<td></td>
</tr>
<tr>
<td>ESR (female)</td>
<td>Up to 20 mm/hr is normal</td>
<td></td>
</tr>
</tbody>
</table>
CPK (male) <12 units/ml >70 units/ml
CPK (female) <10 units/ml >55 units/ml
ANA Normal findings are no ANA detected in a titer with a dilution of >1:32
CRP Rheumatoid factor Abnormal if present

81. What do these figures represent?

Laboratory values are usually recorded as follows:
- WBC, white blood cell count
- Hb, hemoglobin level
- Hct, hematocrit level
- Plt, platelet count
- Na, sodium concentration
- K, potassium concentration
- Cl, chloride concentration
- CO₂, bicarbonate concentration
- BUN, blood urea nitrogen level
- Cr, creatinine level
- Gluc, glucose level

BIBLIOGRAPHY

CHAPTER 17 QUESTIONS
1. A 70-year-old male taking medication for hypertension and diabetes develops profound muscle weakness after 4 days of vomiting and diarrhea. The patient has a history of chronic, stable constipation. Serum potassium level is 2.3 meq/L (range 3.5–5.0 meq/L). Which of the following is not associated with his symptoms of severe hypokalemia?
   a. Vomiting
   b. Diuretic use
c. Acidosis
d. Diarrhea
e. Alkalosis

2. Which of the following are not associated with hypercalcemia?
   a. Malignancy
   b. Sarcoidosis
c. Hyperparathyroidism
d. Vitamin D deficiency
e. Thiazide diuretics
3. Severe hemorrhage, petechiae, purpura, epistaxis, hematuria, and bruising are signs of which condition?
   a. Leukocytosis
   b. Thrombocytosis
   c. Thrombocytopenia
   d. Hypoalbuminemia
   e. Hypocalcemia
1. What are nerve conduction studies (NCS) and needle electromyography (EMG)?

NCS is the electrical stimulation and recording of a peripheral nerve’s action potential. Its purpose is to test the integrity of the myelin sheath of motor (efferent fibers) and sensory (afferent fibers) portions of the peripheral nervous system. EMG is the analysis of bioelectric potentials generated by the motor unit (anterior horn cell, its axon and complement of neuromuscular junctions, and muscle fibers). It is a measure of the motor portion of the peripheral nervous system only. Both tests are performed during the patient encounter. Needle EMG must be performed and interpreted in real time. NCS/EMG studies are also known as clinical electrophysiology, electroneuromyography, electrophysiologic evaluation, or electrodiagnostic testing.

2. What is the usefulness of NCS and needle EMG?

As a part of physical therapy tests and measurements, an NCS/EMG study is an extension of the neuromuscular examination of the peripheral nervous system. It identifies normal or abnormal physiologic findings by location and by characteristics and offers information regarding prognosis. NCS/EMG studies are used to identify compression neuropathies and diseases of peripheral nerve and muscle tissue. Needle EMG facilitates the identification of botulinum toxin injection sites for movement disorder management in spasticity, dystonia, and dysphonia. NCS/EMG is also used during surgery to help identify and protect viable nervous tissue during tumor resection and to aid in monitoring spinal cord motor and sensory function during stabilization procedures. Because it is not pathognomonic for any disease, NCS/EMG by itself is not diagnostic. The definitive diagnosis is determined by the evaluation gleaned from information that includes the patient’s history, review of systems, laboratory and imaging tests, and neuromusculoskeletal examination, which may include an NCS/EMG study.

3. When is it most appropriate to refer for an NCS/EMG test?

NCS/EMG studies are helpful in answering the clinical question: is there nerve involvement? The most common referral seen from the primary care setting is an evaluation for an entrapment neuropathy (eg, carpal tunnel syndrome or ulnar neuropathy at the elbow). The next most common referral is to differentiate between bilateral lumbosacral radiculopathies caused by spinal stenosis and peripheral polyneuropathy. They are also invaluable for diagnosing primary muscle disease, including muscle weakness that may occur as a major side effect from the pharmacologic management of hyperlipidemia (use of statin drugs).

NCS/EMG tests are not as helpful when pain is the only symptom. They can be extremely helpful when pain is associated with numbness, tingling, and/or weakness. True motor weakness is a red flag that obligates a thorough evaluation.

4. What are examples of compression neuropathies and diseases of nerve and muscle that NCS/EMG studies can help identify?

Compression neuropathies are localized injuries caused by a mechanical fault:

- Carpal tunnel syndrome (median neuropathy at the wrist)
- Ulnar neuropathy at the elbow
- Handlebar palsy (ulnar neuropathy at the wrist)
- Radiculopathy — cervical or lumbosacral
- Thoracic outlet syndrome
- Backpacker’s palsy (long thoracic nerve palsy)
- Suprascapular nerve palsy
- Saturday night palsy (radial nerve palsy)
- Radial tunnel syndrome
- Meralgia paresthetica (lateral cutaneous nerve of the thigh)
- Fibular nerve palsy (peroneal nerve palsy)
- Tarsal tunnel syndrome

Diseases of nerve and muscle:
- Neuropathy (diabetic, uremic, alcoholic, hereditary, toxic, Guillain-Barré syndrome, and motor neuron disease)
- Plexopathy (idiopathic brachial neuritis, Parsonage-Turner syndrome, neuralgic amyotrophy, diabetic amyotrophy, and neoplasms)
- Myopathy (myositis, muscular dystrophy, and myotonia)
- Neuromuscular junction (myasthenia gravis, myasthenic syndrome, and botulism)

5. Define NCS terminology.
- Latency—the time interval in milliseconds (ms) between the electric stimulus that depolarizes the nerve and the recording of a motor or sensory action potential. An abnormal latency is reported as prolonged.
- Amplitude—the size of the motor or sensory action potential. It is measured in millivolts (mV) or microvolts (μV). It reflects the number of axons available for stimulation. An abnormal amplitude is reported as low.
- Nerve conduction velocity—the calculated conduction speed along the myelin is reported in meters per second (m/sec). It is calculated by dividing the distance between two points of stimulation by the time it took for the nerve impulse to travel between these two points. An abnormal conduction velocity is reported as slow.
- Duration—the width of the recorded action potential measured in milliseconds (ms). It reflects the summation and timing of all fibers available for stimulation. An abnormal duration is reported as temporal dispersion.

6. Define needle EMG terminology.
- Insertional activity—the recording period measuring the timing of inserting a sterile flexible wire electrode into muscle tissue. It reflects the integrity of the muscle cell membrane. The normal range is less than 200 ms. Abnormal insertional activity may be reported as increased (>200 ms reflecting acute muscle cell membrane instability) or decreased (<50 ms reflecting a loss of electrical viability of the muscle cell membrane).
- Rest activity—the normal resting state of voluntary muscle is silent (no electrical activity/flat baseline). The needle EMG recording of abnormal spontaneous potentials indicates muscle cell membrane instability. The spontaneous appearance of positive sharp waves and fibrillation potentials indicates an acute process of muscle cell membrane instability and may be present in neuropathic or myopathic conditions. Other abnormal spontaneous discharges include waxing and waning discharges, complex repetitive discharges and fasciculations that indicate chronicity.
- Voluntary activity—the evaluation of a motor unit’s configuration and rate. A normal motor unit has four phases or less (times crossing the baseline), is less than 5 mV in amplitude, is more than 5 ms and less than 16 ms in duration, and fires at a frequency of less than 10 Hz. A neuropathic motor unit may have more than four phases (polyphasic), greater than 5 mV in amplitude (larger than normal amplitude), greater than 16 ms in duration (wide duration), and/or fires faster than 10 Hz before recruitment of the next motor unit. A myopathic motor unit may be less than 300 μV in amplitude (low amplitude), less than 5 ms in duration (short duration), and fire many motor units with minimal effort. This distinguishes a neuropathic process from a myopathic process.
- Maximum contraction—the normal pattern for a maximal voluntary contraction should fill the oscilloscope screen with firing motor units. A neuropathic condition would exhibit reduced motor unit recruitment along with a loss of firing voluntary motor units. A myopathic condition would exhibit early motor unit recruitment (unable to isolate individual voluntary motor units well) along with a loss of medium (2–3 mV) to large (4–5 mV) amplitude motor units.

7. Define the NCS/EMG pathologic process terminology.
- Demyelinating process—describes the pathologic state of a nerve when its impulses travel at a significantly slower conduction velocity. When the amplitude is within normal limits, this suggests the existence of a disease process or injury of the myelin (see focal demyelinating process below).
Typically reported for Guillain-Barré syndrome (acute inflammatory demyelinating polyneuropathy), and Charcot-Marie-Tooth disease (hereditary motor sensory neuropathy type I).

- Axonal process—describes the pathologic state when the nerve conduction evoked potentials have low amplitudes and/or acute denervation changes are present on needle EMG. Typically reported for alcoholic, uremic, and drug-induced/toxic neuropathies and hereditary motor sensory neuropathy type II.
- Mixed-type polyneuropathic process—describes a pattern including both demyelination and axonal involvement. Typically reported for diabetic neuropathy.
- Focal demyelinating process—when the nerve conduction is normal distally (below) and proximally (above) to a nerve injury but slow over (across) the segment, the location of the nerve injury is identified. This usually has a good prognosis of recovery.
- Conduction block—when stimulation proximal (above the lesion) records a nerve conduction evoked potential low in amplitude compared with the distal (below the lesion) recording and their durations are similar, there may be a conduction block of the myelin. This usually has a good prognosis for recovery.
- Reinnervation—when a larger than normal amplitude (>5 mV) motor unit is present on needle EMG, it indicates an unaffected adjacent motor unit has sprouted terminal axons to reinnervate nearby denervated muscle cells. The motor unit’s anterior horn cell determines the muscle cell type (type I or type II). This is a sign of recovery.

8. List the normal values for nerve conduction studies.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Distal Latency</th>
<th>Amplitude</th>
<th>Conduction Velocity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper Limb</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Motor nerve (8-cm distance)</td>
<td>&lt;4.2 ms</td>
<td>&gt;4 mV</td>
<td>50–60 m/sec</td>
</tr>
<tr>
<td>Sensory nerve (14-cm)</td>
<td>&lt;3.5 ms</td>
<td>&gt;10 μV</td>
<td>40 m/sec</td>
</tr>
<tr>
<td>Lower Limb</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Motor nerve (8-cm distance)</td>
<td>&lt;5.5 ms</td>
<td>&gt;2 mV</td>
<td>40–50 m/sec</td>
</tr>
<tr>
<td>Sensory nerve (14-cm)</td>
<td>&lt;4.0 ms</td>
<td>&gt;10 μV</td>
<td>35 m/sec</td>
</tr>
</tbody>
</table>

9. What are the limitations of NCS/EMG studies?
NCS/EMG studies may be normal when a patient has a clinical presentation of a radiculopathy. Radiculopathies are nerve injuries that are preganglionic (proximal to the dorsal root ganglion). When sensory nerve root fibers are primarily involved, the needle EMG test will be normal. It takes 14 to 21 days for acute muscle cell membrane instability changes, such as fibrillation potentials and positive sharp waves, to develop and be detected by needle EMG examination. In a completely severed nerve or when severe conduction block is present, voluntary motor units are absent in muscles innervated by the nerve distal to the lesion site. Also, NCS records only large-diameter motor and sensory fiber function. Small-diameter nerve fiber injury or disease is not detectable by NCS/EMG studies.

10. What are the sensitivity and specificity values for NCS/EMG studies?
In general NCS/EMG studies are moderately sensitive (rule out/disease is not present) and highly specific (rule in/disease is present).

<table>
<thead>
<tr>
<th>Condition</th>
<th>Determines</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carpal tunnel syndrome</td>
<td>Conduction delay, conduction block +/- or axonal loss</td>
<td>&gt;85%</td>
<td>95%</td>
</tr>
<tr>
<td>Ulnar neuropathy at the elbow</td>
<td>Conduction delay, conduction block +/- or axonal loss</td>
<td>37%–86%</td>
<td>95%</td>
</tr>
<tr>
<td>Cervical radiculopathy</td>
<td>Presence and location of axonal injury of the motor nerve root</td>
<td>50%–71%</td>
<td>65%–85%</td>
</tr>
<tr>
<td>Lumbosacral radiculopathy</td>
<td>Presence and location of axonal injury of the motor nerve root</td>
<td>29%–92%</td>
<td>37%–100%</td>
</tr>
<tr>
<td>Distal symmetric polyneuropathy</td>
<td>Type and severity of neuropathy Conduction delay, conduction block +/- or axonal loss</td>
<td>80%–83%</td>
<td>72%–89%</td>
</tr>
<tr>
<td>Tarsal tunnel syndrome</td>
<td>Conduction delay, conduction block +/- or axonal loss</td>
<td>Unknown</td>
<td>Unknown</td>
</tr>
<tr>
<td>Fibular neuropathy syndrome</td>
<td>Conduction delay, conduction block +/- or axonal loss</td>
<td>Unknown</td>
<td>Unknown</td>
</tr>
</tbody>
</table>
11. Can myopathies as well as neuropathies be determined by an NCS/EMG study?
Yes. In many myopathic processes, the NCS is usually normal. On the needle EMG examination, positive sharp waves and fibrillation potentials are present at rest (acute muscle cell membrane instability) accompanied by early motor unit recruitment during minimal voluntary contractions. Most often these motor units have lower than normal amplitudes (<300 μV) and short durations. Although the anterior horn cell along with its axon and neuromuscular junctions remains intact, the loss of muscle fibers results in short-duration low-amplitude motor units. The pattern of myopathic abnormalities is typically confined to the most proximal muscles of the limbs and trunk and is symmetric. In contrast, a neuropathic process can demonstrate low motor evoked amplitudes on NCS as a result of motor fibers available for stimulation. On needle EMG examination, positive sharp waves and fibrillation potentials are present at rest (acute muscle cell membrane instability) along with fewer motor units firing during voluntary muscle contractions (reduced motor unit recruitment). The surviving motor units have larger than normal amplitudes (>5 mV) and wide durations because they sprout to capture denervated muscle fibers within their territory (reinnervation). The pattern of abnormalities is typically confined to a specific peripheral nerve distribution (root, plexus, nerve) or may be distal and symmetric when disease is present.

12. Can neuromuscular junction disorders be determined by an NCS/EMG study?
Yes. The neuromuscular junction disease, myasthenia gravis, is shown on needle EMG when the motor units drop out quickly and/or reduce in amplitude (unstable). In addition, during a repetitive stimulation NCS test with stimulation rates of three per second, a train of evoked motor potentials shows a drop in amplitude of more than 10% between the first and fifth evoked potential recording. This is caused by pathologic changes in the acetylcholine receptors on the postsynaptic side of the neuromuscular junction that block the transmission process and cause motor weakness. Normal conditions show no change in amplitude.

13. What is a somatosensory evoked potential (SSEP) study? When is SSEP appropriate?
SSEP is the study of sensory nerve pathways between the extremity and the dorsal column in the spinal cord to the somatosensory cortex. It is performed by stimulating the peripheral nerve and recording through multiple channels at more proximal segments along the nerve, including one or more sites along the spine and at the somatosensory cortex. When a weakness or loss of sensation is significant and there are signs that the location of the lesion could be of central nervous system (spinal cord or brain) origin, the use of SSEP is appropriate. SSEP would be more likely to show a nerve root (preganglionic) sensory nerve lesion than an NCS/EMG study.

14. Is there any value in requesting an NCS/EMG study for a patient with a suspected nerve injury during the first 3 weeks (21 days) of injury?
Yes, to determine whether there is total loss of nerve continuity. It requires 5 to 10 days for injured peripheral nerves to deteriorate completely because of wallerian degeneration distal to the suspected lesion. Nerve conduction distal to the lesion may look normal immediately after nerve severance and continue to look relatively normal for up to 3 days. Early serial NCS testing can differentiate the presence of a complete peripheral nerve lesion versus a potential for good recovery. A preserved evoked amplitude present 4 days after the onset of the lesion has a good prognosis.

15. What is wallerian degeneration, and how long does it take a peripheral nerve lesion of this type to recover?
Wallerian degeneration is disruption of the myelin and axons along the entire length of the nerve below the site of the lesion. The symptoms take effect immediately, but it takes 21 days for acute denervation changes to develop on needle EMG. Because the epineurium remains intact, nerve regeneration can readily take place in a health body. However, it is often slowed when metabolic comorbidities are present. The approximate rate of nerve regeneration is 6 mm per day for a root level lesion, 2 mm per day for a forearm level lesion, and 1 mm per day for a hand or lower leg level lesion.

CASE STUDY
A 21-year-old male long jumper had a 4-day history of right foot weakness along with numbness and tingling in the lateral leg and dorsal foot, which reportedly followed a training room treatment of ice application secured with an elastic wrap for a lateral distal hamstring strain. Manual muscle test demonstrated 2/5 strength of dorsiflexion, eversion, toe extension, and great toe extension. Sensation was decreased to pinprick along the dorsum of the foot. Patellar and Achilles monosynaptic reflexes were a 2+ bilaterally. The deep fibular
nerve conduction velocity recording from the extensor digitorum brevis was 30 m/sec across the fibular head segment and 42 m/sec through the lower leg. The deep fibular nerve had an amplitude of 800 μV proximal to the fibular head and 3 mV with stimulation below the fibular head. The NCS of the superficial fibular sensory nerve had a distal latency of 3.9 ms and an amplitude of 13 μV. The needle EMG examination demonstrated reduced motor unit firing in the tibialis anterior, fibularis longus, and extensor hallucis longus. The remainder of the muscles selected for study including the short head of the biceps femoris, tibialis posterior, and lumbosacral paraspinal muscles were normal. There were no positive sharp waves or fibrillation potentials seen at rest in all muscles tested.

A repeat test 17 days later recorded a conduction velocity of 28 m/sec from the deep fibular nerve across the fibular head segment and 40 m/sec distal lower leg segment. The motor evoked potential was 900 μV with stimulation above the fibular head and 3 mV with stimulation below the fibular head. The superficial fibular sensory nerve distal latency was 3.9 ms, and its evoked amplitude was 6 μV. The needle EMG demonstrated positive sharp waves and fibrillation potentials in the right anterior tibialis, fibularis longus, and extensor hallucis longus, along with reduced motor unit recruitment. The remainder of the needle EMG examination, including the short head of the biceps femoris, tibialis posterior, and lumbosacral paraspinalis was unremarkable. No change was noted in the muscle grades from the initial examination.

16. From the information given previously, can you locate and describe this nerve injury and give a prognosis?

The NCS/EMG studies are consistent with a severe focal demyelinating injury to the common fibular nerve below the branch to the short head biceps femoris at the fibular head with evidence of conduction block and acute denervation changes. Focal demyelination is confirmed by the isolated slowing of the deep fibular nerve across the fibular head segment. The drop in amplitude with proximal stimulation confirms conduction block. Acute denervation is confirmed by the presence of positive sharp waves and fibrillation potentials along with reduced motor unit recruitment in the right common fibular nerve distribution below the branch to the short head biceps femoris. Prognosis is good and it is estimated that recovery will take around 2 to 3 months.

BIBLIOGRAPHY


CHAPTER 18 QUESTIONS

1. What reflects the measure of time on an NCS?
   a. Amplitude
   b. Conduction velocity
   c. Latency
   d. Waveform
   NOTE: This is a recall item.

2. Which part of the needle EMG procedure determines whether the lesion is neuropathic or myopathic?
   a. Insertional activity
   b. Maximum contraction
   c. Resting activity
   d. Voluntary activity
   NOTE: This is a recall item.

3. A 37-year-old male complains of severe neck pain and left arm pain and numbness in the little finger for the past few months. NCS is normal. Needle EMG shows the presence of positive sharp waves and fibrillation potentials in the lower cervical paraspinal muscles. Based on these findings, which of the following is the most likely involved?
   a. C8 spinal nerve
   b. Lower trunk
   c. Medial cord
   d. Ulnar nerve
   NOTE: This is a synthesis item.
1. What are the common myotomes tested in an upper and lower quarter screening examination?

<table>
<thead>
<tr>
<th>Spinal Segment Level</th>
<th>Myotome (a group of muscles supplied by one ventral nerve root)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C3–4</td>
<td>Shoulder elevation and cervical rotation</td>
</tr>
<tr>
<td>C5</td>
<td>Shoulder abductors and external rotators</td>
</tr>
<tr>
<td>C6</td>
<td>Elbow flexors and wrist extensors</td>
</tr>
<tr>
<td>C7</td>
<td>Elbow extensors and wrist flexors</td>
</tr>
<tr>
<td>C8</td>
<td>Thumb and finger extensors</td>
</tr>
<tr>
<td>T1</td>
<td>Hand intrinsic muscles</td>
</tr>
<tr>
<td>T3–12</td>
<td>Segmental innervation of muscles in thoracic and abdominal walls</td>
</tr>
<tr>
<td>L2–3</td>
<td>Hip flexors</td>
</tr>
<tr>
<td>L3–4</td>
<td>Knee extensors</td>
</tr>
<tr>
<td>L4–5</td>
<td>Ankle dorsiflexors</td>
</tr>
<tr>
<td>L5</td>
<td>Great toe extensors, hip abductors</td>
</tr>
<tr>
<td>S1</td>
<td>Plantar flexors</td>
</tr>
<tr>
<td>S2–3</td>
<td>Foot intrinsic muscles</td>
</tr>
</tbody>
</table>

2. What are the common dermatomes tested in an upper and lower quarter screening examination?

<table>
<thead>
<tr>
<th>Spinal Root</th>
<th>Dermatome (area of skin supplied by sensory neurons that arises from a dorsal root ganglion)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1</td>
<td>Top of head</td>
</tr>
<tr>
<td>C2</td>
<td>Side of head</td>
</tr>
<tr>
<td>C3–4</td>
<td>Lateral neck and top of shoulder</td>
</tr>
<tr>
<td>C5</td>
<td>Lateral shoulder and arm</td>
</tr>
<tr>
<td>C6</td>
<td>Lateral forearm, thumb, and index finger</td>
</tr>
<tr>
<td>C7</td>
<td>Middle and ring fingers</td>
</tr>
<tr>
<td>C8</td>
<td>Ring and little fingers</td>
</tr>
<tr>
<td>T1–2</td>
<td>Medial forearm and arm</td>
</tr>
<tr>
<td>L1–2</td>
<td>Groin</td>
</tr>
<tr>
<td>L2–3</td>
<td>Anterior and medial thigh</td>
</tr>
<tr>
<td>L4</td>
<td>Medial lower leg</td>
</tr>
<tr>
<td>L5</td>
<td>Lateral lower leg and dorsum of foot</td>
</tr>
<tr>
<td>S1</td>
<td>Posterior lateral thigh and lower leg and lateral foot</td>
</tr>
<tr>
<td>S2</td>
<td>Plantar surface of foot</td>
</tr>
<tr>
<td>S3</td>
<td>Groin</td>
</tr>
<tr>
<td>S4</td>
<td>Perineum region, genitals</td>
</tr>
</tbody>
</table>

3. What are the commonly tested deep tendon reflexes?

<table>
<thead>
<tr>
<th>Stretch Reflex</th>
<th>Spinal Root Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jaw jerk</td>
<td>Trigeminal nerve (cranial nerve V)</td>
</tr>
<tr>
<td>Biceps</td>
<td>C5 (C6)</td>
</tr>
<tr>
<td>Brachioradialis</td>
<td>(C5) C6</td>
</tr>
</tbody>
</table>
### Classify the cranial nerves, their functions, and how they are tested.

<table>
<thead>
<tr>
<th>Cranial Nerve (CN)</th>
<th>Function</th>
<th>Test</th>
<th>Clinical Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>I—Olfactory</td>
<td>Olfaction (smell)</td>
<td>Place common strong smells (eg, coffee, lemon juice, cloves, peppermint) under each naris (closing untested side); eyes closed</td>
<td>Smell may be lost post trauma because of tearing of olfactory striae from cribriform plate of ethmoid bone; anosmia or hyposmia post traumatic brain injury or skull fracture may be indicative of cerebrospinal fluid (CSF) rhinorrhea in which CSF leaks into the nasal cavity (seen in whiplash, closed head injury)</td>
</tr>
<tr>
<td>II—Optic</td>
<td>Vision</td>
<td>Test visual fields; test visual acuity using a Snellen chart</td>
<td>Accurate assessment of visual fields greatly aids in localization of neurologic dysfunction; for example, bitemporal hemianopia is a common clinical presentation of tumors within the pituitary gland</td>
</tr>
<tr>
<td>III—Oculomotor</td>
<td>Most extraocular muscles (elevates eyelid, turns eye up, down, in), pupil constriction, and lens accommodation</td>
<td>Check pupillary size for symmetry and pupillary light response; both eyes should look forward and move smoothly and symmetrically (no nystagmus); check ability of subject to track examiner’s finger, moving it up, down, and toward midline</td>
<td>Dysfunction of CN III, IV, or VI produces diplopia with head held in neutral position; patients often present with cervical deviation to correct diplopia; cervical deviation may be mistaken for a torticollis deformity</td>
</tr>
<tr>
<td>IV—Trochlear</td>
<td>Superior oblique extraocular muscle (moves eye down and in)</td>
<td>Test subject’s ability to move eyes diagonally downward and toward midline</td>
<td>See CN III</td>
</tr>
<tr>
<td>V—Trigeminal</td>
<td>Sensation from face (including cornea and anterior tongue) and motor innervation of muscles of mastication</td>
<td>Perform sensory testing of face (sharp/dull discrimination, light touch); check ability to clenches teeth and open mouth; subject should blink eye with gentle brushing of cornea (tests afferent limb of corneal blink reflex); jaw jerk reflex</td>
<td>An upper motor lesion produces little dysfunction because of bilateral cortical input to the lower motor neuron innervating the muscles of mastication; lower motor neuron lesion results in unilateral paralysis and atrophy of muscles of mastication</td>
</tr>
<tr>
<td>VI—Abducens</td>
<td>Lateral rectus extraocular muscle (moves eye laterally)</td>
<td>Test subject’s ability to move eyes away from midline</td>
<td>See CN III</td>
</tr>
<tr>
<td>Section</td>
<td>Test</td>
<td>Description</td>
<td></td>
</tr>
<tr>
<td>---------</td>
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</tr>
<tr>
<td>VII—Facial</td>
<td>Muscles of facial expression, taste anterior tongue, tearing, and salivation</td>
<td>Check symmetry and smoothness of facial expressions at rest and during voluntary movement; both eyes should blink with corneal brushing (efferent limb of corneal blink reflex); test taste sensation in anterior two thirds of tongue. Swelling within facial canal results in weakness in ipsilateral facial muscles and loss of taste from ipsilateral anterior two thirds of tongue (Bell’s palsy); there will be drooping of the cheek and eye (ptosis) on the involved side.</td>
<td></td>
</tr>
<tr>
<td>VIII—Vestibulocochlear</td>
<td>Hearing and vestibular function (balance)</td>
<td>Rub fingers by each ear (subject should hear equally from both ears); Rinne test (vibrating tuning fork on mastoid process, then near external ear canal; assess hearing acuity); Weber’s test (vibrating tuning fork on vertex of skull or forehead; subject notes which side is louder); move head slowly side to side to check vestibuloocular reflex; eyes should move in opposite direction of head movement. Common cause of cochlear damage to this nerve is an acoustic neuroma — a tumor of the Schwann cells that myelinate this nerve.</td>
<td></td>
</tr>
<tr>
<td>IX—Glosso-pharyngeal</td>
<td>Gag reflex, swallowing, taste posterior tongue, and salivation</td>
<td>Check gag reflex; check taste sensation in posterior tongue. Lesions of this nerve seldom occur alone; sudden pain of unknown cause that begins in throat and radiates down side of neck in front of ear posterior mandible usually precipitated by swallowing or protrusion of jaw is known as glossopharyngeal neuralgia.</td>
<td></td>
</tr>
<tr>
<td>X—Vagus</td>
<td>Phonation, swallowing, and thoracic and abdominal viscera regulation</td>
<td>Have subject say “ah”; observe elevation of soft palate. Lesions result in hoarse voice and difficulty swallowing; patient often complains of food and fluid regurgitation into nasal cavity.</td>
<td></td>
</tr>
<tr>
<td>XI—Accessory</td>
<td>Trapezius and sternocleido-mastoid</td>
<td>Test trapezius and sternocleido-mastoid muscle strength. Usually related to radical neck surgery (as in resection of laryngeal carcinomas) that involves dissection of lymph nodes.</td>
<td></td>
</tr>
<tr>
<td>XII—Hypoglossal</td>
<td>Tongue movements</td>
<td>Stick tongue out and observe for symmetric movement of tongue. Upper motor neuron lesion results in weakness without atrophy and deviation to side opposite lesion; lower motor neuron lesion results in paralysis and atrophy of tongue muscles on affected side, and tongue deviates to same side as lesion; common causes are metastatic tumors or cerebral infarction.</td>
<td></td>
</tr>
</tbody>
</table>
5. Define terminology describing common sensory impairments.

The root word *esthesia* means “feeling” or “sensation.”

- **Hypoesthesia**—diminished sensation
- **Hyperesthesia**—heightened sensation
- **Anesthesia**—the complete lack of sensation in a particular dermatome, peripheral nerve distribution, or region. The prefix *an* means “none.”
- **Paresthesia**—abnormal sensation, often described as pins and needles. The prefix *para* means “aside” or “beyond.”
- **Dysesthesia**—unpleasant sensations that occur in response to a usually benign stimulus. The prefix *dys* generally means “bad.” Dysesthesia has been described as “Dante-esque type of pain.”

6. Define the terms light touch, two-point discrimination, and stereognosis.

- **Light touch**—assesses the ability of the patient to perceive the application of soft brushing to the skin (eg, using camel-hair brush, piece of cotton, or tissue). The sensation of light touch is carried by the anterolateral system (spinothalamic) and dorsal column-medial lemniscal system. A person who has complete absence of light touch sensation generally has peripheral nerve or spinal cord damage.

- **Two-point discrimination**—assesses the ability of the patient to perceive the application of two points of contact applied simultaneously to the skin as one or two points. The determination of this is a function of the density of Merkel receptors in the skin (palm, high density of receptors; back, low density) and the integrity of the dorsal column-medial lemniscal system. Static two-point discrimination sense is transmitted to the spinal cord by slowly adapting large-diameter (type I) afferent nerves, while moving two-point discrimination testing evaluates rapidly adapting fibers.

- **Stereognosis**—the ability to recognize common objects (eg, keys, coins, pencil, paper clip, comb, fork) placed in the hand without visual clues. If the patient is unable to name the object, and other touch sensory modalities are intact, it suggests damage in the contralateral parietal cortex.

7. What is the interrater and intrarater reliability of the following?

A. **SHEMEES-WEINSTEIN MONOFILAMENT TESTING FOR LIGHT TOUCH**

Reports of interrater reliability for the assessment of light touch using Semmes-Weinstein monofilaments have ranged from good to only slight or fair, while intrarater reliability has been assessed as moderate to good. Inconsistency of standardized testing measures and variations in peripheral nerve tested and the presence or absence of pathology in the subject may explain the variation in reports on light touch reliability.

B. **VIBRATION SENSIBILITY TESTING**

Vibration testing stimulates pacinian corpuscles and assesses the function of large-diameter rapidly adapting peripheral nerves and the dorsal column-medial lemniscal central pathways. Using mechanical testing devices, the intrarater reliability of the assessment of vibration sense has been described as good. Moderate reliability has been reported for interrater reliability. Age and height were associated with minimal threshold values of the feet but not of the hands as determined through multiple regression analysis.

C. **TWO-POINT DISCRIMINATION SENSIBILITY TESTING**

Although numerous studies have described the reliability of two-point discrimination testing, interpreting these results to apply them to clinical practice has been hampered by the lack of standardized testing procedures and the inability to quantify subject cognitive function. Reliability testing has ranged from moderate and good to poor. The cooperation of the subject and the ability of the subject to attend to the stimulus have been suggested to influence two-point discrimination measures, as do central training effects.

There appears to be little carry-over between static two-point discrimination tests and function, although moving two-point discrimination testing (which tests rapidly adapting afferent fibers) has been shown to correlate with object identification tests. Likewise, the sensitivity of two-point discrimination testing to detect change over time is poor.

Reports of the reliability of two-point discrimination testing vary according to the age and sex of subjects, the peripheral nerve tested, and whether the subject is symptomatic or asymptomatic. Testing procedures also vary with the starting position (wide or narrow distances), the amount of pressure applied, and the instrument used to apply the stimulus.

It is questionable whether any reliability measures of sensibility can be used as a reference to judge the presence of pathology. It is recommended that results from any sensory testing procedures not be used as the sole means of developing diagnoses of peripheral or central nervous system origin.
8. Define kinesthesia versus proprioception.

Kinesthesia is the awareness of movement. The tester moves the subject’s extremity or joint passively through a small range of motion (~10 degrees) by holding bony prominences with a fingertip grip. The subject is asked to describe the direction (e.g., up, down, in, out) and the range (e.g., initial, mid, terminal) while the extremity/joint is in motion. Impaired kinesthesia indicates dysfunction in the peripheral nerves, spinal cord, brainstem, or cerebrum. Proprioception is joint position sense and awareness of joints at rest. The tester grips bony prominences and avoids stimulating touch (pressure) receptors that provide the subject with additional information (e.g., grip laterally to move the hallux up/down). The tester moves the subject’s extremity/joint through the range of motion and then holds it in a static position. The subject is asked to describe the position or duplicate it with the contralateral extremity. Proprioception is intact in individuals with cerebellar ataxia but is impaired in individuals with sensory ataxia (lesions in peripheral sensory nerves, dorsal roots, dorsal columns of the spinal cord, or medial lemnisci).

9. List some of the special neurologic tests, and explain their clinical importance.

<table>
<thead>
<tr>
<th>Test</th>
<th>Description</th>
<th>Response</th>
<th>Clinical Importance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Babinski’s sign</td>
<td>Plantar surface of foot is stroked with key or fingernail in a sweeping motion from posterior and lateral border toward ball of foot</td>
<td>Extension of great toe, with or without fanning of other toes</td>
<td>Indicates upper motor neuron lesion</td>
</tr>
<tr>
<td>Oppenheim reflex</td>
<td>Anterior border of tibia is stroked</td>
<td>Presence of Babinski’s sign</td>
<td>If Babinski’s sign is present, indicates upper motor neuron lesion</td>
</tr>
<tr>
<td>Hoffman’s sign</td>
<td>Distal phalanx of index, middle, or ring finger is subjected to rapid, gentle stroking</td>
<td>Reflexive flexion of thumb distal interphalangeal joint of any other finger not struck</td>
<td>If present, indicates upper motor neuron lesion</td>
</tr>
<tr>
<td>Bulbocavernous reflex</td>
<td>Dorsum of penis is tapped</td>
<td>Retraction of bulbocavernous portion of penis and contraction of anal sphincter</td>
<td>Absence of reflex indicates damage to pudendal nerve, sacral autonomic efferent nerves, or upper motor neuron</td>
</tr>
<tr>
<td>Abdominal reflex</td>
<td>Upper or lower abdominal musculature is gently stroked</td>
<td>Motion of umbilicus toward stroking</td>
<td>Reduction or absence indicates upper motor neuron damage or involvement of pertinent spinal level reflexes (T7–T9, upper abdominal region; T11–T12, lower abdominal region)</td>
</tr>
<tr>
<td>Romberg’s sign</td>
<td>Subject stands with feet close together and then closes eyes</td>
<td>Subject increases sway or falls with eyes closed</td>
<td>Indicates dorsal (sensory) column disease or pathology (e.g., cervical spondylosis, tumor, tabs dorsalis) or peripheral neuropathy; unsteady with feet together and eyes open or closed indicates central nervous system dysfunction (e.g., cerebellar ataxia, vestibular disorder)</td>
</tr>
<tr>
<td>Rapid alternation movements</td>
<td>Subject performs rapid forearm pronation and supination, or ankle plantar flexion and dorsiflexion</td>
<td>Inability to perform movement (dysdiadochokinesia)</td>
<td>Indicates ipsilateral cerebellar dysfunction, especially lateral hemispheres</td>
</tr>
<tr>
<td>Test</td>
<td>Description</td>
<td>Observation</td>
<td>Notes</td>
</tr>
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<td>----------------------</td>
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<td>-----------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------</td>
</tr>
<tr>
<td>Finger to nose</td>
<td>Subject extends finger away from face and then toward nose, and repeats this movement</td>
<td>Subject able to perform movement smoothly, correctly estimating distances and location</td>
<td>If movement is not smooth or there is overshotting or undershotting of movement, then may indicate cerebellar dysfunction (asynergy)</td>
</tr>
<tr>
<td>Tandem gait</td>
<td>Subject walks in a straight line while touching the heel of the one foot to the toe of the other</td>
<td>Subject able to perform task without unsteadiness or deviation from the line</td>
<td>Unsteadiness and inability to maintain tandem stance indicative of potential truncal ataxia as a result of cerebellar vermis pathology; rule out peripheral neuropathy</td>
</tr>
<tr>
<td>Tactile extinction</td>
<td>Apply random single and double simultaneous stimuli (eg, touch the subject’s left hand followed by touching both the left hand and right face)</td>
<td>Inability to detect a stimulus only in the presence of another stimulus</td>
<td>Typically associated with parietal lobe pathology; occasionally as a result of frontal lobe pathology; may involve either right or left hemisphere</td>
</tr>
<tr>
<td>Hoover’s test</td>
<td>Subject supine, tester places one hand under each heel; subject instructed to perform a straight leg raise</td>
<td>Tester should feel downward pressure under contralateral heel</td>
<td>Inability to raise leg and absence of pressure under contralateral heel is suggestive of nonorganic paresis (eg, malingering, conversion disorder)</td>
</tr>
</tbody>
</table>

10. **Who was Babinski?**

Joseph Felix François Babinski was a French physician born in Paris in 1857. He trained under the famed neurologist Jean Martin Charcot. Babinski first described the "cutaneous plantar reflex" in 1896. In that paper, he described the existence of a similar response in infants that was present until approximately 7 months of age. Babinski attributed the presence of this reflex in adults to involvement of the pyramidal tract. The Babinski sign is also called the extensor plantar response.

11. **Define referred pain and radicular pain.**

- **Referred pain**—pain that is felt at a site removed from the source of involvement. It may be caused by irritation of a nerve root or by tissue supplied by the same nerve root. Cardiogenic pain may be referred to the left axilla and left arm region because of the shared sensory distribution of the T2 spinal nerve (intercostobrachial nerve), and spinal segments that receive afferent pain from the gallbladder also receive afferent input from the shoulder region. Patients commonly describe referred pain as constant, poorly localized or diffuse, or aching.
- **Radicular pain**—a specific type of referred pain that is felt in a dermatome, myotome, or sclerotome of an involved peripheral nerve root. Compression of the C5 nerve root may affect sensation on the anterior shoulder. Patients commonly describe radicular pain as sharp, shooting, band-like, electric, or zinging.

12. **What is a burner or a stinger?**

A burner or a stinger is a traction or compression injury to a cervical nerve root or brachial plexus trunk. Often the injury involves the C5 or C6 nerve root or upper trunk of the brachial plexus, with burning, numbness, tingling, or weakness in the distribution of the involved root or trunk. The mechanism of injury can involve distraction of the pectoral girdle from the neck, either through excessive shoulder girdle depression or through forced hyperlateral flexion of the neck. Forced oblique hyperextension of the neck may also cause ipsilateral compression injuries of the cervical nerve roots or upper brachial plexus trunks.
13. How accurate is reflex, sensory, and muscle strength testing in the diagnosis of cervical radiculopathy?
The testing accuracy of reflexes, sensation, and muscle strength in the diagnosis of cervical radiculopathy is considered fair. Side-to-side reflex changes and muscle weakness associated with cervical radiculopathy agree with surgical findings about 77% of the time, while sensory loss correlates approximately 65% of the time. The combination of segmental hyporeflex, dermatomal sensory loss, and myotomal weakness is most specific (99%) for predicting cervical root disease. Myotomal weakness is the most accurate single variable predictor of cervical radiculopathy (81% accuracy).

14. What is the prognosis for individuals with cervical radiculopathy?
According to a review of the literature, up to 90% of patients improve with conservative treatment, with greater support for an active versus a passive treatment approach. The following five factors are suggested to adversely affect outcomes at 6 months: history of reoccurring cervical radiculopathy for more than 5 years, more than three episodes, bilateral paresthesia, female over 50 years of age, and worsening symptoms. Predictors for short-term successful outcomes with physical therapy intervention for cervical radiculopathy include individuals less than 54 years of age, dominant arm unaffected, symptoms do not worsen when looking down, a multimodal treatment approach including manual therapy, cervical traction, and deep neck flexor muscle strengthening for at least 50% of all visits. When three of four of these variables are present, there is a positive likelihood ratio of 5.2 and the posttest probability of success is 85%; when all four variables are present, there is a positive likelihood ratio of 8.3 and the posttest probability of success is 90% (data calculated based on the following outcome measures: Neck Disability Index, Patient-Specific Functional Scale, Numeric Pain Rating Scale, and Global Rating of Change).

15. What is the minimal clinically important difference (MCID) and minimal detectable change (MDC) for common outcome measures for individuals with cervical radiculopathy?
The Neck Disability Index: MDC ranges from 10.2 to 13.4; the MCID ranges from 7.0 to 8.5. The Patient-Specific Functional Scale: MDC ranges from 2.1 to 3.3; MCID ranges from 2.0 to 2.2. The Numeric Pain Rating Scale: MCID of 2.2; MDC of 4.1.

16. What signs and symptoms are the most useful in diagnosing lumbosacral radiculopathy?
Segmental reflex loss is the most accurate single predictor of lumbosacral radiculopathy (63%). There is a 97% probability of lumbosacral radiculopathy secondary to disc bulge if straight leg raise (SLR) test is positive (0.78–0.97 sensitivity), contralateral SLR test is positive (0.85–1.0 specificity), lumbar flexion reproduces pain, and lumbar extension is decreased but not limited by pain. Other highly specific tests for lumbosacral radiculopathy include 1) the combination of abnormal myotomal weakness, abnormal sensation, abnormal reflexes, and a positive SLR (specificity, 1.0), 2) extensor hallucis longus weakness (specificity, 0.7), 3) ankle dorsiflexion weakness (specificity, 0.70), and 4) ankle plantarflexion weakness (specificity, 0.95).

17. What is the best strength test to determine weakness of the quadriceps in patients with known L3–L4 radiculopathy?
In L3 and L4 radiculopathy, unilateral quadriceps weakness was best detected by a single leg sit-to-stand test. This may not always be practical; thus testing of the knee extensors in flexion is the next best method, followed by knee extensor strength testing in the knee-extended position.

18. How valuable are the Achilles tendon reflex and the Hoffmann reflex in detecting L5/S1 root compression?
The Achilles tendon reflex and the Hoffmann reflex (H-reflex) are not valuable in detecting L5 root compression. However, they are valuable in detecting S1 root compressions. The H-reflex (electrically stimulated) is more specific (91%) than sensitive (50%) when evaluating S1 radiculopathy. The Achilles tendon jerk is also more specific (90%) than sensitive (32%) when evaluating S1 radiculopathy.

19. What is a syrinx?
A syrinx is a neuroglial cell-lined, fluid-filled cavity. The Latin word syrinx means “tube.” When this occurs within the spinal cord, the condition is known as syringomyelia; in the brainstem it is called syringobulbia. The cause of a syrinx is not fully understood; possible mechanisms of pathology are associated with the accumulation of cerebrospinal fluid within the spinal cord or brainstem, genetic malformations, and the proliferation and subsequent regression of embryonic cell rests. The syrinx generally is restricted to the cervical and upper thoracic regions, with extensions into the medulla occurring occasionally. If the cavity is
within the central canal, it will interrupt the decussating spinothalamic tract fibers, and the patient will experience bilateral loss of pain and temperature sensations around the level of the lesion. If the cavity extends laterally into the lateral funiculus of the spinal cord, the lateral corticospinal pathway will be involved, with ipsilateral upper motor neuron signs and symptoms. Patient symptoms may include gastrointestinal disturbances, including nausea, vomiting, eating disturbances, and weight loss. This is often caused by involvement of neural regions that mediate esophageal reflexes and gastrointestinal reflexes. Joint arthropathy may be seen in individuals with syringomyelia. It has been reported that syringomyelia is the second most common cause of Charcot’s joint.

20. What is Horner syndrome?

Horner syndrome is a disease in which there is an interruption of sympathetic nervous system innervation to the head and face region. It is usually caused by a brainstem lesion. Signs and symptoms include miosis (constricted pupil from uncompensated parasympathetic nervous system input to pupil), ptosis (drooping of the eyelid from lost sympathetic innervation of the levator palpebrae superioris tarsal muscle), enophthalmos (eyeball appears to be sunken into its socket), anhidrosis (absence of sweat production on the affected side of the face), and flushing (increased superficial blood flow on the affected side of the face).

21. Differentiate the following common symptoms associated with vestibular disorders: dizziness, vertigo, lightheadedness, dysequilibrium, and oscillopsia.

- Dizziness—vague sensation of whirling, lightheadedness, or feeling as though one will fall.
- Vertigo—the illusion of movement (eg, the environment is spinning) and is typically associated with unilateral vestibular hypofunction (UVH), benign paroxysmal positional vertigo (BPPV), or a unilateral central (brainstem) lesion affecting the vestibular nuclei.
- Lightheadedness—a feeling of imminent fainting typically as a result of nonvestibular disorders (eg, hypotension, hypoglycemia, anxiety).
- Dysequilibrium—the feeling as though one is off balance and may be indicative of bilateral vestibular hypofunction (BVH), chronic UVH, BPPV, or diminished somatosensation or weakness in the lower extremities.
- Individuals with “oscillopsia” perceive objects known to be stationary as moving. This can occur with vestibular hypofunction in which the vestibulo-ocular reflex (VOR) is diminished (the VOR maintains stability of images on the retina during head movements).

22. What is benign paroxysmal positional vertigo (BPPV)? What is the most common positional test to examine it, and what interventional approaches are employed to treat it?

BPPV, a biomechanical disorder, is the most common peripheral vestibular pathology. Symptoms include nystagmus and vertigo with head position changes, dysequilibrium, and occasionally nausea. BPPV is theoretically caused by dislodged otoconia in the semicircular canals in which the otoconia becomes adhered to the cupula (cupulolithiasis) or floats freely (canalithiasis). The Dix-Hallpike test is used to examine for BPPV: subject sits in a long-sit with head rotated 45 degrees to one side and is moved to a supine position with the head extended 30 degrees beyond horizontal while maintaining the 45-degree rotation; the tester observes the eyes for nystagmus. BPPV caused by canalithiasis is treated using the canalith repositioning maneuver (moving the subject’s head through a sequence of positions to move the otoconia out; movements depend on side and canal affected). BPPV caused by cupulolithiasis is treated using the Liberatory (Semont) maneuver (rapidly moving the subject through positions in order to dislodge the otoconia). Brandt-Daroff exercises for BPPV involve 5 to 20 repetitions, two to three sets per day: the subject moves from sitting to sidelying with the head rotated 45 degrees toward the ceiling, the subject remains in this position for 30 seconds or until the vertigo stops, returns to sitting, and repeats on the other side.

23. What are the diagnostic criteria, common symptoms, and prognosis for individuals with concussion or mild traumatic brain injury (mTBI)?

Diagnostic criteria for concussion, also known as mTBI, typically include normal structural imaging (eg, computed tomography scan), a Glasgow Coma Scale score of 13 to 15, loss of consciousness length of time 0 to 30 minutes, and posttraumatic amnesia or altered mental state of 0 to 1 day. Common symptoms are physical (headache, postural/balance impairments, visual deficits (eg, blurred vision, light sensitivity), nausea, sleep disturbance, fatigue, and tinnitus), cognitive (decreased attention, concentration, memory, processing, and executive function), and behavioral (irritability, anxiety, depression, agitation, aggression, and impulsivity). Regarding prognosis, individuals typically fully recover within 3 months; 10% to 20% may experience postconcussive syndrome and continue to report symptoms years post injury.
CHAPTER 19 QUESTIONS

1. What signs/symptoms are the specific in detecting lumbosacral radiculopathy?
   a. Segmental reflex loss and myotomal weakness
   b. Ankle plantarflexion weakness and segmental reflex loss
   c. Dermatomal numbness, segmental reflex loss, and myotomal weakness
   d. **Positive straight leg raise, dermatomal pain, segmental reflex loss, and myotomal weakness**

2. You administer the Neck Disability Index (NDI), Patient-Specific Functional Scale (PSFS), and Numeric Pain Rating Scale (NPRS) during an examination for a patient with cervical radiculopathy. When writing goals, in accordance with achieving the minimal clinically important difference for each measure, you state that the patient will demonstrate improvement by:
   a. 7 points on the NDI, 2 points on the PSFS, and 3 points on the NPRS
   b. 10 points on the NDI, 1 point on the PSFS, and 3 points on the NPRS
   c. 7 points on the NDI, 2 points on the PSFS, and 1 point on the NPRS
   d. 5 points on the NDI, 2 points on the PSFS, and 3 points on the NPRS

3. What test is the most valuable in detecting an L5 root compression?
   a. Electromyography
   b. Nerve conduction studies
   c. Achilles tendon reflex testing
   d. Hoffman reflex

4. During an examination, the patient is unable to identify the direction of movement or the range of the movement when you move the patient’s limb passively. You document a deficit in:
   a. Stereognosis
   b. Proprioception
   c. **Kinesthesia**
   d. Light touch

5. A patient describes symptoms of vertigo and nystagmus with changes in head position. You suspect benign paroxysmal positional vertigo (BPPV). To examine for BPPV, you perform:
   a. The Liberatory (Semont) maneuver
   b. Brandt-Daroff movements
   c. **A Dix-Hallpike test**
   d. The Canalith repositioning maneuver
1. What is research?
Research is a controlled, systematic approach to obtain an answer to a question. Experimental research involves the manipulation of a variable and measurement of the effects of this manipulation. Non-experimental research does not manipulate the environment but may describe the relationship between different variables, obtain information about opinions or policies, or describe current practice. Basic research is generally thought of as laboratory-based research, in which the researcher has control over nearly all aspects of the environment and subjects. Clinical or applied research usually uses entire, intact organisms in a more natural environment.

2. What are variables?
Variables are measurements or phenomena that can assume more than one value or more than one category. A categorical or discrete variable is one that can assume only certain values and often is qualitative (no quantity or numeric value implied). Continuous variables are ones that can assume a wide range of possible values and are usually quantitative in nature.

3. Define independent variable and dependent variable.
- Independent variable—the variable that is manipulated by the researcher
- Dependent variable—the variable that is measured by the researcher

Independent variables often are qualitative, and dependent variables usually are quantitative. The different permutations of the independent variable are called levels. To be an independent variable, there must be at least two levels; if some aspect of the research has only one possible value or category, it is a constant.

4. Describe other types of variables.
Extraneous or confounding variables are phenomena that are not of interest to the researcher but may have an effect on the value of the dependent variable. Extraneous variables must be controlled as much as possible, usually by holding some aspect of the research constant. A covariate is a phenomenon that affects the dependent variable and is not of interest to the researcher but that the researcher is unable to control.

5. How accurate are measurements?
The observed measurement of any phenomenon is composed of a true score and error. Error may be systematic, in which case all scores are increased or decreased by a constant amount, or random. Systematic error, generally, is the result of using the measurement instrument incorrectly or improper calibration of the instrument. Random error is precisely that—random. Even if the true score is constant, and there is no systematic error, repeated measurements of a phenomenon do not produce identical scores. It is generally assumed that the effects of all of the sources of random error cancel each other, such that the measured score is the best estimate of the true score. If the true score is constant, repeating the measurement and calculating an average score may be a better estimate of the true score. If the true score is labile or is altered as a consequence of the measurement, repeated measurements may reduce the accuracy of the measurement.
6. Define measurement reliability.
Reliability is related to consistency or repeatability. In the absence of a change in the true score, how similar are repeated measurements of the same phenomenon? Intrarater reliability is a measure of how consistent an individual is at measuring a constant phenomenon, interrater reliability refers to how consistent different individuals are at measuring the same phenomenon, and instrument reliability pertains to the tool used to obtain the measurement. If a measurement cannot be performed reliably, it is difficult to ascribe changes in the dependent variable to the effects of the independent variable, rather than measurement error.

7. Describe statistical procedures used to estimate reliability.
The intraclass correlation coefficient (ICC), which is based on an analysis of variance (ANOVA) statistical procedure, is a popular means of estimating reliability. A means of measuring absolute concordance is the kappa statistic. In the past, a Pearson or Spearman correlation procedure often was used to estimate reliability; these procedures are insufficient as measures of reliability because they measure covariance, not agreement.

8. Define measurement validity.
Measurement validity is an indication of whether the measurement is an accurate representation of the phenomenon of interest. Some clinical measurements have obvious validity. For example, using a goniometer to measure the angle between two bones with the joint as the axis is generally accepted as a valid indication of the status of the tissue that limits motion at that joint. For other measurements, the relationship between what is measured and what is inferred from the measurement is more tenuous. To establish the validity of a clinical test, a more direct measurement that is considered a gold standard is established. If acceptable numbers of patients with a positive Lachman’s test have anterior cruciate ligament tears and those without tears have a negative Lachman’s test, the Lachman’s test is considered a valid test for anterior cruciate ligament integrity. There is no universal definition of acceptable numbers; this is left to the researcher to defend and the clinician to accept or reject.

9. What is a research design?
A research design is a plan or structure of the means used to answer the research question or to gather the information for a nonexperimental study. There are three basic designs for experimental research:

1. A completely randomized design uses a single independent variable and assigns different groups of subjects to each level of the independent variable. Because each subject receives only one type of treatment, this design is also called a between-subjects design. If the independent variable is the type of brace and there are three levels (ie, three different braces are being used), then an individual subject would be measured while using only one of the three braces.

2. A repeated measures design uses a single independent variable and measures each subject under all levels of the independent variable. If the independent variable is the dosage of a drug and levels are 200, 400, and 600 mg/day, then each subject would be measured while taking each of the three dosages.

3. A factorial design uses two or more independent variables. A completely randomized factorial design is one in which all of the independent variables are independent factors, meaning an individual subject is measured under only one condition. If the two independent variables are type of brace and dosage of a drug, and there are three levels of each variable, then nine groups of subjects would be studied. A within-subjects factorial design measures each subject in all levels of all variables. Using the brace and dosage variables, each subject would be measured with each brace and dosage (eg, brace A and 200 mg, brace A and 400 mg, brace A and 600 mg). A mixed factorial design uses at least one independent factor and at least one repeated factor. If subjects are assigned to only one brace but are measured with all three drug dosages, the design is mixed.

10. Which descriptive statistics are most useful for describing a set of data?
It depends on the data. If the data are distributed normally, the three measures of central tendency are equal; in this case, the mean is most often used to describe the typical performance. If there are a few scores at one extreme or the other in the set of data, the median is considered the best
measure of central tendency. For example, in the data set 2, 4, 5, 7, 83, the mean is 20.2, and the median is 5; 5 is more descriptive of the typical score than 20.2. The standard deviation (or variance) is the most descriptive value for the variability of a data set that is distributed normally, and minimum-maximum may be the best measure of variability in data sets that are best described with the median.

11. Are the terms normal distribution, bell curve, and Gaussian distribution equivalent? Yes, in that all three terms refer to the shape of a frequency histogram constructed using the scores from any measurement that is the sum of a true score and multiple, small, independent sources of error. Nearly any physiologic or anatomic parameter that is measured in a large group of individuals falls into a normal distribution. For example, suppose the maximal aerobic capacity is measured in 500 individuals selected at random. The scores are counted and grouped into increments of five (e.g., the number of subjects with a maximal aerobic capacity of 0–5, 6–10, 11–15), and the results are used to construct a bar plot with the increments on the x-axis and the number of individuals in each bin on the y-axis. If the average value was 36, and the standard deviation was 6, the resulting plot might look like the figure. Most scores were between 31 and 35, with fewer scores at each extreme. For example, the number of scores in the 6 to 10 range is approximately equal to the number of scores in the 51 to 55 range. In a perfectly normal distribution, 68% of the scores will be found within one standard deviation of the mean; in this example, 340 of the 500 scores should be between 26 and 38 (32 ± 6), 95% of the scores will be within two standard deviations of the mean, and 99% of the scores will be within three standard deviations of the mean.

12. Are there distributions other than a normal distribution? Yes, especially with small samples, skewed distributions are possible. A skewed distribution results when there are a few extreme scores at one end or the other of the distribution. For example, if most of the scores are low, but there are a few high scores, the distribution might be similar to the figure. This distribution is skewed to the right by the few extremely high scores. If there are a few extremely low scores, the distribution is skewed to the left. The direction of the skew is determined by drawing (or imagining) a line connecting the top of each bar in the histogram and stating to which side of the plot the tail extends.
13. Can the same concepts be used with a skewed distribution; that is, are 68% of the scores within one standard deviation of the mean?

No. These values hold true only for a normal distribution. In the case of a skewed distribution, the median is a better descriptor of the typical score, and the minimum-maximum better describes the variability in the set of data.

14. What are inferential statistics?

When data are collected, the researcher needs to determine the probability of obtaining a particular set of scores by chance alone. The procedures used to calculate this probability are called inferential statistics and are the heart of testing an experimental hypothesis. There are different procedures used based on the research design, the nature of the research question (what the researcher is trying to answer), and the nature of the data.

15. Describe the fundamental concept of inferential statistics.

In the simplest case, consider a randomized design, with a single independent variable having two levels and a single dependent variable. Suppose a researcher posed the following question: what is the effect of adding neural glide techniques for the median nerve to the standard treatment for patients with carpal tunnel syndrome? The independent variable is treatment, and the levels are standard and neural glide. The dependent variable could be number of days until the patient is free of symptoms for 10 consecutive days. A sample of patients with carpal tunnel syndrome is selected at random from the population of patients with carpal tunnel syndrome, and the patients in the sample are assigned at random to one of the two treatment levels. Because the patients have been selected at random from the population and then assigned at random to one of the two treatment groups, it is a reasonable assumption that the mean and standard deviation for the dependent variable would be the same for both treatment groups if there is no effect of adding neural glide to the standard treatment. All of the subjects are treated until the criterion for discharge is met (ie, free of symptoms for 10 consecutive days), and the data are summarized. If the standard group recovered in an average of 40 days, with a standard deviation of 7 days, and the neural glide group recovered in 32 days, with a standard deviation of 6 days, did the treatment work? There is a difference in the average days to recovery, but is that difference large enough to conclude that it was as a result of the neural glide, or could it be attributed to chance alone? Perhaps the subjects in the neural glide group did not have as severe compression of the median nerve at the beginning of the study and recovered more quickly despite the neural glide. The essence of inferential hypothesis testing is to answer the following question: what is the probability of having obtained a difference in days to recovery of this magnitude as a result of random factors? If this probability is low enough, the researcher can conclude that the treatment had a beneficial effect and should become a part of standard practice.
16. How is the correct statistical test chosen?

The short answer is that it depends on the question being asked:

- If the desire is to learn about the association between two variables (e.g., the relationship between thigh girth and knee extensor force), a correlation coefficient should be calculated.
- If the question concerns prediction (e.g., if a patient has knee range of motion of 5–60 degrees on the second postoperative day, how many days will the patient likely remain in the hospital?), a regression analysis is appropriate.
- If the question is whether a treatment has an effect (e.g., does spinal traction reduce the signs and symptoms of a lumbosacral root compression?), a chi-square, analysis of variance (ANOVA), or t-test, which is a special case of the ANOVA, is appropriate.

However, because there are different types of data and different types of restrictions placed upon the testing, the answer is more complicated. There are four levels of data: nominal, ordinal, interval, and ratio. Information measured on a nominal scale results in a name only; that is, it does not imply a quantity. Left versus right and red versus blue are examples of nominal data. If a numeral is assigned to information on a nominal scale, a quantity is not implied; if red is coded 1 and blue is coded 2, it does not mean that blue is twice as much as red.

An ordinal scale implies a rank order, with some quantitative value. The person who finishes a race first receives the number 1, meaning this person finished the race in a shorter time than the second place finisher. However, the amount of time between first and second place is not likely the same as the amount of time between fifth and sixth place.

For statistical purposes, there are no meaningful differences between an interval and a ratio scale; both imply not only a rank order but also an equivalence between points on the scale. The difference between 80 and 95 is the same as the difference between 25 and 40; in both cases, it is 15.

For a correlation study, a Spearman rho (for Spearman, who developed the procedure, and rank order) is used for ordinal data. A Pearson correlation coefficient is calculated for interval data. In both cases, the coefficient can vary between $-1.00$ and $+1.00$. A value of zero means that there is no correlation, and a value of 1.00 signifies the correlation is perfect. If the sign is $+$, the value of one variable increases as the other increases. If the sign is $-$, the value of one variable decreases as the other increases.

For experimental studies, those designed to determine whether there is a difference, a chi-square is computed for data that are nominal. There is some disagreement regarding the appropriate analysis when the data meet the definition of ordinal or interval. It is almost universally agreed upon that to perform a traditional ANOVA, the set of data should have a normal distribution, and the variance of the sets should be similar (the definition of similar is usually lacking; a rule of thumb is that the variance of one set should be no more than twice the other set). There are formal tests that can be used to determine whether the data are normally distributed and whether the variances are equal; these are beyond the scope of this book and are generally of little or no interest to the clinician. Some authors further state that the data must meet the definition of interval or ratio; in fact, some researchers ignore the more important requirements of normal distribution and equality of variance and claim that the tests are robust enough that any data on an interval or ratio scale can be analyzed with a traditional ANOVA. However, the scale of the data was not an issue when the traditional ANOVA approach was developed. Therefore if the data are normally distributed, and the variances are equal, then a traditional ANOVA is appropriate, regardless of the scale of the data. Often, especially with the small sample sizes usually used in rehabilitation research, the two requirements of normal distribution and equality of variance are not met, even with ratio data, and a traditional ANOVA is inappropriate.

If the question is whether two groups differ on the dependent variable, and the data are normally distributed with equal variances, a t-test is appropriate. The t-test is a special case of the ANOVA, developed to make the calculations easier. With software, it is just as easy to use an ANOVA because the information is the same. If there are more than two levels of a single independent variable, or if there is more than one independent variable, the ANOVA can be extended to handle the variables. The type of ANOVA performed is often referred to by the number of rows and columns that are required to represent all of the permutations of the independent variables. A $2 \times 3 \times 2$ ANOVA means that there were three independent variables (because there are three numerals), two of the independent variables had two levels, and the third variable had three levels (the value of the numerals). The exception is a $1 \times 4$ ANOVA, which has only one independent variable, with four levels; if the value of one of the numerals is 1, it cannot represent a variable (because, by definition, variables have more than one possible value). If the data are not normally distributed, or the variances are not equal, a nonparametric equivalent is appropriate.
17. Differentiate between parametric and nonparametric statistical procedures.

Parametric statistical procedures are performed on data that have a normal distribution, such as the distribution observed in a population. Nonparametric procedures are performed on data that do not have a normal distribution, that is, a skewed distribution, as often is observed in a sample. Parametric procedures include the ANOVA and t-test, and nonparametric procedures include the chi-square, Kruskal-Wallis, and Spearman rho. As mentioned earlier, some authors add the requirement that the data have the characteristics of an interval or ratio scale in order to conduct parametric procedures, but this is debatable. Nonparametric procedures are often regarded as second-class procedures, used only when the data are extremely skewed. However, nonparametric procedures are nearly as powerful as their parametric equivalents when the data are normally distributed and more powerful than parametric procedures when the data are skewed. Because of the small sample sizes typically used in orthopedic and rehabilitation research, nonparametric procedures should likely be used more often.

18. How is the appropriate type of statistical analysis determined?

19. Other than intuition and clinical experience, how can the best clinical tests be identified?

The performance of clinical tests (eg, straight-leg raise, Lachman test, shoulder impingement tests) can be measured in many ways, some more enlightening than others. The point of a clinical test is to sort patients into two basic categories: those who truly have the disorder and those who truly do not have the disorder. Depending on the situation, disease, dysfunction, or pathology can be substituted for the term disorder.

It is often difficult or hazardous to know with absolute certainty whether a disorder is present. For example, the definitive test for a ruptured anterior cruciate ligament (ACL) is direct visualization of the ligament, with an arthrotomy, arthroscopy, or, potentially, MRI. Obviously, it would be unreasonably hazardous to subject all patients with a clinical history suggestive of an ACL rupture to a surgical procedure, and MRI is expensive. These definitive tests are considered gold standards against which the results of a less invasive or less expensive test are compared.

The typical approach to establishing the performance of a clinical test is to conduct both the clinical test and the definitive test (gold standard) on a group of patients, some of whom have the disorder and some of whom are free of the disorder. Specific values are then calculated, and the clinician can determine how confident one can be in the results of the test. The clinical test is not always what is typically considered a test: It can be a specific question asked during the patient interview (such as, “Did you hear a pop before your knee gave way?”), or it can be a combination of tests and interview information, such as whether the straight-leg raise is positive and the patient has pain radiating from the back to the buttock and down the posterior thigh.

**Samples Used**

<table>
<thead>
<tr>
<th>PURPOSE OF ANALYSIS</th>
<th>NATURE OF DISTRIBUTION</th>
<th>INDEPENDENT</th>
<th>RELATED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Show a difference</td>
<td>Normal</td>
<td>ANOVA</td>
<td>Repeated measures ANOVA</td>
</tr>
<tr>
<td></td>
<td>Skewed</td>
<td>Chi-square for frequency; Mann-Whitney or Kruskal-Wallis</td>
<td>McNemar’s for frequency; Wilcoxon signed rank</td>
</tr>
<tr>
<td>Determine degree of association</td>
<td>Normal</td>
<td>Pearson or linear regression</td>
<td>Pearson or linear regression</td>
</tr>
<tr>
<td></td>
<td>Skewed</td>
<td>Contingency coefficient for frequency; Spearman rho</td>
<td>Contingency coefficient for frequency; Spearman rho</td>
</tr>
</tbody>
</table>
20. What is meant by sensitivity, specificity, positive predictive value, and negative predictive value?

These terms are used to describe the usefulness of the clinical tests described previously. It is easiest to comprehend these values if a $2 \times 2$ table is constructed, with the results of the definitive test entered in the columns, and the results of the clinical test entered in the rows. A study conducted by Roach et al. can illustrate the calculation and use of these values. Among other variables, the researchers determined the usefulness of asking patients with degenerative disk disease (DDD) and low back pain whether they also had pain radiating down the lower member; this was the clinical test used to predict the presence of spinal stenosis (the target disorder). Out of 17 patients with the target disorder (spinal stenosis), 16 had a positive clinical test (ie, they had pain radiating down the lower member). Out of 89 patients with DDD and low back pain, but without the target disorder, 70 had pain radiating down the lower member. The table illustrates how to calculate the values.

<table>
<thead>
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<th>STENOSIS</th>
<th>NO STENOSIS</th>
<th>ROW TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radiating leg pain</td>
<td>Positive</td>
<td>a = 16</td>
<td>b = 70</td>
</tr>
<tr>
<td>Negative</td>
<td>c = 1</td>
<td>d = 19</td>
<td>c + d = 20</td>
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<td>a + c = 21</td>
<td>b + d = 89</td>
<td>a + b + c + d = 106</td>
</tr>
</tbody>
</table>

Sensitivity is the proportion of patients with a disorder who also have a positive clinical test; it is the probability of having a true positive test. It is calculated by dividing the number of patients with the target disorder and a positive test by the number of patients with the target disorder: Sensitivity = a / (a + c). Using the example above, 16 / (16 + 1) = 0.94. This means that of 100 patients with stenosis, 94 will have pain radiating down the lower member.

Specificity is the proportion of patients without the disorder who also have a negative clinical test; it is the probability of having a true negative test. It is calculated by dividing the number of patients without the target disorder and a negative test by the number of patients without the target disorder: Specificity = d / (d+b). Thus 19 / (19+70) = 0.21. This means that of 100 patients with DDD, but without stenosis, only 21 will not have pain radiating down the lower member.

Sensitivity and specificity deal with reality; they are based on knowing for certain whether the target disorder is present. The reason clinicians use a clinical test in the first place is that they are trying to determine whether the target disorder is present; reality usually is unknown.

21. Do other performance characteristics depend on a knowledge of reality?

No. Positive predictive values (PPVs) and negative predictive values (NPVs) deal with the situation of having a patient and the results of a clinical test. This is the usual situation that confronts a clinician.

PPV is the proportion of patients with a positive clinical test who also have the target disorder. It is calculated by dividing the number of patients with a positive clinical test and the target disorder by the total number of patients with a positive clinical test: PPV = a / (a+b) = 16 / (16+70) = 0.19. This means that of 100 people with pain radiating down the lower member, only 19 will have stenosis.

NPV is the proportion of patients with a negative clinical test who also do not have the target disorder. It is calculated by dividing the number of patients with a negative clinical test and free of the target disorder by the total number of patients with a negative clinical test: NPV = d / (d+c) = 19 / (19+1) = 0.95. This means that of 100 people without pain radiating down the lower member, 95 will not have stenosis.

22. What is the principal drawback to the PPV and NPV?

These values change with changes in the prevalence of the target disorder; if the target disorder is uncommon, there are many more false-positive results, and the PPV goes down. Because the sensitivity and specificity deal with reality, they are not affected by changes in the prevalence of the target disorder.
23. Is there a way to combine the best characteristics of sensitivity, specificity, PPV, and NPV?
Yes; likelihood ratios are often considered a useful approach for clinical decision making. Likelihood ratios are expressed as odds and are calculated from values used to calculate sensitivity and specificity. The likelihood ratio of a positive test (LR+) is the quotient of the sensitivity and the complement of the specificity, ie, the sensitivity divided by 1 minus the specificity. In the previous example, the LR+ is 0.94 / (1 − 0.21), or 1.19. This means that the odds of a patient with the target disorder (ie, stenosis) having a positive test (ie, radiating leg pain) are 1.19 times greater than those for a patient without the target disorder. Another way of viewing a LR+ value is that it gives the odds that a patient with the target disorder would be expected to have a positive test. A likelihood ratio of 1.00 is of no value; a patient with a positive test is equally likely to have the target disorder as not.

The likelihood ratio of a negative test (LR−) is the quotient of the complement of the sensitivity and the specificity—that is, 1 minus the sensitivity divided by the specificity. In this example, the LR− is 1 − (0.94 / 0.21) = 0.29. This means that the odds of a patient with the target disorder (stenosis) and a positive test are only 29% as great as those of a patient without the target disorder. An alternative way of viewing the LR− is to determine the inverse of the LR− (or divide the specificity by the complement of the sensitivity) and use this value to decide how much more likely an individual without the target disorder is to have a negative test than an individual with the target disorder. The reciprocal of 0.29 is 3.5 (or 0.21 / [1 − 0.94] = 3.5); therefore an individual without stenosis has odds 3.5 times greater of having a negative test than an individual with stenosis. Either approach is appropriate, but some clinicians find the second method more intuitive. An LR− value of 1.00 is equivalent to flipping a coin to determine the meaning of a negative test.

24. Define the terms prevalence and incidence.
Prevalence is the proportion of a population that has a particular disorder or condition at a specific point in time. If in a population of 233,658 there are 253 individuals with carpal tunnel syndrome (CTS), the prevalence of CTS is 253 / 233,658 = 0.0010828. Because prevalence typically is a small number, it usually is multiplied by an appropriate constant and expressed as the number of cases per 1000 or 10,000. In this example, the prevalence of CTS would be about 1 per 1000.

Incidence is the rate of development of new cases of a disorder in a particular at-risk population over a given period of time. As with prevalence, the value usually is small and is multiplied by an appropriate constant and expressed as the number of cases per the constant for a given period of time. If a new manufacturing plant opens and employs 2355 people, and 89 people develop CTS during the calendar year from January 1, 1999, to December 31, 1999, the incidence of CTS is (89 / 2355) × 1000 = 38 cases for that 1-year period. One difficulty in calculating incidence is in determining the denominator; it is unlikely that there will be 2355 people employed by the plant on January 1 and December 31. If the population is not constant, the size of the population at some point is selected to represent the size for the entire time period; usually, it is the midpoint of the time period, that is, July 1, 1999, in our example. Another difficulty is in defining the at-risk population. If one is determining the incidence of pregnancy, obviously males, premenarche girls, and postmenopausal women would not be included in the denominator. In the manufacturing plant that employs 2355 people, it may be that only the 985 people who work with impact tools are at risk for CTS.

25. Discuss risk ratios and odds ratios.
Risk ratios and odds ratios are used to determine how likely it is that an individual with a particular risk factor will or will not develop a disease. The calculation of these ratios is similar to the calculation of likelihood ratios, PPVs, and NPVs. A risk ratio is calculated by dividing the incidence for the disorder for one group by the incidence for the disorder for another group; the two groups are considered to be at risk or not at risk. For example, if the manufacturing plant employs 2355 people and 985 of the employees use impact tools, the following question could be asked: “what is the risk of an employee who uses impact tools developing carpal tunnel syndrome (CTS) compared with an employee who does not use impact tools?” A 2 × 2 table could be constructed as follows:
The incidence (expressed as a proportion) of CTS in the at-risk group is \( \frac{a}{a + b} = \frac{297}{985} = 0.30 \), and the incidence of CTS in the not-at-risk group is \( \frac{c}{c + d} = \frac{43}{1370} = 0.03 \). The risk ratio is then \( \frac{\frac{a}{a + b}}{\frac{c}{c + d}} = \frac{0.30}{0.03} = 10 \). An individual who uses impact tools is 10 times more likely to develop CTS compared with an individual who does not use impact tools. As with PPVs and NPVs, which also are calculated using the data in the rows of the table, the risk ratio is changed easily by changes in the prevalence of the condition; the rarer the disorder, the higher the risk ratio.

An odds ratio is calculated using the information in the columns of the table, and similar to sensitivity and specificity, the odds ratio is not changed by changes in prevalence. The odds that someone with CTS uses impact tools is \( \frac{a}{c} = \frac{297}{43} = 6.9 \). The odds that someone without CTS uses impact tools is \( \frac{b}{d} = \frac{688}{1327} = 0.52 \). The odds ratio is \( \frac{\frac{a}{c}}{\frac{b}{d}} = \frac{6.9}{0.52} = 13.3 \), which means that someone with CTS is 13.3 times more likely to use impact tools. In contrast to the risk ratio, the odds ratio is not changed by changes in prevalence of the disorder.

### 26. Discuss how a clinician can judge the effectiveness of a treatment or prevention program.

One approach to assessing treatment effectiveness is by using relative risk reduction (RRR), absolute risk reduction (ARR), and the number needed to treat (NNT) estimates. To illustrate the use of these concepts, consider the effectiveness of an educational program (a back school) for the reduction of the incidence of low back pain (LBP) in an industrial setting. The fundamental question is whether a back school reduces the rate of LBP, and if it does, is it cost-effective? Because a history of LBP before attending the back school is likely to have an impact on the development of LBP during the study period, people enrolled in the study would need to be divided into those with and those without a history of LBP. After completing the back school, the subjects would be followed for a period of time, and the number of cases of LBP that occur among the subjects with and without a history of LBP would be recorded for subjects who had attended and who had not attended the back school. These data along with the calculation of the incidence are used to produce the RRR, ARR, and NNT values:

### Incidence of LBP (As a Proportion)

<table>
<thead>
<tr>
<th></th>
<th>Control (C), no back school attendance</th>
<th>Experimental (E), back school</th>
<th>RRR</th>
<th>ARR</th>
<th>NNT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prior history of LBP</td>
<td>Yes</td>
<td>0.43</td>
<td>0.17</td>
<td>0.61</td>
<td>0.26</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>0.13</td>
<td>0.06</td>
<td>0.54</td>
<td>0.07</td>
</tr>
</tbody>
</table>

The RRR values of 0.61 and 0.54 signify that the risk of developing LBP is reduced by 61% and 54% among individuals with a prior history of LBP and individuals without a prior history of LBP, respectively. What is missing from the RRR is the fact that the ARR for individuals without a history of LBP is relatively trivial. The reciprocal of the ARR yields a value that is potentially useful; the NTT is the number of people who would have to attend the back school to prevent an episode of LBP in one person. Therefore four people with a prior history of LBP should be sent to the back school to prevent the development of LBP in one person. Of the people who do not have a history of LBP, 14 need to attend the back school to prevent LBP in one
A researcher conducts a comprehensive meta-analysis comparing Oswestry scores of patients after 2 weeks of intervention for acute low back pain. The treatments compared were spinal manipulation versus education and walking as tolerated. The following figure provides the results of the research. Based on this figure, answer the following questions.

<table>
<thead>
<tr>
<th>Study name</th>
<th>Outcome</th>
<th>Statistics for each study</th>
<th>Std diff in means and 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Std diff</td>
<td>Lower limit</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Standard error</td>
<td>Variance</td>
</tr>
<tr>
<td>York, 2011</td>
<td>Oswestry</td>
<td>-0.502</td>
<td>0.323</td>
</tr>
<tr>
<td>Adams, 2005</td>
<td>Oswestry</td>
<td>-0.235</td>
<td>0.321</td>
</tr>
<tr>
<td>Bennett, 2008</td>
<td>Oswestry</td>
<td>0.087</td>
<td>0.243</td>
</tr>
<tr>
<td>Stanley, 1998</td>
<td>Oswestry</td>
<td>-0.443</td>
<td>0.206</td>
</tr>
<tr>
<td>Russell, 2012</td>
<td>Oswestry</td>
<td>-0.750</td>
<td>0.156</td>
</tr>
<tr>
<td>Miller, 2013</td>
<td>Oswestry</td>
<td>-0.353</td>
<td>0.280</td>
</tr>
<tr>
<td>Dunn, 2007</td>
<td>Oswestry</td>
<td>1.140</td>
<td>0.221</td>
</tr>
<tr>
<td>Fisher, 2011</td>
<td>Oswestry</td>
<td>0.616</td>
<td>0.214</td>
</tr>
<tr>
<td>Jackson, 2009</td>
<td>Oswestry</td>
<td>-0.600</td>
<td>0.202</td>
</tr>
<tr>
<td>Ward, 2013</td>
<td>Oswestry</td>
<td>-0.266</td>
<td>0.117</td>
</tr>
<tr>
<td>Overall</td>
<td>Oswestry</td>
<td>-0.192</td>
<td>0.063</td>
</tr>
</tbody>
</table>

Comparison of manipulation versus walking therapy for acute low back pain.

27. What is this type of plot called?
A forest plot.

28. Of the 10 studies that met the inclusion criteria for the study, how many resulted in a statistically significant difference between the treatment groups, which treatment was favored, and what is the basis for your determination of statistical significance? Six reported a statistically significant difference (Stanley, Russell, Jackson, and Ward favored manipulation, whereas Dunn and Fisher favored walking). If the line representing the 95% confidence interval does not include a standardized difference between the means of 0, the difference is statistically significant at an alpha level of 0.05.

29. Was the overall analysis statistically significant? How do you know the answer from the figure alone? Yes, there was a statistically significant difference overall, in favor of manipulation. This is demonstrated by the diamond symbol, which does not include the value of zero difference.

30. Which study had the most subjects? How can you tell? Ward, 2013, had the most subjects based on the size of the symbol representing the standardized difference in means and 95% confidence interval (CI) for that study. The symbol size is proportional to the sample size in a forest plot.

31. What is the difference between linear regression and logistic regression? Linear regression uses one or more predictor variables (sometimes called independent variables) to estimate the value of a predicted variable (sometimes called the dependent variable) when the predicted variable is a continuous variable. Logistic regression is fundamentally the same concept, except the predicted variable is a categorical variable.

32. Describe a situation where linear regression would be helpful. Suppose you wanted to provide a better prognosis for patients beginning treatment for chronic neck pain. You select the Neck Pain and Disability Scale (NPAD, scored on a 0–100 scale) as the outcome measure. Based on a thorough review of the evidence, you determine that you will measure five variables...
at the beginning of treatment and determine the NPAD score again after 3 weeks of treatment. After collecting data from a sample of patients, you then create a regression equation using the five predictor variables to estimate the final NPAD score of the sample. This equation could then be used for patients in the future to estimate the NPAD score after 3 weeks of treatment.

33. What does a linear regression equation include?

The general form for the regression equation is \( \hat{y} = m_1x_1 + m_2x_2 + \ldots + m_x + b \pm \text{error} \)

The \( \hat{y} \) is the estimated value of the predicted variable (read as “\( y \) hat”), the \( m \) is the slope of the regression line for each predictor variable, the \( x \) is the value of the predictor variable, \( b \) is the \( y \)-axis intercept (a constant), and error is the standard error of the estimate. Suppose in the NPAD example given earlier, age in years, duration of symptoms in weeks, and whether litigation was pending were found to be meaningful predictors of the final NPAD score. The final equation could be

\[
\text{NPAD} = 0.87(\text{age}) + 1.43(\text{duration}) + 3.55(\text{litigation}) + 3.3 \pm 4
\]

To then calculate the estimated final NPAD score, you would multiply the patient’s age in years by 0.87, add the product of the duration of symptoms in weeks by 1.43, then add the product of the value of the litigation variable times 3.55, and add 3.3. The actual value will be within ± 8 points for 95% of your patients, assuming the patients are similar to those who constituted the sample used to derive the equation.

34. How can you multiply 3.55 times the litigation variable?

Litigation is a categorical variable and must be coded as 0 or 1. In this case if litigation was pending, the value of 1 is assigned, so litigation adds 3.55 to the predicted score. If litigation is not pending, it is coded as a 0, and that term drops from the equation.

35. Describe a situation where logistic regression would be helpful.

Basically the same situation as for linear regression, but the predicted variable is categorical instead of continuous. Suppose you wanted to predict whether patients who were undergoing a total hip arthroplasty would be able to be discharged or would need to be transferred to a facility for further rehabilitation. This is a categorical variable (“home” or “not home”) and could be coded as 0 for “not home” and 1 for “home.” Obviously, the outcome could be coded 1 for “not home” and 0 for “home,” but the key is that the logistic regression equation will predict the status coded as “1.” A logistic regression equation can be used just as a linear regression equation, but the value predicted is the probability of group membership in the group coded as “1.” Suppose in the discharge environment example one predictor variable was the preoperative 6-minute walk distance measured in meters, and the Exp(B) for that variable is 7.3. The predicted membership was in the group “not home.” This means that if comorbidities are present, the odds of being discharged “not home” are 7.3-fold greater than if the patient does not have comorbidities. Remember, the odds ratio helps adjust the odds of the outcome, not the probability.

36. Is this how logistic regression equations are typically used?

No. Instead, the results of the equation are expressed in odds ratios, usually called “Exp(B).” Suppose in the discharge environment example, a researcher reports that one predictor variable was “comorbidities” and was coded as 0 if “none” and 1 if “present” (so a categorical predictor of a categorical result), and the Exp(B) for that variable is 7.3. The predicted membership was in the group “not home.” This means that if comorbidities are present, the odds of being discharged “not home” are only 0.93 as large as an individual who walked only 1 meter less.

37. Does the same process hold true for continuous predictor variables?

Fundamentally, yes, but with a very important distinction. If the predictor variable is continuous, the Exp(B) gives the change in odds of membership in the group coded as “1” for each unit change in the predictor variable. Suppose in the discharge environment example one predictor variable was the preoperative 6-minute walk distance measured in meters, and the Exp(B) for this variable is 0.93. Typically, an odds ratio of 0.93 is considered a trivial, unimportant value, but in this case, it means that for each additional meter walked in this test, the odds of being in the group discharged “not home” are only 0.93 as large as an individual who walked only 1 meter less.

38. Describe the approach to probability proposed by Thomas Bayes.

Bayesian statistics is a general term used to describe an approach to probability proposed by Thomas Bayes in the mid-18th century. Because of the complexity of the computations and the
development of other approaches to data (now called classical statistics), the method did not gain widespread use until the 1950s with the development of computers. The essential elements of Bayesian statistics is that the probability of an event can be best estimated when the baseline probability is adjusted when new information becomes available. The terms commonly used are “prior” for the baseline probability, “likelihood” for the new information, and “posterior” for the final probability statement.

39. What is Bayes’s Theorem?

\[ p(A \mid B) = \frac{p(B \mid A) \cdot p(A)}{p(B)} \]

\( p(A \mid B) \) is the probability of the event A if the evidence is present, \( p(B \mid A) \) is the probability of the evidence being present if the outcome (the event) also occurs, \( pA \) is the probability of the event given no additional evidence, and \( pB \) is the probability of the evidence being present irrespective of the outcome.

40. How can Bayes’s Theorem be applied to clinical practice?

The most obvious example is in the use of a diagnostic test. Suppose the following data were collected from a sample of patients.

<table>
<thead>
<tr>
<th>Truth about Disease</th>
<th>Present</th>
<th>Absent</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Test result</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>30</td>
<td>4</td>
<td>34</td>
</tr>
<tr>
<td>Negative</td>
<td>7</td>
<td>24</td>
<td>31</td>
</tr>
<tr>
<td></td>
<td>37</td>
<td>28</td>
<td>65</td>
</tr>
</tbody>
</table>

\( p(A) \) is the probability of the disease being present before any knowledge of the test result is obtained. Here, this value is \( 37/65 = 0.569 \), and it is the same as the prevalence of the disease.

\( p(B) \) is the probability of the evidence being present irrespective of the disease. Here, this value is the proportion of positive test results, \( 34/65 = 0.523 \).

\( p(B \mid A) \) is the probability of the evidence being present if the outcome is also positive, \( 30/37 = 0.811 \), and this is the same as the sensitivity of the test.

Solving this equation gives a value of 0.882, and it is the probability of the disease being present given a positive test result. From a diagnostic perspective, this is the same as the posttest probability and will be the same result if one calculates the sensitivity, specificity, and likelihood ratios.

41. Are there other uses for a Bayesian approach?

Yes, this approach can be used for inferential analysis of events (categorical data) from a randomized controlled trial. Although there are passionate proponents for the Bayesian approach and equally passionate proponents for classical analysis, the two approaches complement each other and may well be best used together.

BIBLIOGRAPHY

CHAPTER 20 QUESTIONS

1. The mean of a data set is 57 and the median is 50. Which of the following best describes this distribution?
   a. Normal  
   b. Skewed to the right  
   c. Skewed to the left  
   d. Skewed, but in an unknown direction

2. Patients with an acute onset of low back pain are randomly allocated to two groups. Group A receives NSAID medication and a booklet describing the pathology and treatment of low back pain in general terms and is advised to walk at a comfortable pace for 30 minutes per day. Group B receives NSAID medication and a booklet describing the pathology and treatment of low back pain specifically for that subject's pain according to the treatment-based classification method and is advised to perform specific therapeutic exercise for 30 minutes per day. After 3 weeks, 13 of 25 patients in Group A and 8 of 29 patients in Group B continue to experience activity-limiting low back pain. How many patients would need to be treated as Group B subjects were treated to prevent one patient continuing to experience activity-limiting low back pain after 3 weeks compared with treating every patient as Group A subjects were treated?
   a. 4  
   b. 6  
   c. 12  
   d. 15

3. A researcher reports that patients with knee pain and a history of a twisting injury have an Exp(B) of 3.52 for having a final diagnosis of an ACL tear. What does this mean?
   a. The probability of an ACL tear in those with a twisting injury is 3.52-fold greater than in those without a twisting injury.  
   b. That for every patient with an ACL tear, but no history of a twisting injury, there are 3.52 patients with an ACL tear and a history of a twisting injury.  
   c. The odds of an ACL tear in those with a twisting injury are 3.52-fold greater than the odds of an ACL tear in those without a twisting injury.  
   d. It is not possible to determine the meaning of this value without also knowing the p value.
1. Define the terms evidence-based medicine (EBM) and evidence-based practice (EBP).

- **EBM**—David Sackett originally defined EBM as “the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients. It means integrating individual clinical expertise with the best available evidence.” The later definition was broadened to “the integration of best research evidence, clinical expertise and patient values…when these three elements are integrated, clinicians and patients form a diagnostic and therapeutic alliance which optimizes clinical outcomes and quality of life.”

- **EBP**—the application of this EBM framework to clinical practice, which encompasses EBM now, is central to the development of clinical practice guidelines and cost-effectiveness studied in health care or organizational policy.

2. What does it mean to practice EBP?

- Asking clear, concise, and relevant questions about one’s patients that are readily answerable with a literature search
- Efficiently and effectively searching the available literature for articles that might answer the questions
- Evaluating the merits of the most relevant articles from the search result and assessing the validity and value of the most important and strongest articles for practice
- Implementing the findings in the care of patients by forming a new practice pattern (eg, consistent and overwhelming evidence against a therapeutic intervention from the literature or no positive effect ever shown) or supporting the practice pattern begun with the patient.

3. Define the different study types in a hierarchical manner.
• Systematic reviews and meta-analyses—systematic reviews and meta-analysis are syntheses, appraisals of evidence, to provide information on effectiveness of treatment or accuracy of diagnosis/ prognosis, which guide clinical practice. Meta-analyses are a quantitative systematic review, given RCTs of high quality and commeasurable outcomes. Meta-analyses attempt to quantify what a given effect size will be for an intervention or the precision and accuracy of a diagnostic test.

• Randomized controlled trials—individuals are selected at a specific time in the history of a diagnosis and randomly allocated to two or more groups; one is an intervention group and the other group is a control, or no intervention, group. Randomization reduces the risk of bias caused by group differences. These studies are sine qua non for evaluating cause-effect and therapeutic efficacy.

• Cohort study—individuals assembled at a specific time in the history of a diagnosis are divided into two groups, and one group receives an intervention and the other does not. Efforts are made to match the groups by characteristics.

• Case-control study—this is a retrospective study of patients compared with characteristic-matched subjects who are not ill or have not received an intervention.

• Case series—in this expansion of a case study, the investigator describes observations of a series of similar cases.

• Descriptive study—this case study is designed to analyze factors important to cause, care, and outcome of the patient’s problem. These studies are most important for generating hypotheses.

4. Why are randomized controlled trials considered the ‘high-level’ evidence in studies of treatment effectiveness?
Randomization of subject assignment helps avoid selection bias in the control and intervention groups by matching of characteristics in the groups. Other research designs in randomized controlled trials that reduce the risks of biases include the blinding of the assignment of interventions to the patients and the provider, if possible (ie, double blinding); concealment of outcomes; and monitoring for contamination from other interventions. Randomized controlled trials can discern causal relationships with interventions (ie, treatment) from other causes (eg, spontaneous recovery) across populations.

5. What is a systematic review?
A systematic review is a thorough review and summary of the research on a particular topic about a clinical problem. The review has a specific methodology that distinguishes it from a general review. Systematic reviews should capture the homogeneity or heterogeneity of the various study methods and designs. The review’s conclusions should come from studies that ask the same research questions. Simply comparing the number of positive studies with negative studies is inadequate. Systematic reviews include an assessment of the quality of the various studies and weighting of studies, with higher weight given to larger studies and randomized controlled trials. Systematic reviews often precede the development of an RCT or clinical guideline on a topic. The Cochrane collaboration is the ‘gold standard’ for systematic reviews worldwide. The Cochrane collaboration is a global network of health practitioners, researchers, patient advocates, and others who provide high-quality systematic reviews, synthesis, and recommendations from these exhaustive reviews. http://www.cochrane.org/.

6. What is a meta-analysis?
A meta-analysis is a variety of systematic reviews that use statistical techniques to combine and summarize quantitative results for similarly constructed studies. This method of combining the results of many studies allows an estimate of the magnitude of intervention or risk factor effect and subgroup analysis. Meta-analysis requires a high degree of homogeneity among the studies examined in terms of design, methodology, and reporting of data. Meta-analyses should be understood as narrow presentations of relevant research on a particular topic, designed to provide more precisely the positive or negative direction of an effect.

7. What are common databases used to access evidence related to clinically generated questions?
• The Center for Evidence-Based Medicine: www.cebm.net
• PEDro (Physiotherapy Evidence resource): www.pedro.org.au
• American Physical Therapy Association: www.PTNow.org
• Medline/PubMed: www.nlm.nih.gov/bsd/pmresources.html
• MedlinePlus: www.nlm.nih.gov/medlineplus/
• Ovid: www.ovid.com
• Researchgate: www.researchgate.net (a means for researchers to share their work online)
• TRIP: www.tripdatabase.com
8. What does it mean to ‘push’ the evidence?
‘Pushing the evidence’ expresses the ‘evidence’ is actively delivered to the practitioner, usually via e-mail. The journal of the APTA (www.ptjournal.apta.org) and the American Academy of Orthopedic Manual Physical Therapy (AAOMPT) offer pushed evidence services, as do private corporations such as Evidence In Motion (EIM). An organizational subscription service for physicians, Essential Evidence Plus (http://www.essentialevidenceplus.com/), offers daily POEMs as pushed assessed evidence.

9. What is a gold standard versus a reference standard in a study of a diagnostic test?
A gold standard test is a test that is as near as possible to 100% specificity and 100% sensitivity. Reference standards are criteria tests that approximate the definitive diagnosis but are not as accurate as a gold standard test. Reference standards are most often imaging studies (eg, MRI, ultrasound) or surgical examination (eg, arthroscopy).

10. How is the pretest probability determined?
Experienced skilled clinicians rely on personal experience and a cognitive process called heuristics or diagnostic rules of thumb. Individual clinicians should develop a sense of their clinic’s patient population and prevalence of disease by referral. Remember that your clinic has different sources of referrals, different clinical expertise and experiences, and different overall patient population than other clinics.

Pretest probability is also established by mechanism of injury, natural history of humans, and the patient’s history. Stratford and Binkley provide an excellent example of a clinician establishing the pretest probability of a meniscus tear based on the clinician’s knowledge of mechanism of injury, natural history, and factors from the patient’s history. They present three different patients: a 14-year-old female volleyball player with anterior knee pain, a 21-year-old soccer player who twisted her knee while kicking a ball, and a 37-year-old furnace repairman who twisted his knee squatting. The estimated pretest probabilities are 1% in the volleyball player, 50% in the soccer player, and 95% in the furnace repairman:

\[
\text{Pretest odds} = \frac{\text{prevalence}}{1 - \text{prevalence}} = \frac{\text{pretest probability}}{1 - \text{pretest probability}}
\]

Soccer player’s pretest probability = 50% = 0.50
Pretest odds = 0.50 / 0.50 − 0.50 = 1 : 1

11. What is the difference between a positive likelihood ratio (+LR) and a negative likelihood ratio (−LR)?
The positive LR is the probability of a positive test if the disease is present (ie, Sn) divided by the probability of a negative test if the disease is absent, whereas the negative LR is the probability of a negative test if the disease is present divided by the probability of a negative test if the disease is absent (ie, Sp).

\[
\text{Sn} / 100\% - \text{Sp} = \text{LR} + \\
100\% - \text{Sn} / \text{Sp} = \text{LR} -
\]

12. What is meant by the terms “SnNouts” and “SpPins”?
These phrases are mnemonics that have been proposed to help remember the most useful aspects of tests with moderate to high sensitivity and specificity.
- SnNout—a test with a high sensitivity value (Sn) that when negative (N) helps to rule out a disease (out)
- SpPin—a test with a high specificity value (Sp) that when positive (P) helps to rule in a disease (in)

13. What are the advantages of sensitivity, specificity, and likelihood ratios over the concept of predictive values (positive predictive value and negative predictive value [PPV and NPV])?
PPV and NPV are the proportion of persons with a positive (or negative) test result who have (or do not have) a disease. The predictive value is the posttest probability of the disease. The problem with predictive values is that they are variable by the population prevalence of a disease or pretest probability of having a disease.

Sensitivity, specificity, and likelihood ratios are properties of the tests themselves and are stable or unchanged by various population risks, prevalence, or pretest probabilities. The advantage lies in the ability to compare tests or to apply these statistics to various populations without recalculation of the values. PPV and NPV will change; sensitivity, specificity, and likelihood ratios are constants.
14. What is a clinical prediction rule (CPR) or clinical decision rule (CDR)?
A clinical prediction rule is a clinical tool that quantifies the contribution of history, physical examination, and basic laboratory testing toward establishing a diagnosis, prognosis, or likely response to treatment in an individual patient.

15. What are some examples of clinical prediction rules (CPRs) for diagnosis?
- Ottawa ankle rule (Stiell et al., 1992)
- Ottawa knee rule (Stiell et al., 1995)
- Canadian C-spine rule (Stiell et al., 2001)
- NEXUS cervical spine study (Hoffman, 1998)

16. What is an example of a clinical prediction rule (CPR) for determining the likely response to treatment in an individual patient?
Flynn et al. developed a CPR for predicting which low back pain patients are most likely to benefit from spinal manipulation. The study found that if four of the five following clinical findings were positive the patient would most likely benefit from spinal manipulation (LR+ = 23):
- Episode of pain < 16 days
- Pain does not extend below the knee
- FABQ work subscore < 19
- Internal rotation of at least one hip > 35 degrees
- Stiffness identified at one or more lumbar segment

17. When should we accept a clinical prediction rule (CPR) or clinical decision rule (CDR) as validated?
McGinn et al. have suggested three stages in development and validation of a CPR: 1) derivation of strong predictive rules, 2) validation in a study of similar clinical settings and application in multiple clinics with variable prevalence and disease outcomes, and 3) an impact analysis of a physician’s behavioral changes and improved patient outcomes and/or reduced cost. CPRs often are in the early stages of development (eg, just derivations of the rules published), and these rules likely should not be used in the clinic. For CPRs that have been broadly validated, the second part of the second step should be considered in decision making (ie, McGinn et al. label this ‘level 2 evidence’ or ‘rules that can be used in various clinical settings with confidence in their validity’).

18. What is comparative effectiveness (CE)?
Comparative effectiveness is comparing two or more treatments to determine which is most effective. In terms of a controlled clinical study, this means comparing a treatment of interest to another treatment, instead of a placebo. Comparative effectiveness can be determined by evaluating similar treatments, different treatments, or determining which types of patients might benefit most from a particular therapy. In terms of CE research, cost utility analyses compare the ratio of dollar costs of a treatment and some quantified measure of benefit (such as quality-adjusted life years) for two or more treatments. CE can be determined by using one or more research designs, such as randomized controlled trials (RCTs), studies of claims records, or medical registries. Different comparative research methods are summarized in the following table.

<table>
<thead>
<tr>
<th>RESEARCH METHOD</th>
<th>ADVANTAGES</th>
<th>DISADVANTAGES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Randomized controlled trials</td>
<td>Yield more definitive results than any other method</td>
<td>Expensive (costs can exceed $100 million) Takes a long time to conduct Typically studies efficacy rather than effectiveness Narrowly define patient population, so results may not be generalizable Objectivity of industry-sponsored RCTs is questionable</td>
</tr>
</tbody>
</table>
19. What is a clinical practice guideline (CPG)?
A CPG has been defined as a “systematically developed statement to assist practitioner and patient decisions about appropriate health care for specific clinical circumstances.” An example would be Low Back Pain: Clinical Practice Guidelines Linked to the International Classification of Functioning, Disability, and Health from the Orthopedic Section of the American Physical Therapy Association.

20. What are levels of evidence for primary research questions?

<table>
<thead>
<tr>
<th>LEVEL</th>
<th>TYPE OF EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Evidence obtained from high-quality diagnostic studies, prospective studies, or randomized controlled trials</td>
</tr>
<tr>
<td>II</td>
<td>Evidence obtained from lesser quality diagnostic studies, prospective studies, or randomized controlled trials (eg, weaker diagnostic criteria and reference standards, improper randomization, no blinding, &lt;80% follow-up)</td>
</tr>
<tr>
<td>III</td>
<td>Case-controlled studies or retrospective studies</td>
</tr>
</tbody>
</table>

Information for this table taken from a report of the Congressional Budget Office and from information formerly posted on the Academy Health website. (www.academyhealth.org)
From AAOS.org.
Levels of evidence refer to a way to classify study quality using a hierarchical rating system with five levels of evidence applied to four types of studies, including therapeutic, prognostic, diagnostic, and economic/decision analysis.

21. What is a grade of recommendation?

<table>
<thead>
<tr>
<th>Grades of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>GRADES OF</strong></td>
</tr>
<tr>
<td><strong>RECOMMENDATION</strong></td>
</tr>
<tr>
<td><strong>A</strong></td>
</tr>
<tr>
<td><strong>B</strong></td>
</tr>
<tr>
<td><strong>C</strong></td>
</tr>
<tr>
<td><strong>D</strong></td>
</tr>
<tr>
<td><strong>E</strong></td>
</tr>
<tr>
<td><strong>F</strong></td>
</tr>
</tbody>
</table>

The overall strength of the evidence supporting recommendations made in these guidelines will be graded according to guidelines described by Guyatt et al. (1995), as modified by MacDermid and adopted by the coordinator and reviewers of this project. In this modified system, the typical A, B, C, and D grades of evidence have been modified to include the role of consensus expert opinion and basic science research to demonstrate biological or biomechanical plausibility. From Delitto A, George S, Van Dillon L, Whitman JM, Sowa G, Shekelle P, Denninger TR, Godges JJ. Low Back Pain: Clinical Practice Guidelines Linked to the International Classification of Functioning, Disability, and Health from the Orthopedic Section of the American Physical Therapy Association. J Orthop Sports Phys Ther. 2012;42(4):A1–A57. http://dx.doi.org/10.2519/jospt.2012.0301.

Recommendation grades are based on the strengths of evidence and are based on the quality and applicability of the study, whether there are critical outcomes reported, or potential harm to patients. Strength is equivalent to the quality of available evidence.

---

22. What are registries?
Registries are a mechanism to monitor, develop, and improve different types of interventions and diseases. A registry is a database of a specific disease, treatment, and outcomes of care collected for analysis and synthesis and is often used for continuous quality improvement of the medical system. A total joint registry, eg, the Swedish Knee and Hip Registry (www.myknee.se & www.shpr.se) is a prospective observational study in which data are collected prospectively regarding hip and knee arthroplasty, primary and revision, but are analyzed retrospectively. Any registry requires a commitment by the profession to support it and collect the data, a strong core team to develop the register, and a restricted number of data to be collected.

23. What are levels of data?
Registry data include patient identifiers and demographics or patient-related data and procedure-related data, such as surgical and clinical outcome data.
- Level I data: patient, surgeon, hospital identifiers, and procedure data
- Level II data: patient factors, comorbidities, surgical data, perioperative care, and complications
- Level III data: patient-reported outcome measures that are generic and disease specific include general health, symptoms, function, activity level, and patient satisfaction
- Level IV data: radiograph analysis

### Levels of Data

<table>
<thead>
<tr>
<th>LEVEL I DATA</th>
<th>LEVEL II DATA</th>
<th>LEVEL III DATA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Personal identification</td>
<td>Comorbidities</td>
<td>Patient-reported outcome</td>
</tr>
<tr>
<td>Sex</td>
<td>Surgical complications</td>
<td>outcome</td>
</tr>
<tr>
<td>Laterality</td>
<td>Height and weight</td>
<td>Socioeconomic data</td>
</tr>
<tr>
<td>Diagnosis</td>
<td></td>
<td>An adverse event</td>
</tr>
<tr>
<td>Death</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Comorbidities</td>
<td>Prophylactic measures</td>
<td>Costs</td>
</tr>
<tr>
<td>Surgical complications</td>
<td>Surgical measures</td>
<td></td>
</tr>
<tr>
<td>Height and weight</td>
<td>(technique, approach, fixation method, and</td>
<td></td>
</tr>
<tr>
<td></td>
<td>timing)</td>
<td></td>
</tr>
<tr>
<td>Implant information</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital identification</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surgeon identification</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reoperation and/or revision</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


### Bibliography

CHAPTER 21 QUESTIONS

1. The three domains of EBP are as follows:
   a. Best evidence, patient values, and professional guidelines
   b. Best evidence, insurance policy, and clinical expertise
   c. Best evidence, patient values, and clinical expertise
   d. Best evidence, professional judgment, and expertise

2. Which attribute is not part of the construct of a randomized controlled trial (RCT) of a treatment?
   a. Randomization of subjects into different intervention arms
   b. Allowing the patients to choose the intervention they will receive
   c. Concealment of the subject to the treatment, or placebo, they receive
   d. Concealment of the outcomes of the treatment, when possible

Haynes, R. B. (2002). What kind of evidence is it that evidence-based medicine advocates want health care providers and consumers to pay attention to? BMC Health Services Research, 2, 3 (Epub 2002 Mar 6).
3. If a study includes an exhaustive literature search, identifies high-quality evidence, and combines comparable outcome results in a statistical analysis to determine the magnitude of the effect (i.e., effect size), or risk factors, or subgroup analysis, then it is labeled a ____.
   a. Systematic review
   b. Narrative review
   c. Editorial commentary
   d. Meta-analysis

4. If a study has a very high sensitivity value (Sn) what answer is generally correct?
   a. If the test is positive it likely goes a long way toward ruling out a pathology
   b. If the test is positive it likely goes a long way toward ruling in a pathology
   c. If the test is negative it likely goes a long way toward ruling out a pathology
   d. If the test is negative it likely goes a long way toward ruling in a pathology

5. If a study has a very high specificity value (SP) what answer is generally correct?
   a. If the test is positive it likely goes a long way toward ruling out a pathology
   b. If the test is positive it likely goes a long way toward ruling in a pathology
   c. If the test is negative it likely goes a long way toward ruling out a pathology
   d. If the test is negative it likely goes a long way toward ruling in a pathology
1. How are different degrees of sprains classified?
   - Grade I—≤ 25% of ligament tearing, mild pain, and swelling without instability
   - Grade II—26% to 75% of ligament tearing, moderate pain and swelling, loss of range of motion, and slight instability
   - Grade III—total disruption of the ligament, resulting in severe pain and swelling, severe loss of range of motion, and joint instability

2. How are brachial plexus lesions classified?
   - Grade I—neurapraxia characterized by transient loss of motor and sensory nerve conduction with complete recovery in ≤ 2 weeks
   - Grade II—significant motor deficits and some sensory deficits lasting at least 2 weeks; full recovery is variable, ranging from 4 to 6 weeks to 1 year (average, 3 months)
   - Grade III—complete loss of nerve function with motor and sensory deficits lasting for at least 1 year with no appreciable improvement during this time

3. Is it dangerous for children and adolescents to lift weights?
   The American Academy of Pediatrics recommends that children and adolescents avoid Olympic-style lifting and power lifting until they have reached skeletal maturity. As long as the athlete uses proper technique with supervision and does not maximally lift, injuries should be minimal. The major injuries that occur are growth plate fractures, usually resulting from improper execution.

4. Describe a good youth strength training program.
   - Phase I: Education—introduce children to proper exercise technique, strength training guidelines, and safety procedures. Focus is on technique. Begin with 1 set of 10 to 15 repetitions in 2 to 3 nonconsecutive training sessions per week.
   - Phase II: Progression—increase gradually the overload placed on the various muscle groups. This may be achieved by increasing the resistance or the number of repetitions, sets, exercises, or training sessions; 2 to 3 sets of 8 to 12 repetitions may be appropriate. New multijoint exercises may be introduced. Monitor child’s response to the exercise session constantly.
   - Phase III: Function—depending on the goal of the workout, the volume of training may increase to 2 to 3 sets of 6 to 8 repetitions on the major muscle groups. If the child is ready, more challenging exercises can be incorporated. Skill technique is still a priority.

5. What is the appropriate initial treatment for someone with an acute sports injury?
   The acronym PRICEMMS is used to describe the initial care:
   - P = Protection from further injury (eg, use of crutches)
   - R = Rest from further activity of the injured part but not complete immobilization of the whole body
   - I = Ice to decrease metabolism and pain
   - C = Compression to minimize swelling
   - E = Elevation to minimize swelling
   - M = Modalities, such as electric stimulation for pain control
   - M = Medication, such as antiinflammatories
   - S = Support, such as taping or bracing

6. List general criteria for return to sport activity.
   - Complete resolution of acute signs and symptoms related to the injury
   - Full dynamic range of motion of all joints
• Adequate strength and proprioception to be able to perform expected skills
• No alteration in normal mechanics and good sport technique
• Performance of sport-specific activity at or above preinjury level
• Good mental and emotional state for return to sport
• Appropriate cardiovascular (aerobic or anaerobic) condition

7. Describe the miserable malalignment of the lower extremity.
   This is the posture used to describe the female athlete with wide hips, femoral anteversion, genu valgum,
   and overpronation of the foot. This type of posture may predispose the athlete to various knee injuries,
   such as patellofemoral pain or anterior cruciate ligament sprain.

8. Why do females sustain so many noncontact ACL injuries?
   Noncontact injuries to the ACL in the female athlete have increased over the past 30 years. Possible
   reasons for this increase include both modifiable and nonmodifiable factors:
   **NONMODIFIABLE FACTORS**
   • Physiologic rotatory laxity
   • Hypermobility of joints in females
   • Alignment: increased valgus at the knee
   • ACL size: smaller ligament with less strength
   • Notch size and shape: narrower notch or A-shaped
   • Hormonal influence: increased laxity during certain portions of the menstrual cycle
   **MODIFIABLE FACTORS**
   • Lands/cuts with excessive knee valgus, internal rotation
   • Tendency to fire quads before hamstrings
   • Lands from jump with straighter knees (ligament dominant)
   • Proprioceptive deficiency
   • Poor training resulting in deficient strength

9. How are contusions treated?
   Ice, compression, and active range of motion are used to treat contusions. Treatments such as heat,
   massage, ultrasound, and passive stretching should be avoided because of the possible development of
   myositis ossificans.

10. Name the most common mechanisms of injury in football resulting in permanent
    cervi cal quadriplegia.
    Axial loading in the form of spearing is the most common mechanism. Hyperflexion and hyperextension
    of the neck also may cause cervical fracture.

11. How is the transmission of pathogens such as HIV and hepatitis prevented?
    To prevent transmission of blood-borne pathogens such as HIV, hepatitis B, or hepatitis C, the following
    specific measures should be followed:
    • Pre-event—dressing of all open wounds with occlusive dressings
    • Use of gloves, disinfectants, bleach, and antiseptics for washing/cleaning surfaces and clothing
    • Receptacles for contaminated clothing, bandages, dressing, and needles; must be available on the
      side of the field of play and in the dressing rooms
    • Removal of players from play if active bleeding is present
    • Control of bleeding, covering of wound with occlusive dressing, and change of blood-stained
      clothing before return to play
    • Wearing adequate, appropriate protective equipment by players
    • Athlete education and empowerment with responsibility to report wounds
    • Caregiver precautions including using and changing of gloves between contacts
    • Covering of minor cuts and abrasions while on the field
    • Airway devices available for use in case of life-threatening emergencies
    • Contaminated areas (eg, mats) wiped down immediately and disinfected with bleach; area should be
      dry before being reused
    • Post-event—wounds should be reviewed and redressed
Soiled clothing and towels should be washed separately.
All personnel involved in coaching and support of a team should be trained in basic first-aid.

12. List injuries that may occur from a fall on an outstretched hand.
- Distal radial fracture
- Radial head fracture
- Scaphoid fracture
- Acromioclavicular separation
- Perilunate dislocation
- Glenoid labrum tear

13. How can a stress fracture of the femoral neck be identified?
These fractures (fatigue fractures) can develop from compression or distraction forces. Fractures resulting from distraction usually appear along the superior cortex of the femoral neck, whereas compressive loads result in fractures to the inferior cortex. Local tenderness at the greater trochanter may radiate into the inner thigh and groin. Pain is not relieved with a cortisone injection. Internal rotation usually is limited, whereas with trochanteric bursitis external rotation is limited. The athlete may complain of night pain. Radiographs may be normal initially. The most reliable test is a technetium bone scan that reveals a focal hot spot. Two special tests that may be helpful in determining femoral fracture are the patellar-pubic percussion test and the fulcrum test.

14. What is the most common athletic injury to the ankle, and what structures are involved?
Lateral ligament sprains are the most common ankle injury. The ligaments involved include the anterior talofibular ligament, calcaneofibular ligament, and posterior talofibular ligament. The anterior talofibular ligament is involved in 60% to 70% of all ankle sprains; a combination of the anterior talofibular ligament and calcaneofibular ligament constitutes 20%; and the remaining 10% consists of injury to the syndesmosis, deltoid, or posterior talofibular ligament.

15. Describe three functional tests that can be used to decide return to sport after anterior cruciate ligament injury.
- Single-leg hop for distance—the athlete stands on the test limb behind the starting line. When ready, the athlete is instructed to jump as far as possible, landing on the same limb. The best of three trials is used. The opposite limb is then tested.
- Single-leg vertical jump—the test is begun with determination of the initial reach height of the athlete. The subject stands erect with the dominant side next to the wall. When ready, the athlete jumps off one leg, as high as possible. The difference between the reach height and the jumped height is the absolute jump height. The test is then repeated on the opposite side.
- Cross-over hop test—a piece of tape that is 6 m in length is placed on the floor. The subject is instructed to perform four consecutive hops on a single limb, crossing over the center line with each hop. The opposite limb is then tested.

16. What is the limb symmetry index (LSI)?
The LSI is calculated for all three of the tests described in the previous question. The LSI is the involved score divided by the uninvolved score, multiplied by 100. An LSI of 85% is ideal for return to sport.

17. List some physiologic changes that occur to the aging athlete.

<table>
<thead>
<tr>
<th>SYSTEM</th>
<th>FUNCTION</th>
<th>DECREASES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>Maximal heart rate</td>
<td>10 beats/min per decade</td>
</tr>
<tr>
<td></td>
<td>Resting stroke volume</td>
<td>30% by age 85</td>
</tr>
<tr>
<td></td>
<td>Maximal cardiac output</td>
<td>20%–30% by age 65</td>
</tr>
<tr>
<td></td>
<td>Vessel compliance</td>
<td>BP 10–40 mm Hg</td>
</tr>
</tbody>
</table>

18. Are the aforementioned physiologic changes a natural part of aging?
Many of the changes may be caused by inactivity rather than the true aging process. Maintaining a consistent exercise program can combat many of these changes.

19. What value does athletic tape provide to a joint?
The effect of tape application is still being investigated by researchers. Possible explanations include improved joint stability, increased joint proprioception, and prevention of injury. Tape loosens during participation about 20 minutes after its initial application.

20. What is kinesiology tape and what is its use?
Kinesiology tape is an elastic tape that can be applied directly to the skin, and the amount of stretch applied depends on the purpose of the taping technique. The tape is felt to cause physiologic effects on several body systems. The systems that are affected by the body are thought to include the circulatory/lymphatic, neural, muscular, and fascial systems, as well as the joints. A recent systematic review by Parreira et al. found that kinesiology tape for the treatment of shoulder, knee, spinal, and foot pain was no better than sham taping.

21. Describe the female athlete triad.
This is a syndrome that results in a negative energy balance. The syndrome is characterized by eating disorders, amenorrhea, and osteoporosis. Anorexia nervosa and bulimia nervosa are the most common eating disorders in females who participate in sports such as gymnastics and distance running. The eating disorders accompanied by heavy training may lead to amenorrhea (cessation of menstrual cycle), and eventually the athlete may develop bone loss or osteoporosis. These athletes then are more susceptible to stress fractures. An eating disorder is a symptom of underlying emotional distress. Eating disorders impair athletic performance.

22. List potential side effects of anabolic-androgenic steroid use.
**GENERAL SIDE EFFECTS**
- Liver dysfunction
- Hair loss
- Immune system dysfunction
- Kidney malignancy
- Liver cysts
- Decreased high-density lipoproteins
- Increased low-density lipoproteins
- Aggressive behavior
- Depression
- Premature epiphyseal closure in children
- Migraine headaches

**IN MALES**
- Testicular atrophy
- Prostate gland problems

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**Age-Related Decreases in Functional Status (Continued)**

<table>
<thead>
<tr>
<th>SYSTEM</th>
<th>FUNCTION</th>
<th>DECREASES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory</td>
<td>Residual volume</td>
<td>30%–50% by age 70</td>
</tr>
<tr>
<td></td>
<td>Vital capacity</td>
<td>40%–50% by age 70</td>
</tr>
<tr>
<td>Nervous</td>
<td>Nerve conduction</td>
<td>1%–15% by age 60</td>
</tr>
<tr>
<td></td>
<td>Proprioception and balance</td>
<td>35%–40% by age 60</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>Bone loss</td>
<td>1% per year</td>
</tr>
<tr>
<td></td>
<td>&gt;35 years old</td>
<td>3%–5% per year</td>
</tr>
<tr>
<td></td>
<td>&gt;55 years old</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Muscle strength</td>
<td>20% by age 65</td>
</tr>
<tr>
<td></td>
<td>Flexibility</td>
<td>Degenerative diseases</td>
</tr>
<tr>
<td>Metabolic</td>
<td>Maximal oxygen uptake</td>
<td>9% per decade</td>
</tr>
</tbody>
</table>

23. List the symptoms, presentation, and treatment of heat exhaustion and heat stroke.

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Mental Status</th>
<th>Rectal Temperature (° F)</th>
<th>Skin</th>
<th>Sweat</th>
<th>Blood Pressure</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heat exhaustion</td>
<td>Fatigue, usually conscious</td>
<td>104</td>
<td>Pale</td>
<td>Perfuse</td>
<td>Narrow pulse pressure</td>
<td>IV fluids, electrolytes, cool with ice</td>
</tr>
<tr>
<td>Heat stroke</td>
<td>Disoriented, confused or incoherent</td>
<td>≥105</td>
<td>Flushed</td>
<td>May not be sweating</td>
<td>Low diastolic pulse pressure</td>
<td>IV fluids, cool with ice, transport to hospital</td>
</tr>
</tbody>
</table>

24. What actions can be taken to prevent heat exhaustion and heat stroke?
- Prevention requires careful monitoring of ambient temperature and humidity
- Regular hydration before, during, and after sports participation is a must
- Consumption of 8 to 16 ounces of water is required for every 15 minutes of strenuous exercise
- Rehydration with 24 to 40 ounces of water after exercise is needed
- Cold water absorbs faster than warm water in the gastrointestinal tract

25. Is extra protein needed when participating in athletics?
Yes. The recommended dietary allowance (RDA) for sedentary individuals is 0.8 g/kg per day. According to the American College of Sports Medicine, endurance athletes require 1.2 to 1.4 g/kg per day, and strength athletes require 1.2 to 1.7 g/kg per day. This protein requirement can be found in a normal diet; extra protein supplements are not necessary. Be aware that female athletes and amenorrheic athletes may not consume enough protein.

26. List examples of foods that contain 10 g of protein.
- 50 g of grilled fish
- 2 cups of cooked pasta
- 35 g of lean beef
- 2 cups of brown rice
- 40 g of turkey
- 3/4 cup of cooked kidney beans
- 2 small eggs
- 120 g of soybeans
- 300 mL of skim milk
- 60 g of nuts
- 3 cups of wheat flake cereal

27. What is glucosamine, and what is it used for?
Glucosamine is a nutritional supplement that has been used for individuals with osteoarthritis. Glucosamine is an essential building block for the synthesis of glucosaminoglycans. Studies have shown that supplementing the body with additional amounts of glucosamine (1500 mg daily) promotes the production of chondrocytes, reduces pain, and increases joint function. In addition to glucosamine, chondroitin sulfate may inhibit several enzymes that degrade articular cartilage. Clinically, chondroitin supplements appear to reduce osteoarthritis symptoms. The American Academy of Orthopedic Surgeons position statement indicates that there is good evidence that glucosamine and chondroitin sulfate may help symptomatically with no side effects.
28. What is chronic compartment syndrome?

The lower leg is divided into four compartments that contain muscles plus neurovascular bundles. An increase in volume in the compartment may result from exercising muscles causing excessive pressure within the compartment (pre-exercise pressure, >15 mm Hg; 1-minute post exercise, >30 mm Hg; 5-minute post exercise, >20 mm Hg; normal values, 5–10 mm Hg). Symptoms of chronic compartment syndrome include compartment tightness, which occurs during or after exercise. Swelling may exist as well as paresthesia over the dorsum of the foot.

29. List treatment options for chronic compartment syndrome.

- Fasciotomy
- Stretching
- Training modification
- Strengthening
- Icing
- Biomechanical correction

30. Why might an athlete collapse on the field?

<table>
<thead>
<tr>
<th>Traumatic</th>
<th>Nontraumatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spinal cord injury</td>
<td>Cardiac (coronary artery disease, arrhythmia, congenital abnormality)</td>
</tr>
<tr>
<td>Thoracic injury (multiple rib fractures, hemothorax, tension pneumothorax, cardiac tamponade, cardiac contusion)</td>
<td>Hyperthermia (hypothermia, hyponatremia)</td>
</tr>
<tr>
<td>Abdominal injury (ruptured viscus)</td>
<td>Respiratory (asthma, spontaneous pneumothorax, pulmonary embolism)</td>
</tr>
<tr>
<td>Multiple fractures</td>
<td>Allergic anaphylaxis</td>
</tr>
<tr>
<td>Blood loss</td>
<td>Drug toxicity</td>
</tr>
<tr>
<td></td>
<td>Vasovagal response (faint)</td>
</tr>
<tr>
<td></td>
<td>Postural hypotension</td>
</tr>
<tr>
<td></td>
<td>Hyperventilation</td>
</tr>
<tr>
<td></td>
<td>Hysteria</td>
</tr>
</tbody>
</table>

31. What is exercise-induced asthma (EIA)?

EIA is characterized by a transient narrowing of the airway after intense exercise lasting longer than 10 minutes. This transient narrowing is associated with bronchospasms. EIA is more common in exercises such as long-distance running and cross-country skiing. A positive test for EIA is a >10% decrease of the forced expiratory volume in 1 second (FEV₁). Management of EIA usually involves the use of a β₂-agonist with a mast cell stabilizer before exercising.

**OBVIOUS SIGNS AND SYMPTOMS**

- Wheezing
- Difficulty breathing
- Chest tightness
- Coughing
- Problems with prolonged exercise

**SUBTLE SIGNS AND SYMPTOMS**

- Stomach pain/nausea
- Fatigue
- Inability to exercise in the cold
- Chest congestion
- Frequent colds
- Dry throat
- Headache

**SPORTS AT RISK**

- Tolerable—archery, baseball, downhill skiing, football, golf, gymnastics, karate, riflery, short-distance running, swimming, tennis, volleyball, and wrestling
- Less tolerable—basketball, cross-country skiing, cycling, ice hockey, ice skating, lacrosse, long-distance running, rowing, and soccer
MANAGEMENT

- Pharmacotherapy
  - First line—β-agonist
  - Second line—mast cell stabilizers
  - Third line—corticosteroids

PREVENTION

- Preactivity (0–60 min)—10 to 15 minute warm-up
- Short bursts of submaximal activity (5–10 min)
- Premedicate 15 to 30 minutes before practice/event
- Post competition—0 to 15 minutes of cool-down

32. What is a concussion?
There is still much confusion regarding the official definition of concussion. The American Academy of Neurology defines concussion as a clinical syndrome of biomechanically induced alteration of brain function, typically affecting memory and orientation, which may involve loss of consciousness. It is usually caused by rotational, angular, and/or lateral forces that cause rotation of the cerebral hemispheres around the upper brainstem. The Fourth International Conference on Concussion in Sport in 2012 defined concussion as a “complex pathophysiological process affecting the brain induced by biomechanical forces.”

33. How does a concussion occur?
Concussion may result from a direct blow to the head, face, neck, or torso, causing an “impulsive force” to be transmitted to the head and resulting in acceleration-deceleration of the brain within the skull. The neuronal injuries caused by the rotational and linear shear result in diffuse axonal injury and membrane dysfunction. This initiates further changes including neurotransmitter release, alteration in cerebral blood flow, mitochondrial dysfunction, and free-radical formation. Loss of consciousness does not occur with all concussions. Suspected diagnosis of concussion generally includes athletes reporting at least one of the following clinical components: somatic symptoms, physical signs, behavioral changes, cognitive impairment, or sleep disturbance. The majority of concussions will self-resolve within 3 weeks.

34. What are the signs and symptoms of a concussion?

SIGNS OBSERVED BY MEDICAL STAFF OR COACH
- Appears dazed or stunned
- Seizure, convulsive movement, tonic posturing, fencing response
- Vomiting
- Confused about assignment or position
- Forgets instruction
- Moves clumsily
- Loss of consciousness
- Shows mood, behavior, or personality changes
- Anxiety, uncontrollable crying, aggression, anger, restlessness
- Holding head
- Cannot recall events prior or after hit or fall

SYMPTOMS REPORTED BY ATHLETE
- Headaches or pressure in head
- Dizziness
- Nausea or vomiting
- Double or blurry vision
- Ringing ears
- Impaired balance
- Sensitivity to noise and/or light
- Feeling sluggish, hazy, foggy, or groggy
- Confusion
- Concentration or memory problems
- Loss of taste or smell
- Difficulty sleeping
- Fatigue and/or drowsiness
- Nervousness, anxious, sadness, irritability, more emotional, and/or mood swings
- Neck pain
WORRISOME SYMPTOMS—NEED ASSESSMENT IN EMERGENCY DEPARTMENT

- Severe or worsening headache
- Seizure or tonic posturing
- Focal neurologic signs
- Looks very drowsy or cannot be awakened
- Recurrent vomiting
- Increasing confusion or irritability
- Weakness or numbness in arms or legs
- Severe neck pain or loss of range-of-motion
- Significant irritability and/or agitation
- Loss of consciousness
- Unequal pupils

35. What is the incidence of concussion for males and females in the United States?
It is currently estimated between 1.6 and 3.8 million sports-related concussions occur each year in the United States. Overall female athletes are more likely to sustain a concussion than their male counterparts although the relationship may be sport dependent. Research indicates several possible theories for this increased risk of concussion. Women experience greater angular-rotation and head-neck peak acceleration and displacement upon impact. Female athletes tend to have both smaller and weaker neck muscles than males. Also, female soccer players are noted to have an increased ball-to-head size ratio. Last, it is possible that female athletes may be more likely to report injuries, although it should noted that research findings differ on this last point.

36. What is the concussion incidence in various sports?

<table>
<thead>
<tr>
<th>MENS SPORTS</th>
<th>ANNUAL NATIONAL ESTIMATE, N</th>
<th>WOMENS SPORTS</th>
<th>ANNUAL NATIONAL ESTIMATE, N</th>
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</thead>
<tbody>
<tr>
<td>Football</td>
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<td>Soccer</td>
<td>1113</td>
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<td>Basketball</td>
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<td>998</td>
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<td>Softball</td>
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<td>576</td>
<td>Volleyball</td>
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<td>332</td>
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<tr>
<td>Ice hockey</td>
<td>315</td>
<td>Field hockey</td>
<td>245</td>
</tr>
<tr>
<td>Baseball</td>
<td>223</td>
<td>Ice hockey</td>
<td>128</td>
</tr>
</tbody>
</table>

Source: National Estimates from the National Collegiate Athletic Association (NCAA) Injury Surveillance Program.

37. Do helmets and mouth guards decrease concussions?
Data are insufficient to support or refute the superiority of one type of football helmet in preventing concussions. There is no compelling evidence that mouth guards protect athletes from concussion.

38. What type of quick “on the field” testing can be done by a trained professional to determine the presence of concussion?
Sport Concussion Assessment Tool (SCAT3) is the most commonly used sideline assessment tool for concussion in children 13 years old and up. This test combines a symptom questionnaire (Graded Symptom Checklist [GCS]), cognitive assessment (Standardized Assessment of Concussion [SAC]), coordination examination, physical signs rating, and a balance test (Modified Balance Error Scoring System [m-BESS]). The SCAT is considered to be both reliable and valid. Furthermore this test can be used acutely or serially throughout the recovery period. In children aged 5 to 12 years old the Child-SCAT3 should be used, although it should be noted that this test has not yet been validated.

The King-Devick test is also being used for rapid sideline concussion assessment. This test combines vision, eye movement (saccades), language function, and attention. This is a <2-minute sideline assessment requiring the athlete to quickly read a series of numbers from three test cards. Increased time to complete the test worsens the athlete’s score, which indicates probable concussion.
39. If a concussion is suspected, what are the guidelines regarding immediate and long-term return to play?

Any athlete with suspected concussion should immediately be removed from play and evaluated by a physician or other licensed health care provider. First aid needs should be addressed and then sideline assessment for concussion should be performed. The player should not be returned to play the same day if concussion is suspected. Any player with worrisome symptoms such as seizure, loss of consciousness, recurrent vomiting, focal neurologic deficits, or increasing confusion or somnolence should be sent to the emergency department for evaluation. All athletes with suspected concussion should be seen for a follow-up by a health care provider familiar with concussive injury within 24 to 48 hours. The final determination regarding a diagnosis of concussion and/or fitness to play is a medical decision based on clinical judgment. In many states only a physician can decide if a player may return to competition after sustaining a concussion or a concussion is suspected.

40. What are the recommendations related to cognitive rest after a concussion?

Cognitive rest is commonly recommended for 24 to 72 hours after an athlete has been concussed although this recommendation is based largely on anecdotal evidence. During this time the athlete should not attempt to study or complete homework. The athlete should avoid screen time or time spent on computers/iPads/phones/laptops/SMART boards. They should avoid loud, bright, or busy venues during this period of rest. Upon return to school the student may need academic accommodations such as extended time for homework or class work. Tests and quizzes may need to be delayed or taken in sections with extended time.

The athlete should also be placed on symptom-limited physical rest until he or she can evaluated by a health care professional. Typically the athlete will remain on physical rest until concussion symptoms have significantly improved. At this point all athletes must complete a minimum 5-day graduated return-to-play protocol. It should be noted that players should be able to complete schoolwork without requiring academic accommodations before returning to full physical activity. In athletes who have developed more chronic symptoms, returning to low-impact, noncontact, low-risk activity can be beneficial.

Graduated Return-to-Play Protocol

<table>
<thead>
<tr>
<th>REHABILITATION STAGE</th>
<th>FUNCTIONAL EXERCISE AT EACH STEP</th>
<th>OBJECTIVE OF EACH STEP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1 Light aerobic activity</td>
<td>Walking, stationary bike, swimming, keeping intensity &lt;70% maximum heart rate; no resistance</td>
<td>Increase heart rate</td>
</tr>
<tr>
<td>Step 2 Sport-specific exercise</td>
<td>Sport-specific drills; no head impact or full-contact activities</td>
<td>Add movement</td>
</tr>
<tr>
<td>Step 3 Noncontact training drills</td>
<td>Progression to more complex training drills; may start progressive weight training</td>
<td>Exercise, coordination, and cognitive load</td>
</tr>
<tr>
<td>Step 4 Full-contact practice</td>
<td>Participate in normal training activities *Athlete must have medical clearance</td>
<td>Restore confidence and assess functional skills</td>
</tr>
<tr>
<td>Step 5 Return to play</td>
<td>*Athlete must have medical clearance</td>
<td></td>
</tr>
<tr>
<td>+ athlete should continue to progress to the next level if asymptomatic at current level</td>
<td>++ Each step should take 24 hours</td>
<td>+++ If any postconcussion symptoms reoccur the athlete should drop back to the previous asymptomatic level and try to progress again after a 24-hour period of rest.</td>
</tr>
</tbody>
</table>

41. What types of advanced assessments or neuropsychological testing can be administered to identify concussion?

According to the Zurich consensus statement, computerized neuropsychological testing is recommended although currently not mandatory. Formal referral for neuropsychological evaluation is more commonly used in athletes with ongoing cognitive difficulty (memory, concentration, attention) or with pronounced symptoms related to mood. Cognitive function is part of the neurologic assessment, and it is frequently used in conjunction with computerized neuropsychological screening tools. One of the most commonly used computerized tests is the ImPACT test. This test measures verbal and memory skills, processing skills, and reaction times in athletes 13 years of age and older. This test is never used in the place of clinical assessment.

The balance error scoring system (BESS), a clinical balance assessment for assessing postural stability, and the sensory organization test (SOT), which uses a force plate to measure a subject's ability to maintain equilibrium, can be used. The SOT test measures both stability and sway in differing sensory conditions that become increasingly difficult. The BESS and SOT are considered reliable, but they can be influenced by noise, anxiety, or orthopedic injury. Serial testing can result in learning effect.

42. Should neuroimaging be used to diagnose a concussion?

Magnetic resonance imaging (MRI) and computed tomography (CT) imaging are not typically used to diagnose concussion because neuroimaging is most often normal in concussive injury. They can be used to rule out worrisome etiology such as intracranial or intracerebral hemorrhage or skull fracture. Examples of circumstances that may indicate imaging would include prolonged confusion, loss-of-consciousness, focal neurologic deficit, worsening symptoms, posttraumatic seizure, posttraumatic amnesia, GCS <15, or a palpable skull fracture. CT of the head without contrast has historically been used in the emergency department, with MRI being more commonly used in athletes experiencing extended recovery.

43. What is the standard of care regarding return to play for an individual who has suffered multiple concussions?

At this time there are very few official guidelines or protocols in place for return-to-play decisions in athletes having experienced multiple concussions. The American Medical Society for Sports Medicine statement includes athletes who have multiple lifetime concussions, concurrent structural abnormalities, persistent diminished brain function, who experience prolonged recovery times, or who exhibit a decrease in injury threshold as those experiencing complex concussion. Treatment plans for athletes with complex concussion remain very individualized and should always be referred to a physician experienced in concussive injury. These athletes may be progressed more slowly through the return-to-play protocol, advised to sit the season out, or take a hiatus from all contact sports. In some circumstances a recommendation for permanent retirement from contact sports will be made.

44. Should medications be administered to individuals suffering from a concussion?

Evidence for the use of pharmacologic treatment in the acute period of concussion is somewhat limited. However, with athletes experiencing severe or prolonged symptoms, treatment during the subacute or chronic period is acceptable by a licensed health care provider familiar with concussive injury. Common medications used for symptom management include selective serotonin reuptake inhibitors (SSRIs), serotonin norepinephrine reuptake inhibitors (SNRIs), divalproex sodium/valproic acid, or other antianxiety or antidepressant for mood disorders. Melatonin is commonly used for sleep difficulty. These are frequently used in conjunction with nonpharmacologic methods of symptom management. For example, various psychotherapy or cognitive-behavioral therapies are available for cognitive or behavioral issues. Vestibular therapy can be useful for athletes with dizziness, balance issues, or difficulty with visual tracking. Similarly, speech-language therapy can be useful for patients experiencing ongoing issues with memory, concentration, or attention difficulty.

45. Is headache common after a concussion, and how is it treated?

Headache is the most common symptom after a concussion. Typically, aggressive treatment is not needed for acute headache. If symptoms are severe the athlete can be permitted to use over-the-counter remedies, such as acetaminophen, ibuprofen, and naproxen, as needed. If concern for hemorrhage exists, patients should use acetaminophen and avoid NSAIDs or naproxen sodium. For prolonged or severe headaches with migrainous or tension-type headache characteristics, a short trial of magnesium, triptans, tricyclic antidepressants, topiramate, or β-blockers may be beneficial. Patients with more chronic headache may also benefit from trigger-point injections, pericranial nerve blocks, or greater occipital nerve blocks. It is important to note that athletes should have stopped the use of all medications that could mask the symptoms of concussion before returning to competition.
CHAPTER 22 QUESTIONS

1. What is the role of medications in the acute management of a concussion?
   a. Analgesics are recommended immediately for 4 to 6 weeks.
   b. Gabapentin is recommended immediately for 6 to 8 weeks.
   c. Steroids are recommended immediately to reduce swelling and inflammation in the brain.
   d. Medication does not speed recovery of a concussion.

2. Female athletes tend to sustain anterior cruciate ligament tears at a higher rate than males. Which of the following pairing of factors is the most probable cause for this gender difference?
   a. Lack of protein in diet, monthly hormonal variations
   b. Landing with excessive valgus at the knee, reliance on ligament for knee stability
   c. Smaller and weaker ligament, lack of motivation
   d. Hamstring dominance pattern with activity, A-shaped intercondylar notch

3. What is the current “best practice” following a suspected concussion?
   a. Rest until acute symptoms resolve, then graded exertion
   b. Return to play if symptoms clear within 30 minutes
   c. Hospitalization for observation up to 3 days
   d. Termination of season and restet before the start of next season
1. What is a diagnosis, and what is a differential diagnosis?

A diagnosis is a named category of specific clinical data that labels a condition and provides characteristics of the condition when communicated to health care professionals. A differential diagnosis is a list of possible diagnoses generated from the patient interview and physical examination, listed in order of likelihood from the most likely to the least likely. In general terms, in the context of physical therapy (APTA Guide to PT Practice, 2001) the diagnosis is used to identify “the impact of a condition on function at the level of the system and at the level of the whole person.”

2. What are examples of impairments that a physical therapist might diagnose?

- Impaired posture
- Impaired gait/locomotion
- Impaired range of motion
- Impaired muscle performance
- Impaired motor function
- Impaired joint mobility
- Impaired neural function

3. What are characteristics of visceral symptoms?

- Location—unilateral or bilateral; poorly localized in terms of specific organ or system (eg, angina)
- Quality—knifelike, boring, deep bone pain, deep aching, cutting, moderate to severe, and/or perceived from the inside out
- Character—symptoms often unrelieved by rest, changes in position, and interventions that would typically affect musculoskeletal disorders. Associated symptoms that do not occur with musculoskeletal disorders can be identified via a careful review of systems.
- Quantity or severity—typically related to exacerbating factors and varies based on organ/organ system and status of disease processes (eg, dull to sharp or mild to severe)
- Onset—recent or sudden but does not typically present as being chronically observed (ie, insidious onset often without an attributable mechanism)
- Duration and frequency—constant or intermittent based on organ/system and attributing factors, gradually progressive, cyclical, or symptom may come in waves
- Aggravating factors—differ based on involved organ/system and status of disease processes (eg, fatty foods will typically aggravate a gallbladder disorder)
- Relieving factors—differ based on involved organ/system and status of disease processes. A specific strategy such as rest may initially relieve symptoms (ie, pain), but there is typically a recurring progression of increasing frequency, intensity, and/or duration of symptoms.
- Client’s perception of the symptom—should be expected to vary among patients and will be influenced by cognitive, affective, cultural, socioeconomic, and environmental factors. For example, patients may self-diagnose, select unwise self-treatments, or perceive certain symptoms, such as coughing, sweating, or diarrhea as normal and not symptoms of illness.

4. What are somatic disorders?

Somatic disorders are musculoskeletal syndromes in which symptoms are caused by nociceptive stimulation of pain-sensitive structures. The origin of somatic pain is mechanical and/or chemical stimulation of nerve endings. Somatic pain may be either localized to a body region and/or referred to other body regions. Somatic pain and somatic referred pain are typically static, aching in quality, and difficult to point-localize.
5. What are characteristics of somatic symptoms?

- **Location**—typically unilateral and described as presenting in one joint or in one body region; somatic referred pain may or may not be present
- **Quality**—achy, deep, sharp, pulling, sore, stiff, and/or cramping pain
- **Character**—local tenderness or pain that is attributable to an activity or underlying pathology (eg, pain exacerbated by overhead activities with secondary impingement tendinosis; morning stiffness with osteoarthritis)
- **Quantity or severity**—mild to severe
- **Onset**—sudden or gradual: sudden associated with acute overload stresses and macrotrauma, and gradual associated with chronic overloading stresses and repetitive microtrauma
- **Duration and frequency**—intermittent to constant: usually intermittent with varying intensity based on activity and/or position with mechanical disorders; constant with acute inflammatory disorders. Symptoms may present as chronologically observed, characterized by asymptomatic periods with exacerbations or progressively exacerbated symptoms attributed to causal factors or progression of the underlying disorder.
- **Aggravating factors**—symptoms are typically exacerbated with specific movement, activities, loading, etc., and the degree of exacerbation is a function of attributing factors or progression of the underlying disorder.
- **Relieving factors**—relieving factors are typically a function of identifying and managing aggravating factors (eg, activity modification, rest, pacing, therapeutic interventions, improved self-management, eliminating attributing factors, positioning, and relative rest).

6. What are radicular disorders?

A radicular disorder is a neurogenic disorder in which signs and/or symptoms are caused by damage or irritation of the spinal nerves or spinal nerve roots. The origin of signs and symptoms is mechanical and/or chemical and is attributable to a block in conduction rather than stimulation of nerve endings. Radicular disorders produce lower motor neuron lesion signs and symptoms, which include muscle weakness, atrophy, hyporeflexia, and sensory changes such as paresthesia and/or numbness. A block in conduction itself does not necessarily cause pain in either the spine or the corresponding extremity, but radicular disorders typically occur concurrently with somatic pain disorders.

7. What is a key characteristic of radicular symptoms?

Radicular pain is described as shooting or lacerating and is typically felt in a relatively narrow band about 4 cm wide; it is often combined with other radicular symptoms such as tingling, numbness, and burning sensations.

8. What is the difference between radicular referred symptoms and somatic referred pain accompanying a radicular disorder?

Radicular symptoms result from a block in conduction rather than nociceptive stimulation of pain-sensitive structures (ie, the spinal nerve or nerve root). Radicular symptoms are typically referred to the distribution supplied by the involved spinal nerve or nerve root, but this assumption must take into consideration the following:

1. The distribution of radicular symptoms is not always distinctive.
2. Radicular pain from a given nerve root does not always follow a consistent distribution.
3. All radicular disorders do not result in referred pain.
4. Radicular symptoms do not always extend to the distal portion of the involved dermatome.

Somatic referred pain is generated by either mechanical or chemical irritation of somatic structures, such as the dural lining on the nerve root or the epineurium of the spinal nerve. Like radicular referred pain, somatic referred pain is felt in body regions separate from the irritated structures (eg, lumbar facet arthrosis can refer pain to the leg).

**SCREENING FOR SYSTEMIC INVOLVEMENT**

9. Why do physical therapists need to screen for systemic involvement?

Physical therapists need to screen for systemic or non–physical therapy involvement because many visceral (organ or organ system) diseases mimic orthopedic symptoms. For example, Jarvik and Deyo (2002) reported that among patients with low back pain being seen in ambulatory primary-care
clinics, 4% will have osteoporosis-related fractures, 2% will have spondylolisthesis (forward displacement of a vertebral body) or spondylolysis (fracture of a portion of the vertebra, which may lead to spondylolisthesis), 2% will have visceral disease, 0.7% will have cancer, and 0.5% will have infections. Given the possibility of such disorders, the clinician must promptly screen patients at risk for such medical conditions and make the appropriate referrals.

10. List common body systems and aggregates of signs/symptoms that may indicate systemic involvement.

<table>
<thead>
<tr>
<th>General</th>
<th>Endocrine/ Metabolic</th>
<th>Genito- Reproductive</th>
<th>Peripheral Vascular</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appetite</td>
<td>Hot/cold intolerance</td>
<td>Contraceptive measures</td>
<td>Claudication</td>
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<tr>
<td>Weakness</td>
<td>Goiter</td>
<td>Pain</td>
<td>Raynaud’s</td>
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<tr>
<td>Fatigue</td>
<td>Irradiation exposure</td>
<td>Mass</td>
<td>Ulcers</td>
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<tr>
<td>Weight loss</td>
<td>Lipid disorder</td>
<td>Lesions</td>
<td>Thrombophlebitis</td>
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<td>Fever</td>
<td>Diabetes</td>
<td>Discharge</td>
<td>Varicosities</td>
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<td>Chills</td>
<td>Change in physical features</td>
<td>Pruritic</td>
<td>Psychiatric</td>
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<td>ENT</td>
<td>VD</td>
<td>Anxiety</td>
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<td>Infections</td>
<td>Sexual dysfunction</td>
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<td>Acuity</td>
<td>Leukemia</td>
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<td>Hay fever</td>
<td>Glasses/contacts</td>
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<td>Asthma</td>
<td>Visual fields</td>
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<td>Diplopia</td>
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<td>Breasts</td>
<td>Scotoma</td>
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</tbody>
</table>
11. What are examples of common “red flags” that typically require physician referral and further investigation?

- Anorexia
- Back and abdominal pain at the same level
- Bilateral symptoms
- Changes in mental status
- Chills
- Constipation
- Diaphoresis (excessive perspiration)
- Diarrhea
- Dyspnea (breathlessness at rest or after mild exertion)
- Early satiety (feeling full after eating)
- Elevated body temperature
- Fecal or urinary incontinence (inability to control bowels or urine)
- Frequency (increased urination)
- Headaches, dizziness, fainting, or falling
- Hematuria (blood in the urine)
- Insidious onset with progression of symptoms
- Melena (blood in feces)
- Nausea
- Night sweats
- Nocturia
- Obvious change in a wart or mole
- Pain at night
- Pain that forces a patient to curl up into fetal position
- Pain unrelieved by recumbency
- Painless weakness of muscles: more often proximal but may occur distally
- Poor or delayed healing
- Sacral pain without history of injury
- Skin lesions
- Thickening of a lump
- Unexplained weight loss
- Unusual bleeding, bruising, or discharge
- Unusual vital signs
- Urgency (sudden need to urinate)
- Visual disturbances
- Vomiting
- Weakness and/or fatigue
- Weight loss/gain without explanation

**CARDIOVASCULAR**

12. True or false: Pain referral patterns associated with myocardial infarction (MI) are the same for men and women.

False. Symptoms of MI do not always follow the classic pattern, especially in women. Women may experience pain referred into the right shoulder in addition to shortness of breath (sometimes occurring in the middle of the night) and chronic, unexpected fatigue.

13. What are silent heart attacks, and who do they commonly affect?

Silent attacks (painless infarction without acute symptoms) are more common among nonwhites, older adults (>75 years), smokers, and adults with diabetes (men and women), presumably because of reduced sensitivity to pain.

14. For myocardial infarctions associated with a blood clot, what time frame for the administration of medications that dissolve clots, promote vasodilation, and reduce infarct size is considered the most crucial?

Administration of medication within the first 70 minutes after the onset of symptoms is associated with improved outcomes.
15. What are typical pain referral patterns for the heart?

- Angina pectoris.
- Myocardial infarction.
Pericarditis.

Dissecting aortic aneurysm.
16. What signs and symptoms are commonly associated with cardiac pathology?
- There is a sudden sensation of pressure in the chest that occasionally radiates into the arms, throat, neck, and back.
- Pain is constant, lasting 30 minutes to hours.
- Pain may be accompanied by shortness of breath, pallor, and profuse perspiration.
- Angina pectoralis has similar symptoms to an MI. However, angina pectoralis is less severe, does not last for hours (rarely more than 5 minutes), and is relieved by cessation of all activity and administration of nitrates.
- Symptoms of MI do not always follow the classic pattern, especially in women.
- Two major symptoms in women are shortness of breath (sometimes occurring in the middle of the night) and chronic, unexpected fatigue.
- A typical presentation may include continuous pain in the midthoracic spine or interscapular area, neck and shoulder pain, stomach or abdominal pain, nausea, unexplained anxiety, or heartburn that is not altered by antacids.
- Silent attacks (painless infarction without acute symptoms) are more common among nonwhites, older adults (>75 years), smokers, and adults with diabetes (men and women), presumably because of reduced sensitivity to pain.
- Nausea and vomiting may occur because of reflex stimulation of vomiting centers by pain fibers.
- Fever may develop in the first 24 hours and persist for 1 week because of inflammatory activity within the myocardium.
- Myocarditis and endocarditis do not produce chest pain but rather chest tightness with breathlessness.

17. What are cardiac red flags?
Pain in the chest lasting longer than 30 minutes, shortness of breath with exertion or when sleeping, increased fatigue, nausea, vomiting, nonproductive cough, nocturia, changes in skin color (blue or ashen), and onset of pain in the early morning hours are all cardiac red flags.

18. What subjective questions should be asked when cardiac dysfunction is suspected?
- Presence of any red flags as previously described
- Questions about pain, regarding the onset, location, and character of the pain
- Additional information regarding dietary habits, cigarette or alcohol use, and exercise habits
- Questions regarding the use of prescription, over-the-counter, or street drugs; especially antihypertensive medications, β-blockers, calcium channel blockers, digoxin, diuretics, and aspirin/anticoagulants

Cervical radiculopathy (C8), ulnar nerve injuries, rotator cuff disorders, upper thoracic dysfunction, pectoralis major strain, subacromial bursitis, acromioclavicular arthritis, and temporomandibular (TM) joint pain mimic cardiovascular pain patterns.

**PULMONARY**

20. Describe the clinical signs and symptoms of acute pleuritis.
Sharp, stabbing substernal pain, especially with exertion, pleural rub on auscultation, and referred upper trapezius and interscapular pain are symptoms of acute pleuritis.

21. How does pulmonary function change with obstructive and restrictive pulmonary disorders?
- Restrictive—normal expiratory airflow, decreased vital capacity, decreased total lung capacity, decreased residual volume, and decreased PaCO₂
- Obstructive—reduced airflow with/without changes in vital capacity, increased total lung capacity, increased residual volume, and increased PaCO₂

22. What are typical pain referral patterns for the lungs?
Primary pain is typically noted over the midchest or involved lung and is often greater anterior as opposed to posterior. Referred pain may be noted in the neck, upper trapezius muscles, proximal shoulders, T1/C8 dermatome, along the ribs, and in the upper abdomen.
Pain patterns associated with pleuritis.

Pain patterns associated with pneumothorax.
23. What signs and symptoms are commonly associated with pulmonary pathology?

- Cough—continuous coughing (possibly indicating an acute or chronic pathology, eg, respiratory tract infection, allergies, bronchitis, emphysema, COPD, lung cancer), time of day cough (eg, environmental exposure to an irritant), night cough (eg, sinusitis or allergies), and early morning cough (eg, bronchial inflammation secondary to smoking)
- Sputum, including color and odor—clear to white sputum (eg, cold [viral infection] and bronchitis), purulent yellow or green sputum (eg, bacterial infections), reddish-brown sputum (eg, tuberculosis and pneumonia), and pink-foamy sputum (eg, pulmonary edema)
- Hemoptysis or blood derived from the lungs or bronchial tubes may result from a large number of conditions (eg, pneumonia, infections, cancer, trauma)
- Shortness of breath without physical exertion or with minimal physical exertion (eg, bronchitis, emphysema, pneumonia, pulmonary embolism, pleurisy, pneumothorax)
- Cyanosis (eg, respiratory acidosis, chronic bronchitis, pneumonia, cystic fibrosis)
- Chest pain that occurs with breathing (eg, pneumonia, pleurisy, lung cancer)
- Changes in respiratory rate or breathing patterns (eg, acute and chronic bronchitis, respiratory acidosis, emphysema)
- Change in normal breath sounds (eg, asthma, bronchitis, pneumonia, emphysema, pleurisy, lung cancer, and bronchiectasis)
- Chest cavity deformities or compensatory breathing patterns (eg, a barrel chest deformity and use of accessory muscle of respiration are indicative of emphysema)
- General undiagnosed symptoms of dizziness, fainting, fever, shortness of breath without exertion, cyanosis, night sweats, tachycardia, especially with a positive pulmonary history

24. What are pulmonary red flags?

- Central nervous system symptoms
- Change in normal breath sounds, especially wheezing
- Hemoptysis, especially with a long-term history of smoking
- Pain increased by recumbency or during sleep, especially if disturbing sleep
- Persistent undiagnosed cough
- Recurrent pulmonary infections
- Sharp pain with breathing, especially on inhalation
- Signs or symptoms of DVT
- Signs or symptoms of insufficient oxygenation or increased carbon dioxide levels
- Splinting used to reduce pain
- Sudden, sharp chest pain with or without trauma combined with changes in respiratory rate, diminished but rapid pulse rate, diminished blood pressure, and changes in respiratory rate
- Undiagnosed neck, shoulder, chest, and arm pain
- Unexplained hoarseness of voice and/or difficulty swallowing
- Unexplained upper extremity weakness
- Unexplained weight loss or gain, especially sudden
- Undiagnosed symptoms of dizziness, fainting, fever, shortness of breath without exertion, cyanosis, night sweats, tachycardia, especially when occurring in a cluster and in combination with specific pulmonary signs and symptoms

25. What subjective questions should be asked when pulmonary dysfunction is suspected?

- Presence of red flags as previously described
- Age (ie, >35-year-old female/ >40-year-old male)
- History of respiratory tract infections, cough, sputum, hemoptysis, dyspnea, infection, fever, chills
- History of smoking
- History of exposure to environmental contaminants
- Personal and family history of cancer
- History of pain exacerbated by inhalation or exhalation (eg, breathing, coughing)
- History of pain that is provoked or alleviated by lying on one side (eg, sleeping)
- History of general self-care and medical management (eg, last TB test, last chest x-ray, immunizations)


Musculoskeletal disorders mimicking pulmonary pain patterns include cervical radiculopathy (C8, T1), cervical and upper thoracic dysfunction (eg, arthrosis and spondylosis), rotator cuff disorders, and acromioclavicular arthritis regional muscle dysfunction (eg, pectoralis major strain, intercostal muscle strain, trigger points).
INTEGUMENTARY

27. What signs and symptoms are commonly associated with integumentary system pathology?

- Changes in a pigmented mole or benign tumor may indicate a possible malignancy.
- Cyanosis (dark bluish or purplish discoloration of the integument and mucous membranes) may indicate hypoxia or hematologic pathology.
- Edema, if generalized, may indicate cardiovascular, pulmonary, or renal dysfunction; localized edema may indicate infection, inflammation, or sudden change in pressure (ie, compartment syndrome). If edema is unilateral, consider a local or peripheral cause; if bilateral, consider a central disorder (eg, congestive heart failure and renal dysfunction).
- Hyperthermia may indicate localized or systemic infection, inflammation, thermal injury; hyperthyroidism or fever is generalized.
- Hypothermia may indicate arterial insufficiency or shock.
- Jaundice (yellowish discoloration of skin and sclera) may indicate liver disease or hemolytic pathology.
- Paleness of the skin may indicate arterial insufficiency, anemia, or shock.
- Redness of the skin may indicate fever, local infection, local inflammation, carbon monoxide poisoning, or polycythemia.
- Unexplained skin lesions may indicate infection, allergic reaction, parasitic infection, thermal injury, herpes, fungal infection, cancer, or neoplasm.

28. List common nail abnormalities and probable causes.

<table>
<thead>
<tr>
<th>Possible Nail Characteristics</th>
<th>Suspected Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brown band around nail plate</td>
<td>Addison’s disease</td>
</tr>
<tr>
<td>Red nail bed</td>
<td>Carbon monoxide poisoning</td>
</tr>
<tr>
<td>Red lunula</td>
<td>Cardiac failure</td>
</tr>
<tr>
<td>Brown discoloration of distal 1/3 of nail plate</td>
<td>Chronic renal insufficiency</td>
</tr>
<tr>
<td>Clubbing of nails</td>
<td>Congenital chronic cyanotic heart disease, emphysema, cystic fibrosis, and chronic bronchitis</td>
</tr>
<tr>
<td>Blue nail bed</td>
<td>Cyanosis or hemorrhage</td>
</tr>
<tr>
<td>Absence or underdevelopment of nail beds</td>
<td>Hereditary onychooosteodysplasia</td>
</tr>
<tr>
<td>Yellow nail bed</td>
<td>Jaundice</td>
</tr>
<tr>
<td>White lines and/or white spots</td>
<td>Leukonychia</td>
</tr>
<tr>
<td>Cuticle darkening and/or dark streaks</td>
<td>Melanoma</td>
</tr>
<tr>
<td>Brown nail plate</td>
<td>Onychomycosis</td>
</tr>
<tr>
<td>Yellow nail plate and bed, and/or nail dystrophy</td>
<td>Psoriasis</td>
</tr>
<tr>
<td>White nail plate</td>
<td>Superficial onychomycosis</td>
</tr>
<tr>
<td>Yellow nail plate</td>
<td>Tetracycline</td>
</tr>
<tr>
<td>Brittle nails and/or splitting of nail beds</td>
<td>Thyroid disease</td>
</tr>
</tbody>
</table>

29. What are integumentary system red flags?

- Sudden enlargement of an existing mole or benign tumor
- New areas of involvement or spreading of an existing mole or benign tumor
- Sudden change in color of an existing mole or benign tumor
- Formation of an irregular border or butterfly appearance to a new or previously existing mole or benign tumor
- A previously flat mole becomes elevated or raised, especially with irregular borders or notching
- Irregular or clumping of colors across a new or existing mole or benign tumor (ie, nonuniform browns and blacks mixed with reds, blues, and/or whites)
- Unexpected, especially sudden, changes, such as scaling, flaking, drainage, itching, redness, swelling, warmth, point tenderness, or bleeding

30. What subjective questions should be asked when integumentary system pathology is suspected?

- Presence of any red flags as previously described
- History of drug or topical agent use and self-care
- History of allergies
- History of circulatory or vasospastic disorders
- History of endocrine disorders (eg, thyroid disease or diabetes)
- History of applicable environmental factors (eg, exposure to radiation or x-rays, living conditions, dietary habits, occupations, leisure activities, travel, emotional stress; especially changes that occurred before or during identification of possible integumentary involvement)
- History of applicable genetic factors (eg, family history, gender, age, race)
- History of gynecologic factors (eg, pregnancy, menstruation, birth control pills)

31. True or false: Malignant melanomas arise from melanocytes in moles.
False. About 40% to 50% of malignant melanomas arise from melanocytes in moles; the remainder arise from melanocytes in normal skin.

32. What is the integumentary presentation of herpes zoster (shingles)?
Symptoms of shingles include vesicular eruptions and neuralgic pain in the cutaneous distributions supplied by peripheral nerves.

33. Describe the signs and symptoms of dysvascular and neuropathic foot ulcer.

<table>
<thead>
<tr>
<th>Dysvascular Foot Ulcer</th>
<th>Neuropathic Foot Ulcer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lesions are painful</td>
<td>Lesions are painless</td>
</tr>
<tr>
<td>Irregularly shaped</td>
<td>Circular in shape</td>
</tr>
<tr>
<td>Multifocal</td>
<td>Develop over bony plantar regions</td>
</tr>
<tr>
<td>Located on toes</td>
<td>Can be associated with callous formation</td>
</tr>
<tr>
<td>Located over nonplantar areas</td>
<td>Tend to be clean and nonnecrotic</td>
</tr>
<tr>
<td>Lesions are typically necrotic</td>
<td>Ulcer regions are warm and pink</td>
</tr>
<tr>
<td>Ulcer regions are typically cool and pale</td>
<td></td>
</tr>
</tbody>
</table>

34. What are the key characteristics of cellulitis?
Key characteristics include the following: poorly defined and widespread distribution that is red, edematous in appearance, and warm to hot with palpation; often accompanies infections.

GASTROINTESTINAL

35. What is the most common intraabdominal disease referring pain to the musculoskeletal system?
It is ulceration or infection of the mucosal lining of the GI tract.

36. How quickly do drug-induced symptoms occur in the GI tract?
Although some medications (eg, NSAIDs, digitalis, and antibiotics) may result in immediate symptoms in patients, it is not uncommon for symptoms to occur as long as 6 to 8 weeks after exposure.

37. What are typical pain patterns for GI pathologies?
- Pain of GI origin can mimic primary musculoskeletal lesions.
- Referral locations can include the following: shoulder, neck, sternum, scapular regions, mid back, low back, hip, pelvis, and sacrum.

38. What signs and symptoms are commonly associated with esophageal pathologies?
Diseases affecting the esophagus can cause the following symptoms: 1) dysphagia (sensation of food catching in the throat), 2) odynophagia (pain with swallowing), and 3) a burning sensation beginning at the xiphoid and radiating to the neck and throat (heartburn).
Causes of dysphagia include stricture, inflammation, neurologic conditions (such as stroke, Alzheimer’s disease, and Parkinson’s disease), drug side effects, and space-occupying lesions. Causes of odynophagia include inflammation, spasm, and viral or fungal infection. Esophageal pain is reported as sharp, knifelike, stabbing, strong, and burning.

39. What signs and symptoms are commonly associated with stomach and duodenal pathologies?
Stomach and duodenal pathologies (peptic ulcers, stomach carcinoma, and Kaposi’s sarcoma) may be associated with early satiety, melena (dark, tarry stools), and symptoms associated with eating. Pain is typically described as aching, burning, gnawing, and cramp-like. It ranges from mild to severe in intensity and typically comes in waves.
40. What signs and symptoms are commonly associated with small intestine pathologies?
Small intestine pain is described as cramping pain (moderate to severe in intensity), is intermittent in duration, and may be associated with nausea, fever, and diarrhea. Pain relief may not occur after defecation or passing gas.

41. What signs and symptoms are commonly associated with large intestine and colon pathologies?
Large intestine and colon pain is described as a cramping pain, dull in intensity, and steady in duration; it may be associated with bloody diarrhea, increased urgency, or constipation. Pain relief may occur after defecation or passing gas.

42. What signs and symptoms are commonly associated with pancreatic pathologies?
Pancreatic pain is described as a severe, constant pain of sudden onset that is burning or gnawing in quality. Associated signs and symptoms include sudden weight loss, jaundice, nausea and vomiting, light-colored stools, weakness, fever, constipation, flatulence, and tachycardia; it may or may not be related to digestive activities.

43. What subjective questions should be asked when GI pathology is suspected?
- Presence of any red flags as previously described
- History of drug or topical agent use, including self-care
- History of previous gastric or peptic ulcer

44. What are GI red flags?
- Difficulty swallowing
- Pain when swallowing
- Pain associated with eating (immediately or 2 to 3 hours post ingestion)
- Changes in frequency and ease of defecation
- Changes in coloration of stools
- Decreased appetite
- Sudden weight loss
- Vomiting
- Gnawing, burning pain
- Migratory arthralgias
- Decreased immune response

45. List common musculoskeletal disorders that mimic GI disorders.
Sports hernia, adductor strain/tear, lumbar disc disease, lumbar facet arthrosis, and symptomatic thoracic movement impairment are all common musculoskeletal disorders mimicking GI disorders.

46. What is the McBurney point, and what is its significance?
It is a point midway between the umbilicus and the right anterior-superior iliac spine used as a guide to locate the position of the appendix. The McBurney point is the most common site of maximum tenderness in acute appendicitis, which is typically determined by the pressure of one finger.

47. List the structures contained in each of the four abdominal quadrants.

<table>
<thead>
<tr>
<th>Right Upper Quadrant</th>
<th>Left Upper Quadrant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ascending colon (superior portion)</td>
<td>Descending colon (superior portion)</td>
</tr>
<tr>
<td>Duodenum</td>
<td>Jejunum and proximal ileum</td>
</tr>
<tr>
<td>Gallbladder</td>
<td>Left colic (hepatic) flexure</td>
</tr>
<tr>
<td>Liver (right lobe)</td>
<td>Left kidney</td>
</tr>
<tr>
<td>Pancreas (head)</td>
<td>Left suprarenal gland</td>
</tr>
<tr>
<td>Right colic (hepatic) flexure</td>
<td>Liver (left lobe)</td>
</tr>
<tr>
<td>Right kidney</td>
<td>Pancreas (body and tail portions)</td>
</tr>
<tr>
<td>Right suprarenal gland</td>
<td>Spleen</td>
</tr>
<tr>
<td>Stomach (pylorus)</td>
<td>Stomach</td>
</tr>
<tr>
<td>Transverse colon (right half)</td>
<td>Transverse colon (left half)</td>
</tr>
</tbody>
</table>
48. List the common signs and symptoms associated with chronic renal failure.
Uremia, dizziness, headaches, heart failure, hypertension, ischemic lower extremity pain, muscle cramps, edema, peripheral neuropathy, weakness, decreased endurance, decreased heart rate, and decreased blood pressure and hypotension, among others

49. What is the costovertebral angle, and what is its significance?
The costovertebral angle is the angle formed on either side of the vertebral column between the last rib and the lumbar vertebrae. Tenderness in this region is indicative of renal disease, and it is a potential site for unintended encroachment on the pleural cavity during surgery.

50. What are the two most common urinary tract infections?

   - Cystitis—Inflammation and infection of the bladder
   - Pyelonephritis—Inflammation and infection of one or both kidneys

51. What is a key feature that typically distinguishes a radicular disorder from renal pain?
Renal pain is rarely influenced by changes in spinal posture or movements of the spine.

52. List common clinically observable signs and symptoms of chronic renal disease.
Hyperpigmentation, bruising, itching, paleness/anemia, redness of the eyes, shortness of breath, uremic breath, tremors, footdrop, weakness/altered movement patterns, decreased ability to concentrate, lethargy, irritability, and impaired judgment

53. What are typical pain patterns for renal pathologies?

   - Bladder and urethra—sharp and localized upper pelvic, lower abdominal, and back pain; painful spasms of the anal sphincter; involuntary straining and an urgent need to empty the bowel with minimal passage of urine or fecal matter; urinary urgency and burning pain with urination
   - Ureter—severe unilateral or bilateral costovertebral angle pain, painful spasms of the anal sphincter, involuntary straining and an urgent need to empty the bowel with minimal passage of urine or fecal matter, malaise, vomiting, nausea, abdominal distention, kidney/ureter tenderness, abnormal tenderness, and pain in a T10 to L1 distribution; a lesion outside of the ureter may be provoked with an active contraction of the iliopsoas muscle
   - Kidney—pain in the posterior subcostal region and in the area of the lower costovertebral articulations, posterior to lateral referred pain into the abdominal region and groin (usually unilateral), malaise, fever, chills, frequent urination, possible blood in urine, nausea and vomiting, abdominal spasms, abnormal tenderness and pain in a T9 to T10 distribution

54. What signs and symptoms are commonly associated with renal pathologies?

   - Bladder and urethra—sharp and localized upper pelvic, lower abdominal, and back pain; painful spasms of the anal sphincter; involuntary straining and an urgent need to empty the bowel with minimal passage of urine or fecal matter; urinary urgency and burning pain with urination
   - Ureter—severe unilateral or bilateral costovertebral angle pain, painful spasms of the anal sphincter, involuntary straining and an urgent need to empty the bowel with minimal passage of urine or fecal matter, malaise, vomiting, nausea, abdominal distention, kidney/ureter tenderness, abnormal tenderness, and pain in a T10 to L1 distribution; a lesion outside of the ureter may be provoked with an active contraction of the iliopsoas muscle
   - Kidney—pain in the posterior subcostal region and in the area of the lower costovertebral articulations, posterior to lateral referred pain into the abdominal region and groin (usually unilateral), malaise, fever, chills, frequent urination, possible blood in urine, nausea and vomiting, abdominal spasms, abnormal tenderness and pain in a T9 to T10 distribution

55. What are renal red flags?

   - Abdominal muscle spasms
   - Abdominal splinting
Abnormal tenderness and pain in a T9 to L1 distribution
Blood in urine (eg, brown or red) or clouding of urine
Changes in sexual function or pain during intercourse
Changes in urinary patterns and/or urine flow
Costovertebral angle pain
Decreased or absent urination
Dependent edema (moderate to significant)
Fever and chills
Genital discharge
Genital lesions
Headaches
Low back and abdominal pain at the same level
Malaise
Masses, lesions, or swelling
Nausea and vomiting
Pain with urination
Proximal lateral thigh and/or lower lateral trunk pain
Shortness of breath
Shoulder pain (usually with ipsilateral kidney problems)
Tenesmus

56. What subjective questions should be asked when renal pathology is suspected?
- Presence of red flags as previously described
- Past medical and surgical history (eg, kidney stones, bladder stones, infections, abdominal injuries, hernias, history of cancer, abdominal surgery, all applicable interventions and outcomes)
- History of abdominal pain (eg, primary and referred pain, influence of movement and position on pain and referred pain)
- History of proximal lateral thigh and/or lower lateral trunk pain (suspect kidney or ureter)
- History of upper pelvic and lower abdominal pain (suspect bladder and/or urethra)
- History of changes in bowel/bladder function (eg, increased frequency of urination, suspect infection; decreased flow or trouble initiating flow, suspect urethral obstruction; decreased diameter of flow, suspect urethral obstruction; feeling of bladder fullness after urination, suspect bladder disorder or enlarged prostate; burning pain during or after urination, suspect sexually transmitted disease or lower urinary tract infection; loss of control, suspect incontinence)
- History of nutritional/dietary changes
- History of relevant associated symptoms (eg, fatigue, nausea, vomiting, vaginal or penile discharge, changes in menstrual cycle and sexual habits as applicable)

57. List common musculoskeletal disorders that mimic renal disorders.
Common musculoskeletal disorders that mimic renal disorders include lower thoracic or lumbar plexus radiculopathy, lumbar and lower thoracic dysfunction (eg, arthrosis, spondylitis, and costal/costovertebral), regional muscle dysfunction (eg, adductor strain), central nervous system disease, meralgia paresthesia, and trauma.

HEPATIC AND BILIARY
58. What musculoskeletal signs or symptoms may be associated with hepatic and biliary dysfunction?
Bilateral carpal tunnel syndrome accompanied by bilateral tarsal tunnel syndrome is a musculoskeletal sign associated with hepatic and biliary dysfunction.

59. What are typical pain patterns of the hepatic and biliary systems?
Pain associated with the liver, gallbladder, and the common bile duct is typically located in the midepigastric or right upper quadrant of the abdomen. Musculoskeletal pain referred from the hepatic and biliary systems may be located in the right shoulder, upper trapezius, or right scapular area or between the scapulae.
Liver, gallbladder, and common bile duct pain (upper right quadrant) and referred pain patterns (shoulder and scapular regions).

60. What signs and symptoms are commonly associated with hepatic and biliary system pathologies?

In addition to the musculoskeletal pain referral patterns listed previously, patients experiencing hepatic or biliary dysfunction may also demonstrate changes in skin color, as well as neurologic symptoms. Skin changes include yellowing of the skin or sclera of the eyes (jaundice), pallor, and orange or green skin. Neurologic signs and symptoms include confusion, sleep disturbances, muscle tremors, hyperactive reflexes, and asterixis (flapping tremor where the patient is unable to maintain wrist extension with forward flexion of the arms).

61. What are hepatic and biliary system red flags?

- Anorexia, nausea, and vomiting
- Arthralgias
- Dark urine and light-colored or clay-colored feces
- Edema and oliguria (reduced urine secretion in relation to fluid intake)
- Excessive belching
- Extreme fatigue
- Gynecomastia
- Neurologic symptoms (confusion, sleep disturbances, muscle tremors, hyperactive reflexes, asterixis, bilateral carpal/tarsal tunnel syndrome)
- Painful abdominal bloating
- Pallor, yellowing of the eyes or skin
- Right upper quadrant abdominal pain
- Sense of fullness in the abdomen
- Skin changes (jaundice, bruising, spider angioma, palmar erythema)
62. What subjective questions should be asked when hepatic and biliary system pathology is suspected?
- Presence of any red flags as previously described
- Recent changes in bowel and bladder habits
- Exposure to needles (including injection, drug use, acupuncture, tattooing, ear or body piercing, recent operative procedure, hemodialysis), exposure to certain chemicals or medications, severe alcoholism, and fever

63. List common musculoskeletal disorders that mimic hepatic and biliary disorders.
Musculoskeletal conditions that may mimic hepatic and biliary pain patterns include symptomatic midthoracic hypomobility, rotator cuff dysfunction, and subacromial/deltoid bursitis.

HEMATOLOGY

64. List the common disorders of erythrocytes, leukocytes, and platelets.

<table>
<thead>
<tr>
<th>Erythrocytes</th>
<th>Leukocytes</th>
<th>Platelets</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anemia</td>
<td>Leukemia</td>
<td>Thrombocytosis</td>
</tr>
<tr>
<td>Aplastic anemia</td>
<td>Leukocytosis</td>
<td>Thrombocytopenia</td>
</tr>
<tr>
<td>Hemorrhagic anemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypochromic (iron deficiency) anemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leukopenia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Megaloblastic anemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pernicious anemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Polycythemia</td>
<td></td>
<td></td>
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<tr>
<td>Sickle cell anemia</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

65. List the signs and symptoms of polycythemia (increased red blood cell mass).
History of headaches, blurred vision, dizziness, fainting, altered mentation, feeling of fullness in the head, altered sensation in the distal extremities, malaise, fatigue, weight loss, easy or unexplained bruising, cyanosis, digital clubbing, and hypertension

66. List the common disorders or conditions that elevate red blood cell levels.
Alcoholism, burns, chronic pulmonary disease (eg, fibrosis), dehydration (eg, vomiting and diarrhea, burns, or use of diuretics), diminished blood-oxygen tension, heart disease (eg, cor pulmonale and congenital), liver disease, renal disease, smoking, and exposure to carbon monoxide

67. List the signs and symptoms of leukocytosis (increased white blood cell count).
Signs and/or symptoms consistent with local or systemic infection (eg, fever) and inflammation or trauma

68. List the common disorders or conditions that elevate white blood cell levels.
Burns, cancer, immune system responses (eg, lupus, rheumatoid arthritis), infections, inflammatory responses (eg, tissue damage), kidney failure, leukemia, lymphoma, malnutrition, multiple myeloma, removal of the spleen, stress (eg, emotional, physical), and tuberculosis

69. List the signs and symptoms of anemia (decreased red blood cell levels).
Pail skin and nails, shortness of breath with little to no exertion (based on degree), heart palpitation, and increased pulse rate; with severe anemia, fatigue, decreased diastolic blood pressure, and changes in mentation

70. List the common disorders or conditions that lower red blood cell levels.
Addison's disease, anemia (eg, blood loss, hemorrhage, pernicious, sickle cell), bone marrow disease, bowel disease, colon cancer, excessive menstrual bleeding, hemolysis, kidney disease, lead poisoning, leukemia, malnutrition, multiple myeloma, stomach ulcers, and vitamin and/or mineral deficiencies (eg, B12, B6, folic acid, iron)

71. List the signs and symptoms of leukopenia (decreased white blood cell levels).
Cough, sore throat, fever, chills, swelling, ulceration of the mucous membranes, increased frequency of urination, painful urination, and persistent infections
72. List the common disorders or conditions that lower white blood cell levels.
Alcoholism, aplastic anemia, autoimmune/collagen-vascular diseases (eg, lupus, AIDS), bone marrow failure, Cushing’s syndrome, disorders of the spleen, infections, liver disease, radiation exposure or exposure to toxic chemicals (eg, chemotherapy), tumors, and viral infections.

73. What are the hematologic red flags?
- Evidence of platelet disorders (eg, bleeding with minor to no trauma, multiple petechiae, purpura, severe bruising, nosebleeds, hematemesis, blood in urine or stool, dark tarry stool, excessive menstrual bleeding; especially when undiagnosed, sudden, and/or unexplained)
- Evidence of anemia, especially in the presence of CNS, and cardiopulmonary manifestations
- Undiagnosed muscle and joint pain in patients with a history of hemophilia
- Undiagnosed variations in hematologic values

74. What subjective information should be obtained when hematologic pathologies are suspected?
- Presence of any red flags as previously described
- History of anemia (eg, excessive bruising or blood loss)
- Medical history including dental procedures (eg, blood transfusion, hemophilia, hepatitis, genetic, major trauma, cancer)
- Laboratory tests (eg, hematocrit, platelet count, hemoglobin concentration)
- Surgical history (eg, transplant surgery, oral surgery, major surgeries)
- History of radiation exposure or exposure to toxic chemicals (eg, chemotherapy, industrial gases)
- Integumentary changes as described in this chapter including bruising, petechia, purpura visible through the epidermis, widespread color changes, itching, body temperature, mobility, and turgor

75. List three early signs and symptoms of anemia.
Difficult or labored breathing, weakness, and fatigue

ENDOCRINE AND METABOLIC DISORDERS

76. What are two primary life-threatening metabolic conditions that can develop if uncontrolled or untreated diabetes mellitus progresses to a state of severe hyperglycemia?
- Diabetic ketoacidosis
- Hyperglycemic, hyperosmolar, nonketotic coma (HHNC)

77. What two patient types may exhibit orthostatic hypotension because of slight dehydration, especially when intense exercise increases the core body temperature?
Athletes and normal adults

78. What signs and symptoms are commonly associated with endocrine system pathologies?
Neuromusculoskeletal signs and symptoms include muscle weakness, myalgia and fatigue, bilateral carpal tunnel syndrome, periartthritis, chondrocalcinosis, spondyloarthropathy, osteoarthritis, hand stiffness, and pain.

79. What are the endocrine system red flags?

DIABETES INSIPIDUS
- Confusion
- Increased frequency of urination (polyuria, nocturia)

SIADH
- Excessive weight gain or loss
- Edema
- Headache, seizures, and muscle cramps
- Vomiting/diarrhea

ADDISON’S DISEASE
- Dark pigmentation of the skin, mucous membranes, and scars
- Hypotension
- Fatigue that improves with rest
• Arthralgias
• Tendon calcification
• Hypoglycemia

CUSHING’S SYNDROME
• Moon face
• Cervicodorsal fat pad
• Protuberant abdomen with accumulation of fatty tissue and stretch marks
• Muscle wasting and weakness
• Kyphosis and back pain secondary to bone loss
• Easy bruising
• Emotional disturbances
• Diabetes mellitus, slow wound healing
• In women: masculinizing effects

GOITER (ENLARGED THYROID)
• Increased neck size
• Hoarseness
• Difficulty breathing and swallowing

HYPERTHYROIDISM
• Proximal weakness, primarily of pelvic girdle and thigh muscles
• >50% of adults over 70: tachycardia, fatigue, and weight loss
• <50 years of age: tachycardia, hyperactive reflexes, increased sweating, heat intolerance, fatigue, tremor, nervousness, polydipsia, weakness, increased appetite, dyspnea, and weight loss
• Musculoskeletal symptoms including chronic periarthritis, pain and decreased ROM, periarticular and tendinous calcification

HYPOTHYROIDISM
• Headaches, excessive fatigue, and drowsiness
• Hoarseness and thick, slurred speech
• Intolerance to cold
• Weight gain
• Dryness of skin
• Nails become increasingly thin and brittle
• Menses become irregular
• Myxedema: nonpitting boggy edema around the eyes, hands, feet, and supraclavicular fossae
• Synovial thickening and joint effusion

THYROID CANCERS
• New onset of hoarseness, hemoptysis, elevated blood pressure

80. What red flags are associated with metabolic disorders?

METABOLIC ACIDOSIS
• Headache, drowsiness, lethargy
• Nausea, vomiting, diarrhea
• Muscle twitching
• Convulsions, coma (if severe)
• Rapid, deep breathing (hyperventilation)

METABOLIC ALKALOSIS
• Nausea, prolonged vomiting, diarrhea
• Confusion, irritability, agitation, restlessness
• Muscle twitching, cramping, weakness
• Paresthesias
• Convulsions, eventual coma
• Slow, shallow breathing

GOUT
• Joint pain and swelling (especially the first metatarsal joint)
• Fever and chills, redness
• Malaise
HEMOCHROMATOSIS
- Arthropathy, arthralgias, myalgias
- Progressive weakness
- Bilateral pitting edema (lower extremities)
- Vague abdominal pain
- Hypogonadism (lack of menstrual periods, impotence)
- Congestive heart failure
- Hyperpigmentation of the skin (gray/blue or yellow)
- Loss of body hair
- Diabetes mellitus

OSTEOPOROSIS
- Episodic back pain, kyphosis (dowager’s hump)
- Decreased activity tolerance
- Early satiety

OSTEOMALACIA
- Bone pain, skeletal deformities, fractures
- Myalgia, severe muscle weakness

PAGET’S DISEASE
- Headache and dizziness
- Periosteal tenderness, bone fractures, vertebral compression and collapse, deformity (bowing of long bones, increased size and abnormal contour of clavicles, osteoarthritis of adjacent joint, acetabular protrusion, and head enlargement)
- Compression neuropathy (spinal stenosis, paresis, paraplegia, and muscle weakness)
- Decreased auditory acuity

81. What subjective information should be obtained when endocrine system pathology is suspected?
- Presence of any red flags as previously described
- Medication use, including use of insulin or cortisol, or excessive use of antacids
- Slow wound healing
- Family history of osteoporosis
- Increase in collar size (goiter growth), difficulty in breathing or swallowing

82. List common musculoskeletal disorders that mimic endocrine system disorders.
Periarthritis and calcific tendinitis of the shoulder is common in endocrine clients and must be ruled out from other musculoskeletal disorders such as rotator cuff dysfunction, rotator cuff tears, slap lesions, labral tears, and subacromial/subdeltoid bursitis.

IMMUNOLOGIC

83. What are the four principal classifications of immunologic disorders?
The four principal classifications are immunodeficiency, hypersensitivity, autoimmunity, and immunoproliferative disorders.

84. Name the only disease known to directly attack the human immune system.
AIDS (acquired immunodeficiency syndrome)

85. How are hypersensitivity disorders classified?
Hypersensitivity disorders are grouped into four types: type I anaphylactic hypersensitivity (allergies), type II hypersensitivity (cytolytic or cytotoxic), type III hypersensitivity (immune complex), and type IV hypersensitivity (cell-mediated or delayed).

86. What neurologic disorders may be associated with immune system dysfunction?
Myasthenia gravis, Guillain-Barré syndrome, and multiple sclerosis are neurologic disorders associated with immune system dysfunction.

87. List examples of autoimmune disorders.
Examples of autoimmune disorders are fibromyalgia syndrome, rheumatoid arthritis, systemic lupus erythematosus, scleroderma, spondyloarthropathy, Reiter syndrome, psoriatic arthritis, Lyme disease, and bacterial arthritis.
88. What signs and symptoms are commonly associated with pathologies of the immunologic system?

AIDS
- Early signs and symptoms include fever, night sweats, fatigue, headache, minor oral infections, cough, shortness of breath, and skin changes (e.g., rash, nail bed changes, dry skin).
- Advanced signs and symptoms include Kaposi’s sarcoma and the presence of opportunistic diseases (TB, pneumonitis, lymphoma, thrush, herpes 1 and 2).

HYPERSENSITIVITY DISORDERS
- Signs and symptoms may be as minor as sinus drainage to as severe as coma or death of the patient.
- Additional signs and symptoms include nausea, prolonged vomiting, and diarrhea; confusion, irritability, agitation, and restlessness; muscle twitching, cramping, and weakness; paresthesias; convulsions and eventual coma; slow, shallow breathing.

NEUROLOGIC DISORDERS
- Signs and symptoms of myasthenia gravis include muscle fatigability and proximal muscle weakness aggravated with exertion, respiratory failure, ptosis, diplopia, dysarthria, and bulbar involvement (alteration in voice quality, dysphagia, nasal regurgitation, choking).
- Signs and symptoms of Guillain-Barré syndrome include muscle weakness (bilateral, progressing from the legs to the arms, to the chest and neck), diminished deep tendon reflexes, paresthesias, fever, malaise, and nausea.
- Signs and symptoms of multiple sclerosis include optic neuritis leading to unilateral visual impairment, paresthesia, nystagmus, spasticity or hyperreflexia leading to ataxia or unsteadiness, vertigo, fatigue, muscle weakness, and bowel and bladder dysfunction. Positive Babinski’s sign, positive Lhermitte’s sign, and absent abdominal reflex are also present.

AUTOIMMUNE DISORDERS
- Signs and symptoms of fibromyalgia syndrome include fatigue, depression, anxiety, short-term memory loss, decreased attention span, headaches, nocturnal bruxism, myalgia, tender points of palpation, tendinitis, bursitis, morning stiffness, low back pain, subjective swelling, and irritable bowel and bladder symptoms.
- Signs and symptoms of rheumatoid arthritis include swelling in one or more joints, early morning stiffness, recurring joint pain or tenderness, impaired joint motion, joint redness and warmth, unexplained weight loss, and fever or weakness.
- Signs and symptoms of systemic lupus erythematosus include constitutional symptoms, arthralgia, arthritis, skin rashes, anemia, pulmonary and renal disorders, CNS signs and symptoms, hair loss, and mouth, nose, or vaginal ulcers.
- Signs and symptoms of systemic scleroderma include calcinosis (abnormal deposition of calcium salts in tissues, particularly over bony prominences), Raynaud’s phenomenon, dysphagia, heartburn, hardening and shrinking of the toes and fingers, and formation of spider-like hemangiomas in the face and hands.
- Signs and symptoms of spondyloarthropathy include back pain with insidious onset; first episode occurs before age 30, episodes of pain last for months, and pain intensifies with rest and decreases with movement.
- Signs and symptoms of ankylosing spondylitis include
  - Early signs and symptoms—intermittent low-grade fever, fatigue, anemia, anorexia, painful limited spinal motion, loss of spinal motion, and inflammation of the iris (iritis or iridocyclitis)
  - Advanced signs and symptoms—constant low back pain, ankylosis of the SI joints and spine, muscle wasting in the shoulder and pelvic girdles, marked cervical kyphosis, decreased chest expansion, and arthritis in the extremity joints
- Signs and symptoms of Reiter syndrome include polyarthritis, SI/low back pain, heel pain, plantar fasciitis, low-grade fever, urethritis (precedes other symptoms by 1 to 2 weeks), and bilateral conjunctivitis and iritis.
- Signs and symptoms of psoriatic arthritis include fever, fatigue, dystrophic nail bed changes, polyarthritis, psoriasis, and sore, swollen fingers.
- Signs and symptoms of Lyme disease include rash, flu-like symptoms, migratory musculoskeletal pain, severe headaches, numbness, weakness and pain in the extremities, and poor motor coordination.
• Signs and symptoms of bacterial arthritis include fever and chills, rapid onset of monoarticular involvement (knees and shoulders most frequent), joint inflammatory symptoms/signs, restricted motion, local tenosynovitis, and skin lesions near the involved joint.

89. What are the immunologic red flags?
In addition to the signs and symptoms described previously, the following red flags should be screened:
• Development of neurologic symptoms 1 to 3 weeks after an injection (Guillain-Barré/C19e syndrome)
• New onset of inflammatory joint pain postoperatively, especially if accompanied by extraarticular signs and symptoms such as rash, diarrhea, urethritis, mouth ulcers, and raised skin patches
• Joint pain preceded or accompanied by skin rash or lesions
• Generalized weakness
• Nail bed changes (eg, dystrophic nail changes associated with psoriasis, atrophy of the fingertips, calcific nodules, digital cyanosis, and tightening of the skin associated with scleroderma)

90. What are other musculoskeletal causes of pain that must be differentially diagnosed from an immunologic disorder?
Because of the multisystem effect of immunologic disorders, it is important that a complete health history is performed to identify if musculoskeletal signs and symptoms are attributable to a mechanical origin, or whether other sources should be investigated. Close cooperation and appropriate co-management with the referring physician are crucial for the proper management of musculoskeletal cases with suspicious origins.

CLINICAL REASONING

91. Do knowledge, efficiency of data collection, and data interpretation improve with experience?
No. The literature suggests that inadequate knowledge and imprecise data collection improve with increasing clinical experience, but data integration and interpretation do not.

92. Why do errors in clinical reasoning occur?
It is well documented that human beings are for the most part noncritical thinkers and that we are prone to deductive and inductive errors in reasoning (ie, judgment errors). Additionally, the cognitive limitation of human working memory leads us to access simpler rather than more complex cognitive or problem-solving strategies (ie, shortcuts in reasoning). In actuality, it is likely that the combination of judgment errors and reliance on shortcuts in reasoning (eg, heuristics) is what leads to most errors in clinical reasoning. Finally, errors will vary based on the difficulty of the patient case, knowledge of content and context, strategy selection, and integration and interpretation of pertinent patient information.

93. What is deductive reasoning?
Deductive reasoning involves reaching a conclusion based on evidence (ie, deductive reasoning combines two or more pieces of evidence to reach a conclusion).

94. What are examples of deductive reasoning errors?
Illogical or poor reasoning, persistence of beliefs despite empiric data to the contrary, rationalizing, justifying, and using biases and heuristics to assess information are examples of deductive reasoning errors.

95. What is inductive reasoning?
Inductive reasoning uses specific pieces of evidence (ie, more than one example) to draw conclusions that are probably, but not necessarily, true (eg, generalizations, cause and effect, and analogies).

96. What are examples of inductive reasoning errors?
Examples include overconfidence in validity of beliefs, confusion of opinion or anecdotal evidence with truth, overestimation of knowledge, and basing a decision on personal interests.

97. What is iterative hypothesis testing?
Iterative hypothesis testing, as described by Kasper and Harrison, is a process used by medical practitioners to increase the efficiency of the interview process. During this process interview questions are used to confirm or refute the evolving diagnostic hypothesis. Iterative hypothesis
testing uses specific questions to probe patient answers. Iterative hypothesis testing does not replace a systematic, thorough, and complete history of present illness, past medical history, review of systems, family history, and the physical examination. Iterative hypothesis testing represents a pattern of application of inductive and deductive reasoning.

98. Give an example of iterative hypothesis testing based on a patient’s perception of illness.
The patient presents with a referral that states: “Lumbar pain, evaluate and treat.” Therapist: “What are you here for today?” Patient: “I have a pinched nerve in my back.” Therapist: “Who was the doctor who diagnosed you with this condition?” Patient: “It was not my doctor.” Therapist: “I am not certain if I understand; how did you determine that you have a pinched nerve in your back?” Patient: “About a year ago my neighbor had the same pain that I am having and he was diagnosed with a pinched nerve in his back.” In this example, if the therapist did not test the hypothesis, a serious error could have occurred.

99. List common errors or biases in clinical reasoning and a potential consequence of the error or bias.

<table>
<thead>
<tr>
<th>Error or Bias</th>
<th>Possible Consequence(s)</th>
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<tbody>
<tr>
<td>Faulty hypothesis testing and clinical reasoning skills</td>
<td>Making clinical decisions based on illogical or faulty reasoning processes or using more simplistic rather than more complex cognitive problem solving strategies</td>
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<tr>
<td>Confusion between deductive and inductive logic</td>
<td>Deductive reasoning errors or drawing conclusions that go beyond the information contained in the premises (eg, Correct: If “A” then “B”; “A” therefore “B,” Incorrect: If “A” then “B,” therefore, if “B” then “A”); inductive reasoning errors or generalizations based on specific observations that are not based on deductive reasoning (eg, All “A” are “B” ≠ All “B” are “A”)</td>
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<tr>
<td>Confusing covariance with causality</td>
<td>Presuming that two or more factors are causally related when two factors have been found to covary</td>
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<tr>
<td>Errors in detecting variance</td>
<td>Making a judgment about the relationship of two factors without understanding how the two factors covary with one another</td>
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<tr>
<td>Causal reasoning errors (ie, assuming that a causal relationship establishes proof)</td>
<td>Failure to recognize unknown factors and processes</td>
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<tr>
<td>Confusing temporal and causal succession (ie, assuming that there is a causal connection based only on temporal sequence)</td>
<td>Establishing erroneous causal relationships (ie, assuming a causal connection or that one event occurred as a result of another, when in fact one event merely occurred after another)</td>
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<tr>
<td>Overreliance on clinical prediction rules and heuristics (ie, shortcuts in reasoning)</td>
<td>Failure to identify the correct disease process, two or more independent disease processes, and/or the implications of comorbidities (eg, making clinical decisions based on choosing the simplest diagnoses capable of explaining the patient’s signs and symptoms)</td>
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<tr>
<td>Confirmation bias (eg, emphasizing or validating information that supports the clinician’s favored hypothesis while negating information that does not)</td>
<td>Failure to identify or address competing diagnoses and limiting examination to tests and measures to those that confirm the suspected diagnoses while ignoring evidence and testing that might disconfirm it; confirmation bias likely represents one of the greatest single sources of error</td>
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<tr>
<td>Persistence of resilient beliefs</td>
<td>Making a diagnosis and/or setting a course of action despite empiric data to the contrary</td>
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<tr>
<td>Adding pragmatic inferences</td>
<td>Making diagnostic assumptions that result in misdiagnoses or faulty clinical decisions</td>
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</table>
Broad generalizations (ie, applying a general statement or rule too broadly)
Failure to sample enough information
Forming inferences or assumptions that do not apply (ie, to a specific patient case, event, pathology, etc.)
Basing clinical decisions on generalizations and limited data and discontinuing the search for additional diagnoses after anticipated diagnoses are made

Erroneous integration and interpretation of data
Failure to synthesize and integrate clinical information deleteriously affects all aspects of the clinical reasoning process (eg, the ability to synthesize and integrate clinical information is the foremost distinguishing feature of a diagnostic expert)

Generating a diagnosis based on availability or recall
Generating a diagnosis based on similarity or pattern recognition
Overestimating the probability of a diagnosis and generating a false sense of frequency
Neglecting the prevalence of competing diagnoses (eg, based on weak and incomplete analogies as opposed to hypothetical deductive reasoning)

Generating a diagnosis based on the patient's perception of illness
Considering too few diagnoses (eg, hypotheses)
A type of confirmation bias in which the clinician seeks to validate a self-reported patient diagnosis
Artificially or prematurely limiting the number of plausible diagnoses

Faulty or insufficient knowledge or skill base
Making clinical decisions based on omissions that stem from a lack of knowledge or omitting more beneficial interventions secondary to lack of knowledge and/or skill

Selecting a course of action based on outcomes
Selecting less beneficial interventions in place of potentially more beneficial interventions (eg, secondary to emphasizing adverse effects, high costs, time, availability, etc.)

Selecting a course of action based on treatment-driven practice patterns
Selecting interventions based on a narrow and often ineffectual or less effective range of treatment options as opposed to selecting interventions based upon a comprehensive examination, evaluation, and differential diagnosis

Selecting a course of action based on omission
Basing clinical decisions on desired or expected outcomes rather than logic and evidence supporting clinical decisions

Considering too few interventions
Choosing the same intervention when there are additional and alternative options available

100. What are the ultimate consequences of clinical decision-making errors?
Consequences range from little to none (eg, in cases where patient conditions are self-limiting and intervention selections are conservative) to potentially catastrophic (eg, in cases where emergent medical conditions are present or developing and appropriate intervention is not provided).

101. What are some examples from within the literature of evidence-based practices that may lead to errors in clinical reasoning?
The use of clinical prediction rules to select interventions such as manipulation for the treatment of conditions such as low back pain may lead to errors in clinical reasoning.

102. What is an example of a clinical prediction rule?
A widely referenced clinical prediction rule was developed by Flynn et al. They concluded that patients with nonspecific low back pain who demonstrated the characteristics of 1) pain for less than 16 days, 2) a FABQ work subscale score of less than 19, 3) at least one hip range of motion of greater than 35 degrees of internal rotation, 4) hypomobility in the lumbar spine, and 5) no symptoms distal to the knee were more likely to respond favorably to an SI manipulation.

103. Why does the previous example provide a context for a potential error in clinical reasoning?
Any time the examination is narrowed to collect only a small subset of data the potential for error increases. The characteristics identified in Flynn et al.'s study are very broad in nature and may result in better patient outcomes in general, regardless of the intervention applied.
CHAPTER 23 QUESTIONS

1. A 64-year-old man is referred to physical therapy by his physician with a diagnosis of right knee osteoarthritis and complaints of right knee pain, swelling, and antalgic gait. During the examination he demonstrates shortness of breath, wheezing, a barrel chest deformity, and the use of accessory muscles for respiration; the patient also reports that he does not tolerate supine positioning. Which of the following conditions is most likely the cause of his respiratory symptoms?
   a. Acute bronchitis
   b. Chronic emphysema
   c. Pneumonia
   d. Weakness of respiratory muscles

2. Which pattern of stiffness is most consistent with rheumatoid arthritis?
   a. Morning stiffness lasting less than an hour
   b. Morning stiffness lasting more than an hour
   c. Stiffness that is relieved with activity
   d. Stiffness that is worsened with activity

3. A 52-year-old male is referred to a physical therapy clinic with a diagnosis of left shoulder primary impingement tendinosis; physical therapy examination and radiologic findings are consistent with this diagnosis. A thorough medical screening reveals associated symptoms of fatigue, as well as an increased appetite, thirst, and urination over the past 3 to 6 months. Additional pertinent information is as follows: Ethnicity: African American; height = 6’2” and weight = 320 lb; HDL cholesterol level = 30 mg/dl and triglyceride level = 450 mg/dl. Which of the following conditions is most likely the cause of his associated symptoms?
   a. Cardiovascular disease
   b. Hypothyroidism
   c. Renal disease
   d. Type 2 diabetes

4. What type of cancer results in the greatest number of deaths annually?
   a. Breast cancer
   b. HPV-related cancer
   c. Lung cancer
   d. Lymphoma
1. Why is medical screening necessary?
Performing a medical screen is an important step in making a physical therapy diagnosis. Medical screening assists the physical therapist in determining whether or not a patient referral to another practitioner is needed. When screening, if findings are suspicious or require diagnostic skills or equipment outside of the physical therapy scope of practice a medical referral is warranted. By gathering information from the patient history and the physical examination the therapist can determine whether a medical condition is present and not yet diagnosed, if a medical condition exists and is clinically stable, or if an existing clinical condition is medically unstable. The therapist uses screening tools such as red and yellow flag findings to recognize potential serious disorders. The therapist may be screening for visceral diseases, cancer, infections, fracture, and vascular disorders and can communicate with the physician regarding a list or pattern of signs and symptoms that have caused concern. Our role does not include suggesting the presence of a specific disease.

2. When positive screening results are found, what are the red flags versus the yellow flags?
Red-flag findings are symptoms or conditions that may require immediate medical attention and are of particular concern that physical therapy should not be the primary provider of service. They are typically indicative of nonmechanical (nonneuromusculoskeletal) conditions or pathologies of visceral origin. Examples may include fever, night sweats, unexplained weight loss, or visual disturbances.

Yellow-flag findings are potential confounding variables that may be cautionary warnings regarding the patient’s condition. They suggest that the physical therapist take pause and monitor the influence of these findings on the patient’s condition. Examples include dizziness, abnormal sensation patterns, fainting, progressive weakness, and circulatory or skin changes.

3. A therapist screens systemic origins of a patient’s signs and symptoms by considering pain referral, patient history, and clinical presentation. What are the most common sites of pain referral from a systemic disease?

Pain from systemic disease is most often referred to the chest, back, shoulder, scapula, pelvis, hip, groin, and sacroiliac (SI) joint, with the back and shoulder being the most common within this group.

4. Therapists must be aware of certain pathologies and their associated sites of pain referral. List the sites of referral and their associated pathologies.

<table>
<thead>
<tr>
<th>Pain Referral Site</th>
<th>Common Pathology</th>
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<tbody>
<tr>
<td>Head, face, and TMJ</td>
<td>Meningitis</td>
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<td>Primary brain tumor</td>
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<td>Subarachnoid hemorrhage</td>
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<td>Cervical and shoulder</td>
<td>Metastatic lesions (leukemia, Hodgkin’s disease)</td>
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<td></td>
<td>Cervical bone tumors</td>
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<td>Cervical cord tumors</td>
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<td>Pancoast’s tumor</td>
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<td>Esophageal cancer</td>
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<td>Thyroid cancer</td>
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<td>Myocardial infarction</td>
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<td>Cervical ligamentous instabilities (possible cord compromise)</td>
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<td></td>
<td>Cervical and shoulder peripheral entrapment neuropathies</td>
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<td>Thoracic spine and ribs</td>
<td>Lumbar spine</td>
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<td>Myocardial infarction</td>
<td>Multiple myeloma</td>
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<td>Unstable angina</td>
<td>Primary bone tumors</td>
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<td>Stable angina</td>
<td>Neurogenic tumors (sacrum)</td>
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<td>Pericarditis</td>
<td>Prostate cancer</td>
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<td>Pulmonary embolus</td>
<td>Testicular cancer</td>
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<td>Pleurisy</td>
<td>Colorectal cancer</td>
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<td>Pneumothorax</td>
<td>Back-related infection (osteomyelitis)</td>
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<td>Pneumonia</td>
<td>Cauda equina syndrome</td>
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<td>Cholecystitis</td>
<td>Spinal fracture</td>
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<td>Peptic ulcer</td>
<td>Abdominal aneurysm</td>
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<td>Pyelonephritis</td>
<td>Lymphoma</td>
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<td>Nephrolithiasis</td>
<td>Endocarditis</td>
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<td>Spinal fracture</td>
<td>Myocarditis</td>
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<td>Metastatic lesions</td>
<td>Acute pyelonephritis</td>
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<td>Pancreatic cancer</td>
<td>Perinephritic abscess</td>
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<td>Breast cancer</td>
<td>Nephrolithiasis</td>
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<td>Multiple myeloma</td>
<td>Ureteral colic (kidney stones)</td>
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<td>Urinary tract infection</td>
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<td>Dialysis (first-use syndrome)</td>
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<td>Renal tumors</td>
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<td>Obstruction (neoplasm)</td>
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<td>Irritable bowel syndrome</td>
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<td>Crohn’s disease</td>
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<td>Diverticular disease</td>
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<td>Pancreatic disease</td>
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<td>Appendicitis</td>
</tr>
<tr>
<td></td>
<td>Retroversion of the uterus</td>
</tr>
<tr>
<td></td>
<td>Uterine fibroids</td>
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<td></td>
<td>Ovarian cysts</td>
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<tr>
<td></td>
<td>Endometriosis</td>
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<tr>
<td></td>
<td>Pelvic inflammatory disease (PID)</td>
</tr>
<tr>
<td></td>
<td>Incest/sexual assault</td>
</tr>
<tr>
<td></td>
<td>Rectocele, cystocele</td>
</tr>
<tr>
<td></td>
<td>Uterine prolapse</td>
</tr>
</tbody>
</table>
Ewing’s sarcoma
Arterial insufficiency
Abdominal aortic aneurysm
Avascular necrosis
Kidney (renal) impairment; kidney stones
Urinary tract infection
Testicular cancer
Abdominal or peritoneal inflammation (psoas abscess)
Ankylosing spondylitis
Appendicitis
Crohn’s disease; ulcerative colitis
Diverticulitis
Osteomyelitis (upper femur)
PID
Reiter’s syndrome
Inflammatory arthritis (RA, SLE, seronegative arthropathies, gout)
Septic hip bursitis
Tuberculosis
Osteomalacia, osteoporosis
Gaucher’s disease
Paget’s disease
Hemochromatosis
Hemophilia
Ectopic pregnancy
Femoral artery catheterization
Knee, leg, ankle, or foot
Peripheral arterial occlusive disease
Deep vein thrombosis
Compartment syndrome
Septic arthritis
Cellulitis

5. List the organs and their respective locations in the abdominal quadrants.

ANTERIOR VIEW OF ABDOMINAL CAVITY

From Goodman CC, Snyder TEK: Differential Diagnosis for Physical Therapists: Screening for Referral, 5e, 2013, St. Louis, Elsevier, Inc.
6. What are the components of the physical examination in the abdominal region when screening for visceral disorders?

The components of the physical examination of the abdominal region are inspection, auscultation, percussion, and palpation. Inspection is the process of visually looking at the body for symmetry, alignment, skin color, and scars present. Auscultation follows inspection during examination of the abdominal region. Auscultation is performed to listen for bowel sounds. Normal bowel sounds are made by movement of the intestines as food and liquid pass through. Bowel sounds occur frequently, on the order of every 2 to 5 seconds, although there is a lot of variability. Palpation in the abdominal region is usually deep palpation (firm pressure) and is used to assess for tenderness and presence of visceral organs. Percussion is used to determine the size, shape, and density of tissue using sound. Direct percussion is performed using the examiner’s fingers to tap directly on the surface of the body. Indirect percussion requires the middle finger of the examiner’s nondominant hand to be positioned against the patient’s skin and for the examiner to strike above or below the IP joint of the third digit with the dominant hand.

Blunt percussion using the examiner's border of the fifth metacarpal (ulnar border) with the hand in a fist is performed when assessing kidneys. The examiner listens for sounds that can range from tympani to a flat sound based on the tissue. From Boissonnault WG: Examination in Physical Therapy Examination: Screening for Medical Disease, 2e, New York, 1995, Churchill Livingstone.

7. What types of percussive sounds can be heard in the body?

Percussion is used to detect changes in the density of an organ. Tissue density can range from high to low with percussive sounds ranging from flat, dull, resonant, hyperresonant, and tympanic. A dull sound indicates the presence of a solid mass under the surface such as the heart. A more resonant (vibrating) sound indicates hollow air-containing structures, such as the lungs and the hollow viscera of the abdomen. Tissues can also have abnormal fluid or a mass altering the typical sound heard in that anatomic location. In addition to producing different notes, percussion can also produce different sensations in the fingers, such as vibration.
8. What are the specific qualities elicited by palpation?

<table>
<thead>
<tr>
<th>Quality</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Texture</td>
<td>The surface characteristics of the skin and hair are noted (brittle, coarse, thick, thin, roughened, or smooth)</td>
</tr>
<tr>
<td>Moisture</td>
<td>Assess the moisture content of the skin, hair, and mucous membranes; are they moist and supple or dry and cracked?</td>
</tr>
<tr>
<td>Skin temperature</td>
<td>Palpate the head, face, trunk, arms, hands, legs, and feet to assess the local skin temperature and the distribution of heat</td>
</tr>
<tr>
<td>Characteristics of masses</td>
<td>When a mass or enlarged organ is discovered, record its size, shape, consistency, mobility, surface regularity, and presence or absence of expansile or transmitted pulsation</td>
</tr>
<tr>
<td>Precordial cardiac thrust</td>
<td>Palpate the precordium (portions of the body over heart and lower chest) for signs of heart action</td>
</tr>
<tr>
<td>Crepitus</td>
<td>During examination of the bones, joints, tendon sheaths, pleura, and subcutaneous tissue, feel for crepitation</td>
</tr>
<tr>
<td>Tenderness</td>
<td>Discomfort or pain on palpation of accessible tissues and over major organs should be noted; how much pressure is required to induce the uncomfortable sensation?</td>
</tr>
<tr>
<td>Thrills</td>
<td>Palpate the precordium for thrills; if bruits (abnormal sounds) are heard in the major arteries, palpate them for thrills</td>
</tr>
<tr>
<td>Vocal fremitus</td>
<td>Palpation of vocal vibrations through the chest wall provides important information about the underlying pleura and lung</td>
</tr>
</tbody>
</table>

9. Describe the special tests for palpation and percussion of the liver in an adult.

The adult liver is normally not palpable below the right anterior inferior costovertebral margin (acute angle created between the vertebral column and the twelfth rib).

With percussion of the liver, the examiner begins at a point lateral to the umbilicus at the midclavicular line and percusses using a technique with the nondominant hand’s second or third digits as the “dummy fingers” and the dominant second and third digits to perform the percussion from the point lateral to the umbilicus in a superior direction. The test is positive if the liver spans greater than 10 cm.

To palpate for hepatomegaly the examiner uses the tips of the fingers with two hands inferior to the ribs in the midclavicular line. The examiner palpates for the liver to descend with inspiration. From Goodman CC, Snyder TEK: Differential Diagnosis for Physical Therapists: Screening for Referral, 5e, 2013, St. Louis, Elsevier, Inc. Continued
10. How strong is the evidence to support the tests for palpation and percussion of the liver?
There is minimal evidence to support the use of palpation and percussion of the liver for hepatomegaly.

11. What is Murphy's sign and how is it performed?
Murphy's sign is a test for cholecystitis. This test is performed with the patient in the supine position and the examiner placing his or her hands on the right upper abdominal quadrant at the inferior costal margin. The patient inspires and the examiner palpates deeply in the subcostal region with the fingertips. The test is considered positive if pain is perceived during inspiration or the patient stops inspiration because of discomfort.

12. Name and describe palpation and percussion tests to assess for splenomegaly.
Nixon's percussion test is performed with the patient in right side lying. The examiner percusses the posterior axillary line from the distal end of the lung to the middle anterior costal margin. This test is
positive when dullness extends over 8 cm above the costal margin, with normal being 6 to 8 cm. Castell’s percussion test is performed with the patient in the supine position. The examiner places his or her fingers over the eighth or ninth intercostal space, in line with the left anterior axillary line, and performs percussion during normal breathing and at full inspiration. The test is positive if dullness is noted during full inspiration.

Palpation of the spleen is performed through a bimanual palpation technique. The examiner stands on the patient’s right side and reaches over to lift the left rib cage with the left hand. The right hand then palpates the costal margin and underneath the ribs to feel the spleen. The test is positive if the examiner is able to palpate the enlarged spleen. Middleton’s maneuver is another test described with the patient lying supine with the examiner on the left side of the patient. The examiner stands at the patient’s left shoulder and palpates the spleen under the left costal margin by flexing the fingers and palpating for the spleen while the patient inspires.

From Boissonnault WG: Examination in Physical Therapy Examination: Screening for Medical Disease, 2e, New York, 1995, Churchill Livingstone.

13. In isolation, palpation and percussion tests for splenomegaly have minimal evidence to support their use and predictive value. When palpation and percussion are performed together does the evidence support the use of these tests? There is moderate evidence to support the use of percussion and palpation tests of the spleen together. Sullivan and Williams (1976) reported sensitivity at 88% and specificity at 83% with a positive likelihood ratio of 5.18 and negative likelihood ratio of 14. Barkun et al. (1991) reported 46% sensitivity and 97% specificity with a positive likelihood ratio of 15.33 (greater than 10 alters posttest probability of a diagnosis to a moderate degree) and a negative likelihood ratio at 0.56.

14. Name and describe palpation and percussion tests to assess for the kidney. Murphy’s percussion test is also known as costovertebral angle tenderness (CVAT) or Murphy’s punch sign and is used to rule out kidney involvement or pseudorenal pain. When performing this percussion test the patient can either be in prone or sitting. The examiner places one hand over the costovertebral angle (CVA) of the patient’s back. Next, the examiner provides a percussive thump with the other hand, allowing the kidney to vibrate. A positive test is noted by either costovertebral tenderness or reproduction of back/flank pain signaling a red flag for renal involvement. If the patient experiences no pain after the thump is performed, then renal involvement is ruled out.

Palpation of the kidney is performed on the right side of the patient in supine position on a hard surface. The kidney is lifted by one hand in the CVA between the twelfth rib and the vertebral column. The patient then takes a deep breath; this causes the kidney to descend. As the patient inhales the anterior hand is pushed firmly and deeply beneath the costal margin in an effort to trap the kidney. When successful,
the anterior hand can palpate the size, shape, and consistency of the organ as it slips back into normal position. The left kidney is usually not palpable because of its position beneath the bowel.

The diagnostic accuracy of this test is unknown as it appears to have not been tested.

15. What structure(s) would be involved if a patient has a positive Murphy’s percussion test, and how confident can a therapist be in the predictive value of this test?

Murphy’s percussion test is preformed to assess for costovertebral tenderness and kidney involvement. The therapist uses the ulnar border (fifth metacarpal) of the fist to thump over the twelfth rib at the CVA with the patient either in sitting or prone position. The test is positive if pain is reproduced in the subcostal region, flank, or lateral aspect of the abdomen.

16. What are the clinical findings related to appendicitis and the medical screens that would be utilized?

- Discomfort and sharp pain in the RLQ of the abdominal area
- Abdominal tenderness and guarding of the RLQ
- McBurney point tenderness
- Tip of twelfth rib tenderness
- Pain in the belly button without trauma
- Right low back pain (LBP) without trauma and insidious onset
- Difficulty standing straight
- Psoas sign
- Obturator sign
- Rovsing sign (pain in RLQ reproduced with rebound testing of the LLQ)
- Increased heart rate and temperature
- Alvarado’s score > 6
17. Where is McBurney’s point and what does tenderness to palpation of this area indicate?
McBurney’s point is located in the RLQ of the abdomen, midway between the umbilicus and the anterior superior iliac spine (ASIS). Rebound tenderness or Blumberg’s sign (examiner palpatess deeply and then releases quickly) in this area would indicate appendicitis. Researchers have found tenderness to be more sensitive and Blumberg’s sign to be more specific.

18. What is Alvarado’s score and what diagnostic utility does it have?
Alvarado score assesses the components of a physical examination for acute appendicitis. Alvarado’s score determines the probability of a patient having acute appendicitis based on six clinical examinations and two laboratory tests. If the patient reports the following, he or she receives one or two points for each. The total score is then calculated out of 10.

Scores <5 were less likely to be acute appendicitis, and scores >6 were more likely. Sensitivity measures were recorded as high as 97% with a negative LR of 0.09 and QUADAS score of 9. When using a cutoff of >7, the specificity has been shown to be 100%.

<table>
<thead>
<tr>
<th>Examination/Test</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>The patient reports that pain migrated from epigastric region to right lower quadrant</td>
<td>1</td>
</tr>
<tr>
<td>The patient reports anorexia</td>
<td>1</td>
</tr>
<tr>
<td>The patient reports nausea and vomiting</td>
<td>1</td>
</tr>
<tr>
<td>The patient has tenderness in the right lower quadrant</td>
<td>2</td>
</tr>
<tr>
<td>Positive Blumberg’s sign (rebound tenderness) over McBurney’s point</td>
<td>1</td>
</tr>
<tr>
<td>Fever</td>
<td>1</td>
</tr>
<tr>
<td>Leukocytosis</td>
<td>2</td>
</tr>
<tr>
<td>Shift to left (white count shifts to left)</td>
<td>1</td>
</tr>
<tr>
<td>Total Score</td>
<td>10</td>
</tr>
</tbody>
</table>

19. What are the clinical signs and causes of an iliopsoas (liacus or psoas) abscess, and what tests can be used to screen for this disorder?
Signs and symptoms include fever, night sweats, pain; LBP, lower abdominal, pelvic, or anterior hip pain (medial thigh or groin); and sometimes knee pain, antalgic gait, pain, and swelling with palpation. Unilateral involvement can be associated with appendicitis, but it can be bilateral with generalized peritonitis. These abscesses are often associated with appendicitis, diverticulitis, Crohn’s disease, kidney infection, or vertebral osteomyelitis status post lumbar spine surgery.

Screening tests for the psoas include the following:
- Heel tap—Involves a gentle tap to the heel of the involved side.
- Hop test—Ask the patient to hop on the involved lower extremity.
- Iliopsoas muscle test—the patient is in the supine position with both lower extremities relaxed.
- The examiner flexes the hip with the knee extended as in a straight leg raise. The examiner places resistance on the thigh to create an isometric hip flexion contraction. Pain is assessed as the patient resists this force with comparison to the uninvolved side. The test is positive if pain is reproduced in the lower quadrant.
- Palpation of the iliopsoas—the patient is in a supine position with the hips and knees flexed and supported at a 90-degree angle. The examiner slowly presses the pads of the fingers into the abdomen in a caudal and medial direction at approximately one third the distance from the ASIS toward the umbilicus. The therapist can isolate the location by asking the patient to initiate hip flexion to help isolate the muscle and avoid palpating the bowel. A positive test is pain reproduction in the lower quadrant or abdomen.

20. In addition to the iliopsoas muscle, what muscle test might be painful in the presence of appendicitis or peritonitis, and how would you test for involvement?
Either of these conditions can irritate the obturator muscle and produce RLQ pain when performing an isometric resistant contraction of the obturator. The test is performed with the patient in a supine position. The examiner performs active assistive hip and knee flexion to 90 degrees. In that position, the hip is rotated internally and externally. The examiner assesses for a painful response in the RLQ.
21. What is the normal size of the aorta?
The abdominal aorta lies slightly to the left of the midline of the body. A normal abdominal aorta is usually less than 3 cm wide. If the patient can relax, the abdomen should be deeply palpated a few centimeters above the umbilicus, slightly left of the midline, to detect an expansile pulse indicating a widened aorta.

22. Describe the palpation and auscultation techniques to assess for an abdominal aortic aneurysm, and discuss the findings that indicate a positive test.
The patient is in a supine position. The therapist applies pressure underneath the left side of the xiphoid process. With the pads of the fingers, gently apply a posterior pressure to assess for a pulse, gradually move inferior until a pulse is felt. In the area that the pulse is strongest, the therapist uses two index fingers over the spot where it is the strongest and then move the fingers apart until the pulse is no longer felt. Move the fingers back in to feel the outer borders of the pulse, and judge the distance the fingers are apart. This process is repeated until the therapist reaches the umbilicus at which the bifurcation of the aorta into the common iliac arteries occurs. Obesity, voluntary guarding, and firm musculature limit the sensitivity of the examination.

  Auscultation of the aorta is performed with a stethoscope over the area of the blood vessel. No abnormal sound should be present, such as a whooshing sound or strong pulse that would indicate a bruit. Aneurysms >5 cm in diameter have a high risk of rupture and should be considered for elective surgery; physical examination is only 75% sensitive for detecting aneurysms of this size.

23. What are Wells criteria for a pulmonary embolism? How confident are you in using this test?

| Clinical signs and symptoms of DVT (pain with palpation of the deep veins and leg swelling at a minimum) | +3.0 |
| Pulmonary embolism is as likely or more likely than an alternative diagnosis | +3.0 |
| Pulse >100 | +1.5 |
| Previous history of DVT or PE | +1.5 |
| Immobilization or major surgery in the past 4 weeks | +1.5 |
| Hemoptysis | +1.0 |
| Active cancer with ongoing treatment or within the past 6 months | +1.0 |

A score less than or equal to 4; PE is unlikely, a score >4; PE is likely

This test has demonstrated higher sensitivity overall than specificity in the studies scoring >10 QUADAS score, ranging from sensitivity of .89 to .93 and specificity of .65 to .98.

24. List the Framingham criteria for heart failure.

  Major criteria
  1. Paroxysmal nocturnal dyspnea or orthopnea
  2. Neck vein distention
  3. Rales
4. Cardiomegaly
5. Acute pulmonary edema
6. S3 gallop
7. Hepatogjugular reflex

Minor criteria
1. Ankle edema
2. Nocturnal cough
3. Dyspnea on exertion
4. Hepatomegaly
5. Pleural effusion
6. Tachycardia (>120 beats per minute)

Heart failure is diagnosed by having two major criteria present or one major criterion with two minor.

25. When should a therapist auscultate for bowel sounds, and what are the normal findings?
Normal sounds are a result of peristaltic activity and consist of clicks and gurgles. Some abnormal bowel sounds are:
- High-pitched tinkling—usually from tension of air/fluid in a loop of dilated bowel. This suggests obstruction.
- Rushes—if located at one area, usually are caused by air/fluid being forced through a small partially occluded lumen. This suggests partial obstruction, especially if associated with concurrent abdominal activity.
- Hyperactive—sometimes normal, but if combined with abdominal complaints can indicate early obstruction or a GI bleed.
- Hypoactive or absent bowel sounds—sometimes normal, but combined with complaints can indicate paralytic ileus (a halt in peristaltic activity as a result of extreme irritation from obstructive peritonitis or unknown reasons). Bowel sounds cannot be said to be absent unless they are not heard after listening for 3 minutes.

26. Which test uses a tuning fork to assess for fractures, and how confident can you be that they are accurate?
Several tests have been described for assessment of fractures with a tuning fork. The Barford test for fracture assessment has been described for the screening of hip or femoral fractures. With this test the patient lies in a supine position, and the examiner places a stethoscope on the pubic symphysis or at the distal end of the femur. The examiner strikes the tuning fork and places it on the patella of the involved lower extremity. The test is considered positive if the examiner hears that the sound is muffled through the involved lower extremity compared with the uninvolved.

The sensitivities for this test have ranged in the low 80s to low 90s. However, the negative likelihood ratios reported are 0.4 and higher. The QUADAS scores were 8 and higher. With this likelihood ratio, there is still a 40% chance that the patient could have a fracture. In summary, there is moderate but not strong evidence to support the use of these tests.

BIBLIOGRAPHY


**CHAPTER 24 QUESTIONS**

1. Which of the following tests would be the best predictor to determine whether a patient has appendicitis?
   - a. iliopsoas muscle test
   - b. + McBurney’s point
   - c. Alvarado’s score >6
   - d. Heel test

2. You are evaluating a 34-year-old female for LBP. She reports that in the past 2 days she has been nauseous to the point of vomiting and has had chills. She relates to significant PMH. You take her temperature and note she has a low-grade fever. Which of the following tests would be most indicated, in addition to lumbar ROM and pain assessment?
   - a. Auscultation of bowel sounds
   - b. Murphy’s sign
   - c. Iliopsoas muscle test
   - d. Murphy’s percussion test

3. You are evaluating a mildly obese 53-year-old female who was referred to you for evaluation of the thoracic region. She relates she has had intermittent mid-thoracic pain, right greater than left for 3 weeks. She now has acute onset of right upper quadrant pain after a work luncheon celebrating the retirement of a colleague. What special test would be most indicated given this patient’s history?
   - a. Murphy’s sign
   - b. Assessment for splenomegaly
   - c. McBurney’s point assessment
   - d. Percussion of the abdomen
1. List the common developmental milestones.
   - Rolls prone to supine: 4 months
   - Rolls supine to prone: 6 months
   - Sits alone: 6 to 7 months
   - Creeps: 9 months (not all children creep)
   - Pulls to stand: 9 to 10 months
   - Cruises: 10 months
   - Walks well: 12 to 14 months
   - Climbs up steps with railing, marking time: 2 years
   - Jumps: 2 years
   - Down steps with railing marking time: 3 years
   - Hops: 4 years
   - Skips: 5 years

2. Describe the normal progression of lower extremity alignment in children.
   - Newborns: varus knees
   - 18 months: straight knees
   - 2.5 years: valgus knees
   - 4 to 6 years: normal alignment

3. When do children develop an adult gait pattern?
   Gait laboratory studies show that the normal pattern of adult gait is established at age 3, although heel strike is seen as early as 18 months. A stable pattern in the adult mode is present by age 7, but stride length continues to increase with increases in height and leg length.

4. What lower extremity changes normally occur with growth?
   Femoral anteversion decreases from 40 to 15 degrees at maturity, whereas tibial rotation increases from 5 degrees of external rotation to 15 degrees at skeletal maturity.

5. What are growing pains?
   Although not well defined, growing pains are nonspecific intermittent pains usually occurring at night but often coming and going. They can be in the quads, calves, or other muscle groups and usually occur during growth spurts. It is hypothesized that they are related to rapid bone growth and muscle fatigue while trying to accommodate the new length. New evidence classifies growing pains as a regional pain syndrome often related to mild sensory processing disorders such as increased responses to cold, vibration, and deep pressure. Generally, slow stretching, warmth, and massage will help resolve symptoms over time.

6. Name the standardized tests commonly used in pediatric physical therapy. When are they useful?

<table>
<thead>
<tr>
<th>Tool</th>
<th>Areas Evaluated</th>
<th>Ages Evaluated</th>
<th>Type of Tool</th>
<th>Strengths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Denver (DDDST II)</td>
<td>Motor, cognitive, language, social, and adaptive</td>
<td>1 week–6.5 years</td>
<td>Screen</td>
<td>Quick and easy to learn</td>
</tr>
</tbody>
</table>
### Table of Motor Assessment Instruments

<table>
<thead>
<tr>
<th>Instrument</th>
<th>Description</th>
<th>Age Range</th>
<th>Evaluation</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peabody (PDMS II)</td>
<td>Gross and fine motor</td>
<td>1–72 months</td>
<td>Evaluation</td>
<td>Allows for emerging skills</td>
</tr>
<tr>
<td>Bayley III</td>
<td>Motor, cognitive, language, social, and adaptive</td>
<td>1–42 months</td>
<td>Evaluation</td>
<td>Considered gold standard</td>
</tr>
<tr>
<td>Movement assessment battery for children (MABC-2)</td>
<td>Motor, dexterity, and balance</td>
<td>3–16 years</td>
<td>Evaluation (screen also available)</td>
<td>Good for evaluation of milder movement disorders</td>
</tr>
<tr>
<td>Toddler infant motor evaluation (TIME)</td>
<td>Motor, functional skills</td>
<td>4 months–3.5 years</td>
<td>Evaluation</td>
<td>Some qualitative assessment; subscales for atypical tone</td>
</tr>
<tr>
<td>Bruiniks-Oseretsky II</td>
<td>Gross and fine motor, bilateral coordination</td>
<td>4–21 years</td>
<td>Evaluation</td>
<td>Tests bilateral coordination and balance</td>
</tr>
<tr>
<td>Pediatric evaluation of disability inventory (PEDI)</td>
<td>Functional activities</td>
<td>6 months–7.5 years (functional level; not chronological age)</td>
<td>Evaluation</td>
<td>Assesses functional skills in children with motor and cognitive disabilities</td>
</tr>
<tr>
<td>Gross motor function measure (GMFM)</td>
<td>Motor skills</td>
<td>5 months–16 years</td>
<td>Evaluation</td>
<td>Used to document progress in children with CP and Down’s syndrome</td>
</tr>
<tr>
<td>Test of infant motor performance (TIMP)</td>
<td>Motor skills</td>
<td>32 weeks’ gestation–4 months</td>
<td>Evaluation</td>
<td>Standardized tool to evaluate early motor skills in infants; can be used preterm</td>
</tr>
<tr>
<td>Miller first step</td>
<td>Motor, cognitive, language, social, and adaptive</td>
<td>2 years, 9 months–6 years, 2 months</td>
<td>Screen</td>
<td>Identify children at risk</td>
</tr>
</tbody>
</table>

7. **How early can children benefit from using a wheelchair or powered mobility?**

For parents who want a convenient way to move the child while shopping, a stroller may be all that is needed, provided it has proper support and does not promote poor positioning. If a child will have the ability to self-propel in the future, he or she should be started with a manual chair and not a stroller-type seating system. For children who will not self-propel, a chair with adequate support and necessary modifications would be appropriate. Some children may need a powered chair but not if the home has insufficient space to make it useful. Children as young as 18 months of age can be competent, independent users of powered mobility. One consistent movement, such as an eye blink or wrist twitch, and some amount of cognitive ability are all that is needed for power mobility to be attempted. Current research looking at the link between mobility and cognition indicates that early simple power mobility as young as 6 to 9 months old can assist children with motor delays to improve exploration and increase curiosity and problem solving. For any seating system, consulting with a therapist with specialized skills in seating and mobility is advised.

8. **What is the role of physical therapy for children with torticollis?**

Children with congenital muscular torticollis demonstrate decreased cervical range of motion in rotation to the same side and lateral flexion to the opposite side of the tight sternocleidomastoid muscle (SCM). It will generally be present as a result of in utero positioning, or postdelivery positioning in
a reclined position before the development of head control, allowing the head to tip to one side. Cervical restriction patterns, other than the aforementioned pattern, may suggest other skeletal or neurologic issues. Often, as a result of the head positioning, shortening of the trunk on the same side as the tightness leads to decreased use of the opposite side of the trunk. Prone positioning often results in hiking of the same side hip with weight shifting away from that side. Radiologic assessment should be considered if any atypical presentation is noted to rule out other pathologies like Klippel-Fiel syndrome. Treatment for torticollis ranges from aggressive stretching, bracing, and positioning to encouraging active motion and using vision to align the head and body. However, active movement and positioning appear to be the most successful, especially in children with positional torticollis or muscular torticollis. Torticollis with palpable sternomastoid tumor is the most resistant to treatment. Early age at initiation of treatment is also associated with positive results from conservative treatment.

9. What is deformational plagiocephaly?
Deformational plagiocephaly is a flattening of the skull, causing asymmetry in alignment of the ears, orbits, or jaw if the flattening is on one side, or elongation of the skull if the flattening is centrally located. It is often accompanied by torticollis, and generally one is the cause of the other. Once evaluation, usually by neurosurgery or plastic surgery, has determined that the change in shape is not caused by premature fusion of the sutures (craniosynostosis), the head shape may benefit from remodeling using molding helmets or bands. Helmets or bands are generally worn 23 hours a day, for 3 to 6 months, and are fabricated by orthotists. Referral for the helmet should occur at or before 5 months of age, as the use of a helmet is most effective before 1 year of age. Cranial measurements can be used to assess progress and determine the need for a helmet; often these are done digitally at the orthotist’s office. Helmets are an FDA-approved device and must be made by certified professionals.

10. Is developmental dysplasia of the hip (DDH) the same as congenital dislocation of the hip (CDH)?
Yes. DDH used to be called CDH, but the newer terminology better describes its dynamic nature. DDH refers to a wide spectrum of hip abnormalities, ranging from complete dislocation of the femoral head to mild acetabular abnormality or laxity of the hip. It is more common in females (in the left lower extremity), in children with a family history of the disorder, and in first-born children. Breech births, decreased uterine space, metatarsus adductus, and torticollis are also associated with DDH. Screening done early in the first week of life shows a higher incidence of DDH than screening later in the first week, indicating some level of spontaneous resolution without treatment.

11. Describe the classic tests used to evaluate DDH.
- Ortolani sign—the child is supine and the examiner grasps the flexed thigh with thumbs on the inner thigh and fingers on the greater trochanters. As the hip is abducted and the greater trochanter is elevated, a clunk is felt, indicating that the hip is reduced. This test is less sensitive after 2 months of age; mnemonic: Ortolani’s = out to in.
- Barlow’s test—begin this test the same as the Ortolani procedure; adduct the flexed hip and gently push the thigh posteriorly, testing for dislocation. This test is also less sensitive after 2 months of age because of muscular development.
- Galeazzi sign or Allis sign—a test of apparent thigh length. The patient lies supine with hips and knees flexed to 90 degrees. In a positive test, one knee is higher than the other, indicative of subluxation/dislocation on that side. The results will not be accurate if bilateral DDH is present.
- Ultrasound is also used to diagnose DDH, but studies have shown no increase in effective diagnosis using ultrasound versus clinical diagnosis.

12. How is DDH treated?
Infants with DDH who are under 6 months of age usually are treated with the Pavlik harness. Previous treatments included double or triple diapering, but this has not been shown to be any more effective than no treatment. Treatment for older children varies with age. In children younger than 1.5 years, reduction probably will be attempted (with or without prior traction), and older children usually need open surgical reduction, possibly with proximal femoral shortening and a pelvic osteotomy (such as the Salter’s or Pemberton’s procedure). Patients who are not corrected in childhood tend to have a high incidence of osteoarthritis and need for surgical intervention later.
13. What is the role of physical therapy in the treatment of DDH?

Although the harness is in place, the therapist can be a resource for positioning that fosters development, such as an adapted prone position. Otherwise, the child remains in supine with hips in flexion, external rotation, and abduction, which limit the development of head control and trunk activity. Occasionally, physical therapy will be requested for a child just out of a harness, because of difficulty with prone positioning and active hip extension after prolonged positioning in hip flexion and external rotation. This can limit the development of rolling and transitions in sitting, as well as movement in prone. If an older child is referred, pool therapy or kicking-out exercises in a warm bathtub at home are excellent choices for treatment. Tricycles and bicycles with adjustable seat heights are also helpful for increasing hip range of motion (ROM) and weight bearing. If a child is treated after age 6, the gluteus medius and maximus have worked at a mechanical disadvantage for a long period and the child may walk with an abductor lurch or trunk shift. Such walking habits are hard to break without the use of visual feedback (eg, walking toward a mirror) but may also be addressed well in an aquatic environment where fear of falling is reduced.

14. What are the various types and causes of clubfoot?

a. Positional—a normal foot that was held in an abnormal position in utero. The bony alignment is normal and the foot is usually corrected by stretching or a short course of casting.
b. Teratologic—associated with neurologic disorders such as spina bifida
c. Syndromic—associated with an overall genetic syndrome such as arthrogryposis. Both teratologic and syndromic clubfeet almost always require surgery as definitive treatment, although casting does help stretch the soft tissues in preparation for surgery.
d. Congenital—present with abnormal bony deformity at birth but not associated with any neuromuscular causes or syndromes

15. What are the components of a clubfoot (talipes equinovarus)?

- Hindfoot varus and equinus
- Supination/adduction of the forefoot
- Medial and plantar rotation of the talus

This can be seen either unilaterally or bilaterally. Usually this is accompanied by altered muscle tone or length, depending on the amount of deformity and age of the child.

16. How are physical therapists involved in treating children with congenital clubfoot?

The best treatment begins as close to birth as possible and consists of repositioning of the foot, either manually or surgically, followed by casting. Forced dorsiflexion by serial casting must be avoided as a rocker-bottom foot may develop. However, splinting in ankle-foot orthosis (AFO), or taping, especially if the infant is in the neonatal intensive care unit with other issues, can be used to gain range of motion and improve positioning. Ponseti’s technique of manipulation and casting, followed by Achilles tenotomy, if needed, has shown up to 90% success, reducing the need for surgical correction. After surgery or casting/bracing, the physical therapist may be involved in teaching postoperative exercises to maintain or regain ROM and to regain strength in the muscles of the calf and foot. The French physical therapy method includes surgical lengthening, physical therapy for range of motion and muscle stimulation, and rigid and elastic taping for positioning. Occasionally a combination of methods is needed to address a significant deformity. However, there is evidence to show delay in motor skill development related to clubfoot; therefore, therapy is needed to address these delays as well.

17. What is brachial plexus palsy (BPP) in infants?

BPP is the term commonly used to describe injury to the brachial plexus during birth. Larger infants (such as those born to mothers with gestational diabetes) are at greater risk, as are breech and assisted (forceps or vacuum) deliveries. Erb’s palsy (C5, C6) or waiter’s tip deformity has the best prognosis, followed by Klumpke’s palsy (C8–T1); complete plexus palsy has the worst prognosis. Several types of nerve injuries can occur, from traction injuries to rupture of the axon with sheath intact, to complete neuronal rupture. Prognosis will also depend on the type of neuronal injury. Occasionally, a clavicle fracture accompanies the injury and should be ruled out.

18. How is BPP treated?

Approximately 80% to 90% of children recover spontaneously, but there are indicators for a better prognosis. Traction injuries tend to recover over time, whereas avulsion injuries are less responsive. If movement is not regained in the first 4 months, the child should be referred for further evaluation and possible surgical intervention. Initial therapy involvement includes positioning to decrease further stretch on the shoulder and prone-supported weight-bearing activities to stimulate muscle activity.
Therapy can also minimize the likelihood of a contracture when the muscles recover to whatever level they can reach. Stimulation of muscle activity can help to diagnose return in an injured nerve and is a very important part of the process. However, return of nerve function can often be accompanied by paresthesias, which can be upsetting to the child and the parents or caregivers. This should be noted as a typical part of the return of function and worked through with firm touch and deep pressure, as light touch can be noxious.

19. What actions can be taken to make a baby move its arms to test for BPP?
Tactile stimulation along the muscles, stimulation of the grasp reflex, vibration, and sharp/dull testing should produce active movement in innervated muscles. The Moro test (dropping the child backward suddenly in a controlled fashion) is occasionally seen but can cause additional tension on the shoulder and should generally be avoided. If movement does not return after 1 to 2 weeks, EMGs are often used to determine the extent of injury.

20. Can physical therapy to reduce spasticity improve function in children with cerebral palsy?
No scientific evidence indicates that physical therapy can reduce spasticity over the long term. Therapy techniques, such as global warmth, deep pressure, and prolonged stretch, have been shown to be effective in the short term; however, significant spasticity often needs to be addressed to improve functional independence. Generally, underneath the stiffness caused by the spasticity is a weak muscle. Evidence has shown that therapy to strengthen spastic muscles has a positive effect on strength and does not negatively influence spasticity. Typical growth and development can also cause changes that shorten spastic muscles and can cause loss of function. This can be addressed by physical therapy.

21. What are some methods of addressing spasticity medically?
Of the various medical treatments for spasticity reduction, the easiest to use is an oral agent such as diazepam (Valium) or oral baclofen. The dosage needed to cross the blood-brain barrier for effectiveness, however, may make the child sleepy. Baclofen delivered intrathecally from a battery-powered pump has helped some children who are severely limited by spasticity and tends to affect legs more than arms. This method allows a much lower dose to be administered, reducing the side effects with orally administered medications. For children with more localized issues, intramuscular injection of botulinum toxin type A (Botox) prevents the presynaptic release of acetylcholine at the nerve-muscle junction. The effects of Botox are short lived, between 2 and 6 months, but allow for strengthening of antagonists to reduce the influence of the spastic muscle on the child’s movement, allowing for improved function.

22. What is Gowers’ sign or maneuver?
Children with significant proximal hip/pelvic girdle weakness and lack of core strength use Gowers’ maneuver to stand up from the floor. The child rolls prone, gets onto the hands and knees, extends the knees and uses the hands to “walk up” the legs until the erect position is achieved. Gowers’ maneuver is not normal. Suspect a muscular dystrophy (most commonly Duchenne’s disease in males) and refer the child to the appropriate physician immediately. Another classic sign of Duchenne’s is the pseudohypertrophy of the calves.

23. Define osteochondritis dissecans (OCD).
OCD is a necrotic bone lesion with no known cause that may affect subchondral bone and adjacent articular cartilage. It is seen most commonly in the knee (in the intercondylar region of the medial femoral condyle). The ankle and elbow are other areas that may be affected. Lesions are staged 1 through 4, with stage 1 being a small area of compression and stage 4 having a displaced loose body. Generally boys are more affected than girls, but the gap is decreasing, as is the age of onset, because of an increase in participation in impact activities and intense sports engagement by both genders. Adult onset is often thought to be an unresolved childhood form but can appear as a new lesion.

24. What tests are useful for the diagnosis of OCD?
The Wilson test may be useful to diagnose OCD of the knee. With the knee flexed to 90 degrees, the tibia is rotated medially. The knee is extended passively and medial tibial rotation is maintained. Pain is detected at about 30 degrees of knee flexion and relieved by lateral tibial rotation. However, there are no studies on the reliability or validity of this test, and it is often negative on an existing lesion. X-rays are also not noted to be useful because of nonspecific results. MRI and MRA tend to have better predictive value, and generally a combination of evaluative techniques is used.
25. **How is OCD treated?**

Activity restriction for 6 weeks to 6 months with nonweight-bearing for 6 weeks is common. Children with open epiphyseal plates tend to respond well to 2 to 3 months of casting, immobilization, or unloader bracing. Isometric exercises are indicated during casting, progressing to active exercise to regain full ROM when the cast is removed, and finally to resisted strength training and return to full activity. If a loose fragment is present or if the subchondral bone is involved, surgery (usually arthroscopic) is indicated. The medical literature tends to prefer surgical intervention, especially in symptomatic patients with unstable lesions, and often it is recommended with stable lesions as well, even in cases of skeletal immaturity. A recent review of the literature was inconclusive regarding the role of conservative treatment in cases with stable lesions.

26. **What is Osgood-Schlatter disease?**

Osgood-Schlatter disease involves enlargement and microfractures of the apophysis of the tibial tubercle (where the quadriceps inserts) and is commonly seen in young, highly active adolescent males who are going through a rapid growth spurt. Males are typically affected from ages 13 to 14, whereas girls more often have symptoms from the age of 11 or 12. The tibial tubercle is usually prominent and tender. The pain is worsened by squatting, jumping, or kneeling and often appears in sports that highlight or emphasize these activities. Given the increase in female participation in similar sports, the ratio of boys to girls is decreasing.

27. **How is Osgood-Schlatter disease treated?**

Treatment is directed at relief of symptoms with heat or ice massage, changes in activity, use of knee pads, and administration of antiinflammatory medication. The condition usually resolves once the tibial tubercle apophysis fuses. Although the problem is being treated, flexibility and isometric strengthening exercises for the quadriceps and hamstring muscles may help. Sometimes a separate ossicle (small bone) develops under the patellar tendon and may need to be removed surgically. Splinting is rarely indicated but has been used in situations where the condition does not resolve. Generally, corticosteroid use is contraindicated and surgery should be avoided in cases of skeletal immaturity.

28. **What is Legg-Calvé-Perthes (LCP) disease?**

LCP disease is idiopathic avascular necrosis (probably episodic) of the femoral head. It is seen most often in children aged 4 to 12 and affects boys more often than girls (4:1). The disease is bilateral in approximately 12% of cases. The hip progresses from synovitis to an avascular stage, to fragmentation, to reossification, and finally heals within approximately 18 to 24 months following reossification. The Stulberg classification system has been used to discuss resulting deformities of the femoral head and neck, with corresponding prognoses. Levels I and II tend to resolve well, level II has additional onset of hip symptoms in adulthood, and levels IV and V will often show additional onset of symptoms in their twenties.

29. **How is LCP disease treated?**

Treatment usually consists of maintaining or regaining hip ROM, especially abduction, to keep the deformable involved segment of the femoral head contained within the acetabulum. Generally, surgery is avoided in children under age 8, as they are still investigating the benefits of surgery for this age group. Unweighting using crutches, limiting impact activities, and use of antiinflammatory medications are typically used. Bracing in an abducted and internally rotated position and osteotomy are other options. However, bracing has not been shown to be effective, and some studies show that children over the age of 8 at onset with more significant presentations do better with surgical intervention. After age 11, conventional surgical interventions do not appear to be as effective. The International Perthes Study Group is currently investigating various factors involved in the treatment of LCP disease, such as surgical versus nonsurgical treatment in children under age 8, effectiveness of weight-bearing versus nonweight-bearing in children 8 to 11, and various surgical interventions for children over the age of 11.

30. **What type of individual is most likely to suffer from a slipped capital femoral epiphysis (SCFE)?**

Obese adolescent males, ages 10 to 16, are most likely to have a “slip” or displacement of the capital (ie, head or proximal) femoral epiphysis. Incidence in the United States is 10.8 per 100,000, versus 2 per 100,000 globally, and may be bilateral in as many as 60% of cases, although severity is not always symmetric. They present with limping and pain in the distal thigh or knee. Because of this symptom, the diagnosis is often incorrect as a result of isolated evaluation of the knee. The condition is more prevalent in cases of obesity but also has a link to race, with higher incidence in African American males.
Slips can be acute or chronic, and patients have limited hip ROM, especially internal rotation. Patients with chronic slips may show shortening of the involved leg. Gait can be waddling in nature with a laterally rotated leg.

31. Describe the treatment for SCFE.
A stable SCFE is generally treated with a single screw pinning, and an unstable SCFE will use reduction, decompression, and internal fixation for successful resolution. Postoperative physical therapy involves regaining hip ROM, strengthening the lower extremity, especially the hip abductors, and protected partial weight-bearing with crutches. Partial weight-bearing is suggested (even if minimal) because non-weight-bearing requires the use of hip muscles to maintain the leg in the air and puts more stress on the hip than resting the foot on the floor. Chondrolysis and avascular necrosis (AVN) are potential late complications.

32. What conditions can affect the young baseball pitcher?
Repetitive stress may cause epiphysiolysis at the proximal humerus (little league shoulder) or stress the medial epicondyle apophysis (little league elbow). Studies have emerged showing that young pitchers who throw more than 100 innings in a season are at higher risk of shoulder and elbow injuries, although types of pitches do not seem to correlate with increased injury. The link to mechanics, including trunk rotation and other factors, is also emerging in the literature, and education programs for coaches are increasing in popularity as the prevalence of club sports has skyrocketed in the past 10 years. Thus the recommendations from American Sports Medicine Institute (ASMI) for preventing injuries in youth baseball pitchers are:

- Watch and respond to signs of fatigue (such as decreased ball velocity, decreased accuracy, upright trunk during pitching, dropped elbow during pitching, or increased time between pitches). If a youth pitcher complains of fatigue or looks fatigued, let him rest from pitching and other throwing.
- No overhead throwing of any kind for at least 2 to 3 months per year (4 months is preferred).
- No competitive baseball pitching for at least 4 months per year.
- Do not pitch more than 100 innings in games in any calendar year.
- Follow limits for pitch counts and days of rest.

33. What is a pectus excavatum (funnel chest) indicative of in a child?
Pectus excavatum is the most common chest wall deformity in children, occurring in 1 of every 400 newborns with male predominance (male-to-female ratio of 3:1). This is a depression of the sternum, and its etiology is generally unknown, although it is often seen in children with cardiopulmonary issues. In these children, depression of the sternum is generally related to poor muscle tone and respiratory insufficiency. Overuse of the diaphragm without support from the lower abdomen generates increased negative pressure in the chest cavity; the flexible joint in this complex is the sternocostal area, which gets pulled in. Abdominal support providing compression in the lower trunk to improve the effectiveness of the diaphragm could help to decrease the pectus. Strengthening of core muscles, including the rectus and obliques, provides the same effect without the need for external support. In cases of severe excavatum, surgery may be indicated to reduce compression on the chest cavity, and the Nuss procedure is one procedure used to correct the sternal position.

34. What is nursemaid’s elbow?
Also referred to as pulled elbow, temper tantrum elbow, or supermarket elbow, nursemaid’s elbow is subluxation of the radial head from the annular ligament. The mechanism of injury is usually a traction force on the arm, often seen when children are swung by the hands or an arm is jerked rapidly. Radiographs showing displacement of 3 mm or more from the capitellum suggest subluxation. Reduction is achieved with supination followed by elbow flexion. Recurrence rates vary from 5% to 39%, depending on the amount of instability at the annular ligament, as this is occasionally torn.

35. How do growth plate injuries in children occur?
Children who have not reached skeletal maturity are at risk for several types of fractures, including avulsions and green stick fractures, secondary to imbalance of muscles from growth or overtraining, or heavy impact use of joints. There is also a growing incidence of growth plate injury following ACL repair in the youth with skeletal immaturity. These findings make it critical to educate parents, athletes, and coaches regarding flexibility, balanced use of muscle groups, and risks of overtraining. It also encourages a critical decision-making process for using surgical correction of ACL pathology in the young athlete.
36. What is the occurrence of scoliosis in youth?
Scoliosis, or a lateral curvature of the spine of more than 10 degrees, has various causes that are present at different ages. Infantile scoliosis of idiopathic cause presents in children from birth to 3 years old. Current prevalence literature discusses a rate of 2% in the adolescent population, where rapid growth and muscle imbalances can be a cause. Neuromuscular scoliosis can present in cases where asymmetric tone is present. Treatment varies between idiopathic causes and neuromuscular causes. In children with idiopathic scoliosis with less than a 25-degree curve, aggressive exercise to address muscle imbalances can help to decrease curvature. Between 25 degrees and 40 degrees, bracing is recommended, but controversy exists over the use of rigid bracing versus dynamic bracing. Greater than 40 degrees, candidates are generally referred for surgical correction. In cases of neurogenic scoliosis, surgical correction may be indicated, but the underlying neurologic cause needs to be addressed as well. Otherwise, pseudoarthroses above or below the surgical rods may develop.

37. What is Sever’s disease?
Also known as calcaneal apophysitis, Sever’s disease can appear in children between the ages of 8 to 15 and is characterized by pain in the posterior and inferior region of the heel. It generally appears in children who are either overweight or very active. The literature is inconclusive regarding effectiveness of treatment, but strategies vary from relative rest, to heel cups, to over-the-counter insert orthotics and taping. Shoe changes may be necessary to control this condition. Stretching of the lower limbs (all muscles groups—easy on gastroc and soleus) can help to unload the insertion of the gastroc.

38. What is Blount’s disease (tibia varum)?
Blount’s disease is diagnosed as early onset or late onset, depending on age of presentation (before or after the age of 4). It is generally diagnosed in children who have had excessive early weight-bearing, are obese, or have metabolic disorders. Some studies indicate a higher incidence in children of African American descent or in females; there is a difference in early versus late-onset presentation. Late-onset groups have more of a distal femoral component, and early-onset groups show more proximal tibial varus and internal rotation. Treatment generally involves observation with alignment correction, if needed, bracing, if needed, and surgical intervention as a last resort.

39. What is metatarsus adductus?
Metatarsus adductus is one of the most common foot abnormalities in young children, with an incidence of 1 to 2 per 1000, with a coincidence of hip dysplasia in 10% of children. Generally this is a flexible deformity that resolves but needs to be monitored as persistent presentation can cause concerns with regards to gait. There is some evidence of benefit to stretching, taping, and shoe modification depending on the cause and presentation.

40. What is flexible pes planus?
Pes planus presents in approximately 14% of the population and is usually flexible. In a nonweight-bearing position the foot presents as if it has a normal arch. However, in a weight-bearing position the foot appears flat, and if the child is asked to rise up on his or her toes, an arch is noted. Parents and grandparents worry that the child has flat feet and what to get rigid shoes. Generally, a normal arch will develop later in childhood, and asymptomatic presentations do not need treatment. If symptoms are present (ie, pain, difficulty with gait and balance) consider an arch support or supportive shoe to decrease the medial collapse. However, going above the ankle changes gait mechanics and should be avoided if possible.

41. What are two common causes of rigid pes planus?

- **Vertical talus** (convex pes valgus)—a rare condition for which the exact cause is unknown; however, it is commonly associated with neuromuscular disease in children (usually with atypical tone or connective tissue disorders). It presents as a rocker bottom foot, and nonsurgical interventions such as casting, bracing, stretching, and exercise are viable options. However, most cases require surgical correction of the deformity.

- **Tarsal coalition** (peroneal spastic flatfoot)—very rare and is an atypical fusion of the calcaneus to either the navicular or the talus. Although tarsal coalition is often present at birth, children typically do not show signs of the disorder until early adolescence. The foot may become stiff and painful, and in some cases the individual may report recurrent episodes of ankle sprains. If nonsurgical approaches such as rest, orthotics, casts, and injections do not relieve the patient’s symptoms, then surgery is required to release the fusion.
42. Are orthotics useful for the correction of foot/gait deviations in children?
As children learn to walk, their foot position changes as a result of movement exploration that encourages strengthening in the muscles of the foot. For example, standing at furniture and rotating causes a supination moment in the same side foot, helping to strengthen the muscles of the medial arch. In general, putting shoes on children learning to walk alters gait mechanics and shifts the use of muscles from the foot to the hip and knee, increasing stance phase time. Therefore a general recommendation is to allow children learning to walk to do so without shoes. Still, some children continue to demonstrate excessive pronation, leading to external rotation of the feet to increase the base of support. For these children, continuing to walk with feet in this position could lengthen the medial structures, causing further damage. There is support for orthotics, using the minimal amount needed to achieve correction, in these cases. Too much support will alter mechanics, but minimal support may allow correction with minimal changes and prevent further damage.

43. What are the considerations for prosthetic use in children with limb deformities or amputation?
Historically, children with lower extremity limb deficiencies were provided with prosthetics once they were of age to be up and walking and, generally, were given a locked knee prosthetic until age 4 to 5. However, current trends encourage early use of a prosthetic for the lower extremity with a functional knee, even during crawling and pulling to stand phases. Less compensatory strategies are noted in these children, leading to fewer deviations as mobility improves. Initiation of upper extremity prosthetic use will vary depending on the function of the residual limb. Children are encouraged to use both hands as early as 6 months old, and delays in development, cognition, and exploration can occur. If minimal function is available in the residual upper extremity, prosthetic use should begin early, with modifications as needed.

44. What is arthrogryposis multiplex congenita (AMC)?
AMC is a disorder of multiple nonprogressive contractures that are present a birth, in specific upper and lower extremity patterns. This disorder occurs in 1 of every 3000 to 6000 live births, and there is evidence of anterior horn cell involvement. There is no effect on cognition, but the contractures in multiple joints are significant and can sway function dramatically. Therefore early multidisciplinary intervention is indicated to reduce contracture, improve joint integrity, address mobility, and provide adaptive equipment as needed. This support will help to improve the participation of the child in daily routines and community activities.

45. What is osteogenesis imperfecta (OI)?
OI is a genetic condition that affects bone and connective tissue, resulting in extreme fragility of bones in 1 of every 10,000 individuals. There are multiple types, related to number, location, and type of fracture, which can occur with or without impact. The use of bisphosphonates over the past decade or so has improved bone mass in these children, resulting in reduced fractures, but reduction in activity can affect muscle strength, aerobic conditioning, and general participation. Therapists need to balance therapy to address these concerns with the risk of fracture and the consequences.

Bibliography


CHAPTER 25 QUESTIONS

1. Which of the following interventions will BEST benefit a child born with a vertical talus and rigid pes planus?
   a. Corrective shoe wear
   b. Joint mobilizations
   c. Corrective surgery
   d. Orthotics
2. You are evaluating a 5-year-old for a right lower extremity concern that requires her to remain nonweight-bearing. You notice that she is fairly impulsive and easily distracted. Upon evaluation, you notice some mild coordination issues. What would be the appropriate assistive device to request for this child?
   a. Lofstrand crutches
   b. Axillary crutches
   c. Walker
   d. Wheelchair

3. You receive a referral for a 3-month-old infant and notice that he has a head tilt to the left with some right rotation. His head shape is symmetrical, but he has some muscle tightness on the left side of his neck and difficulty holding his head in midline. You feel this child has:
   a. Typical torticollis and would benefit from treatment
   b. Atypical torticollis and would benefit from radiological evaluation
   c. Plagiocephaly and would benefit from referral for orthosis
   d. Hypotonia and would benefit from referral to neurology

4. You are evaluating an 8-year-old boy with some clumsiness and poor trunk control. During evaluation, you note that he stands up by walking his hands up his legs. Your first thought regarding diagnosis should be:
   a. Cerebral palsy
   b. Osgood-Schlatter disease
   c. Slipped capita femoral epiphysis
   d. Duchenne's muscular dystrophy

5. All of the following affect boys more than girls except:
   a. Osteochondritis dissecans
   b. Legg-Calvé-Perthes
   c. Slipped capita femoral epiphysis
   d. Nursemaid's elbow
1. What musculoskeletal changes occur as a result of pregnancy?

Increased levels of relaxin, progesterone, and estrogen can cause changes in connective tissue, increasing the risk of some types of soft tissue injuries during pregnancy. Increasing levels of the hormone relaxin, which allows for the softening of the pelvic ligaments needed for passage of the fetus during delivery, affect ligaments globally. The transverse and anteroposterior diameter of the chest also increases secondary to pregnancy-related softening of ligaments. Estrogen and progesterone levels increase, contributing to increasing joint laxity. Joint laxity increases during the tenth week of pregnancy and can continue until 4 to 12 weeks postpartum. Increased weight gain in the pelvis and resulting enlargement of the abdominal girth causes a shift in the center of gravity, increasing risk for loss of balance. Increased lumbar lordosis results in iliolumbar ligament shortening, causing shortening of the psoas and gluteal and hamstring weakness. Increased total body water and plasma levels can affect the nerve connective tissue interface by increasing edema, which can result in nerve entrapment secondary to pregnancy-related changes. The enlarged uterus can exert pressure on the diaphragm, causing shortness of breath.

2. What cardiovascular changes occur as a result of pregnancy?

Total body water increases by 6.5 to 8.5 L, which includes a 1200- to 1300-ml change in plasma volume. This represents a 40% to 50% increase over nonpregnant values, with the peak increase occurring at 32 weeks. Red blood cell mass increases by 300 to 400 ml. This relatively greater change in plasma volume versus red blood cell mass results in a dilutional anemia. Cardiac output increases by 27% to 50%, peaking late in the second trimester; stroke volume increases by 32%, peaking at 24 weeks. Heart rate increases early in pregnancy, peaking at a 15 to 20 beats per minute increase at term. Systemic vascular resistance decreases throughout pregnancy. Although oxygen delivery increases, this is offset by the physiologic anemia of pregnancy resulting from the increased plasma to red blood cell ratios as previously described.

3. How long after delivery do these values remain changed?

Cardiac output returns to prepregnancy levels 10 to 14 days after delivery, and heart rate returns to prepregnancy levels at 6 to 12 weeks after delivery. Stroke volume returns to prelabor values after 24 hours but remains elevated compared with preconception values even after 1 year post delivery. Blood volume decreases rapidly (10%–17%) in the first hour after delivery and continues to decrease for the next 5 days.

4. What respiratory changes occur during pregnancy?

Respiratory rate, inspiratory capacity, and vital capacity remain unchanged. Although tidal volume increases progressively throughout pregnancy, expiratory reserve volume and functional residual capacity are decreased. Because respiratory rate remains unchanged, the increased oxygen demand experienced during pregnancy is met through increased diaphragmatic excursion during respiration.

5. What physical therapy techniques and positions are contraindicated in pregnant clients?

Any technique that involves abdominal compression in mid- to late pregnancy, maintaining the supine position after the first trimester of pregnancy for longer than 4 minutes, any position that raises the buttocks higher than the chest, positions that strain the pelvic floor and abdominal muscles or involve vigorous stretching of hip adductors, or rapid uncontrolled bouncing or swinging movements should all be avoided.
6. What modalities can be applied and what contraindications and precautions must be considered?
Most sources state that ultrasound and electrical stimulation are contraindicated regardless of location during pregnancy, with an additional contraindication for electrical stimulation over acupuncture points. An exception to this is the use of sensory level TENS during labor to relieve pain. Low-level laser therapy is contraindicated for use over the low back, pelvis, and abdomen during pregnancy, and pregnancy should be considered as a precaution for its use for other areas of the body. Because the direct effects on the fetus are unknown, extreme caution should be exercised because of possible detrimental effects including fetal malformation, fetal growth compromise, and preterm labor. Superficial heat may be used over small areas with contraindications and precautions similar to those of a nonpregnant individual; however, use over large areas or that which may risk raising core temperature should be avoided and is considered a contraindication. This includes application of superficial heat over large areas of the trunk or total body immersion in warm or hot water such as during hydrotherapy or in a Jacuzzi. Cryotherapy during pregnancy carries the same precautions as for the nonpregnant individual.

7. What guidelines should be followed for exercise during pregnancy?
An individual’s overall health, obstetric, and medical risks should be considered when prescribing exercise during pregnancy. Prior level of fitness is also considered; active women may remain active, but previously sedentary women must be evaluated more extensively. Strenuous exercise should be avoided; heart rate should not exceed 140 to 150 beats per minute, or 60% to 70% of maximum heart rate. The supine position for longer than 4 minutes, because of decreased venous return, should be avoided after the first trimester. Contact sports resulting in potential impact to the fetus should also be avoided. Scuba diving and exercise at high altitudes, because of risks to fetal circulation, should be also avoided. Stretches should be controlled, and certain abdominal exercises should be avoided if diastasis recti (separation of the connective tissue midline in the front abdominal wall) develops.

8. Can a woman participate in aerobic exercise during pregnancy?
In addition to the exercise guidelines previously mentioned, the American College of Obstetricians and Gynecologists (ACOG) lists specific guidelines for aerobic exercise. Absolute contraindications include hemodynamically significant heart disease, restrictive lung disease, incompetent cervix/cerclage, multiple gestation at risk for premature labor, persistent second or third trimester bleeding, placenta previa after 26 weeks of gestation, premature labor during the current pregnancy, ruptured membranes, and preeclampsia or pregnancy-induced hypertension. Relative precautions listed by the ACOG include severe anemia, unevulated maternal cardiac arrhythmia, chronic bronchitis, poorly controlled type 1 diabetes, extreme morbid obesity, extremely underweight (BMI <12), a history of extremely sedentary lifestyle, intrauterine growth restriction in current pregnancy, poorly controlled hypertension, orthopedic limitations, poorly controlled seizure disorder, poorly controlled hyperthyroidism, and heavy smoking.

9. What are some characteristics of pregnancy-related back pain?
Mechanical and hormonal factors contribute to back pain during pregnancy. Pregnancy-related back pain can be further classified into pelvic girdle pain (PGP) and low back pain (LBP). Pelvic girdle pain can begin as early as 12 weeks of gestation when hormonal levels are high and is described as occurring between the iliac crest and gluteal fold with radiation to the distal end of the buttock. PGP is four times as prevalent as lumbar pain. Lumbar pain typically occurs over the lumbar spine and sacrum and can radiate distally as far as the foot. Lumbar pain is more prevalent in the last trimester with the added weight gain and into the postpartum period. PGP provocation testing and neural tension testing assist in differentiating PGP and LBP.

10. What is diastasis rectus abdominis (DRA), and what causes it?
DRA is defined as a separation of the two muscle bellies of the rectus abdominis at the linea alba. It appears as an increase in the width of the linea alba, or the interrecti distance (IRD). It is caused by the separation of the connective tissues brought upon by the mechanical stress sustained as a result of the enlarging uterus, which in combination with increased levels of relaxin, progesterone, and estrogen can result in a splitting of the connective tissue joining the two rectus abdominis muscle bellies. Other factors, such as age, genetic aspects, and a previous occurrence of significant weight loss can also predispose an individual to developing a diastasis rectus as the rectus muscles are stretched.
11. How is DRA diagnosed or measured?
DRA is typically measured by palpation. With the client in a hook-lying position, the clinician places the fingertips perpendicular to the opposing muscle bellies of the rectus abdominis. The size of the DRA is described by indicating how many fingers fit between the two muscle bellies above, below, and at the umbilicus while the individual performs a partial curl-up with the head and scapulae lifted off the supporting surface. Because this method has been found to be neither reliable nor valid, the use of calipers or ultrasound imaging is recommended to obtain more reliable and valid measurements.

12. Why is DRA significant, and what problems can it cause?
Abdominal pain, back pain, and a lack of ability to complete tasks requiring abdominal activation has been noted in individuals with known DRA. DRA has also been suggested to be related to stress urinary incontinence, fecal incontinence, pelvic organ prolapse, and pelvic pain.

13. What can be done to treat DRA?
A “drawing-in” of the umbilicus activates the transverse abdominis. This “drawing-in” maneuver with quick and sustained pelvic floor contractions and performance of the “drawing-in” maneuver while performing various upper and lower extremity resistance exercises have also been shown to reduce DRA. Other techniques frequently utilized among therapists include myofascial work, manual approximation of the rectus abdominis muscle bellies while performing a partial curl-up, taping, joint mobilization of pelvic and lumbar structures, and use of an abdominal brace during activities pre- and postpartum.

14. What causes symphyseal pain, and what signs and symptoms may be noted?
The pubic symphysis is innervated by the pudendal and genitofemoral nerves and branches off of the iliohypogastric and ilioinguinal nerves. Widening of the interpubic gap and increased movement are primarily as a result of the increased estrogen and relaxin production experienced during pregnancy. Movement of bone ends stimulates nociceptive mechanoreceptors, resulting in pain. Typical symptoms include pubic pain, which may radiate to the anterior or medial region of one or both upper thighs; sacroiliac or low back pain may also be experienced. Pain may be noted during weight-bearing activities, especially those involving lifting one extremity or single leg stance. Walking, climbing stairs, and rolling over in bed may exacerbate symptoms. Palpation of the pubis that remains painful after 5 seconds is positive for pubic symphysis pain, whereas pain that last only when directly palpated is considered a tender pubic symphysis. A clicking or grinding in the joint, along with a waddling gait, may also be present.

15. What is the most effective treatment for pubic symphysis pain?
Instruction in activity modification, particularly avoidance or limitation of single leg weight bearing or rolling over in bed with hips abducted, can effectively address pregnancy-related or postpartum symphysis pain. Activation of the transverse abdominis and pelvic floor before activities of daily living can also minimize symptoms. Lumbar, hip, and lower extremity stretching, stabilization exercises, including isometric exercises for the transverse abdominis (by addressing core stabilization), pelvic floor, latissimus dorsi, gluteus maximus, and hip adductors can also address symptoms. Performance of various upper and lower extremity resistance exercises while maintaining pelvic floor and transverse abdominis contraction has been noted to improve pain and functional status.

16. What is meralgia paresthetica?
Meralgia paresthetica refers to a sensory mononeuropathy of the lateral femoral cutaneous nerve as it exits the pelvis and enters the lower extremity. Entrapment likely occurs as the nerve passes around the anterior superior iliac spine or through the inguinal ligament. This condition often occurs during pregnancy as a result of mechanical changes related to increased intraabdominal pressure; the occurrence of meralgia paresthetica can also be related to increased lumbar lordosis or can occur after delivery from prolonged positioning in the lithotomy position during delivery. Symptoms typically include anesthesia, paresthesia, or allodynia on the anterolateral thigh that may be exacerbated by prolonged standing but may also be aggravated by sitting. Deep palpation along the inguinal ligament may reproduce these symptoms. Unique to this condition is an absence of motor deficits.

17. How is meralgia paresthetica treated?
Nerve sliding and flossing and soft tissue techniques to release and relax medial and anterior thigh soft tissue are utilized to address factors contributing to lateral femoral cutaneous nerve entrapment. Treatment of pelvic and sacroiliac (SI) dysfunction, abdominal stabilization exercises, and transverse abdominus training can also decrease pain and improve function. Modification of activities of daily living,
including avoiding exacerbating activities such as prolonged standing or restrictive clothing, or belts that may contribute to symptoms, is also warranted. Abdominal support that lifts the gravid abdomen off the inguinal area can often relieve symptoms.

18. Describe the structure and function of the pelvic floor.
The pelvic floor refers to the pelvic diaphragm, which arises from the posterior superior pubic rami, inner ischial spines, and obturator fascia. The fibers of the pelvic diaphragm insert around the vaginal and rectal openings at the perineal body. The diaphragm is composed of the coccygeus and levator ani muscles. The pelvic floor creates a sling support for the internal organs and openings for the urethra, vagina, and anus.

19. What causes pelvic floor dysfunction?
Pelvic floor dysfunction can be related to any trauma, surgery, or weakness associated with the pelvis. For example, total hip replacement, episiotomy, vaginal delivery, or back surgery could cause pelvic floor dysfunction. When the normal spine or pelvic mechanics are interrupted or when trauma has occurred to the pelvic floor, the patient can be susceptible to pelvic floor pain or dysfunction.

20. How is pelvic floor muscle function assessed?
After obtaining a referral and consent and determining what state practice allows, physical therapists can perform an internal pelvic floor examination to assess pelvic floor muscle function. Resting muscle tone, muscle strength, and muscle endurance and coordination can be assessed; various scales are available to assess these components. The modified Oxford scale is a manual muscle grading scale that measures strength on a scale from 0 to 5 with 0—no response, 1—flicker, 2—weak contraction, 3—moderate contraction with some degree of lift, 4—good contraction against some resistance, and 5—normal muscle contraction, strong squeeze, and lift. The Brink scale assesses muscle strength by considering duration of muscle contraction, the pressure generated upon the examiner’s digit by the surrounding pelvic floor muscles, and the vertical displacement of the examiner’s finger when the subject performs a pelvic floor contraction. Each component is rated on a four-point scale, with total possible scores ranging from 3 to 12. The PERFECT (P=Power/pressure, E=Endurance, R=Repetitions, F=Fast, E=Every, C=Contraction, and T=Timed) scheme utilizes the modified Oxford scale to assess power but also measures the amount of time the pelvic floor contraction can be maintained before fatigue (E) and the number of times this degree of contraction can be generated when a 4-second rest is provided between contractions (R) and then after a 1-minute rest, the number of 1-second contractions that can be performed (F). The “ECT” in the PERFECT acronym reminds the tester to time and record the events noted herein when utilizing this scheme.

21. Define pelvic organ prolapse.
The word prolapse is derived from the Latin word *prolapsus* ("to slip" or "fall") and refers to the pelvic organs descending into or through the vagina.

22. How is physical therapy involved in treating pelvic organ prolapse?
Physical therapy has not been proven to improve prolapse because this is an anatomic defect; however, it can reduce symptoms and prevent progression. Physical therapy treatment involves:

- Therapeutic exercises for the pelvic floor for strength and conditioning; can be performed against gravity or with gravity eliminated, depending on the severity of the prolapse and the amount of muscle atrophy
- Abdominal retraining and conditioning for strengthening and stabilization and for decreasing intraabdominal pressure with activity
- Neuromuscular reeducation/modality intervention as needed, depending on the amount of decreased proprioception evident
- Pessary use and surgical correction

23. What patient population is most at risk for pelvic organ prolapse?
Women who have had vaginal deliveries or a history of chronic constipation or obesity are often implicated as being in the highest risk category for developing pelvic organ prolapse. However, retrospective studies have shown that nulliparous women involved in high-impact sports and repetitive heavy lifting occupations also have a high incidence of pelvic organ prolapse. Abdominal training for women should be monitored so that technique does not increase intraabdominal pressure, and women should be instructed to activate a pelvic floor contraction to counteract pressure changes at the level of the perineum as well.
24. Describe the five types of incontinence.

1. Stress—involuntary loss of urine during physical exertion (ie, coughing, lifting) in the absence of detrusor contractions
2. Urge—loss of urine with urgency; with active detrusor contractions
3. Mixed—both stress and urge incontinence
4. Overflow—the bladder overfills; an outlet obstruction or underactive detrusor may be present
5. Reflex—present with neurologic lesions; urine leaks without warning

25. Describe physical therapy treatment for incontinence.

Treatment for genuine stress incontinence includes pelvic floor (Kegel) exercise instruction, resistive pelvic floor exercise with vaginal-weighted cones, dietary counseling to avoid diuretics and bladder irritating substances (ie, caffeine), biofeedback via air pressure or surface electromyography, and electrical stimulation with in-home or office-unit devices.

26. Is urinary incontinence common in the nulliparous female athlete?

Research has shown that women/girls involved in high-impact sports such as basketball, cheerleading, gymnastics, tennis, and field hockey had occurrences of urinary stress incontinence with sporting activity.

27. What are the expected outcomes of physical therapy for incontinence?

Kegel conducted several studies to assess the efficacy of strengthening pelvic floor musculature to control incontinence. He showed improvement in three fourths of the women in study samples. In the Kegel or pelvic floor exercise, cortical impulses contract fast-twitch, striated periurethral sphincter muscles via the pudendal nerve. It is theorized that these muscle fibers are able to hypertrophy with prolonged training. No optimal number of repetitions has been standardized, but several protocols have been proposed for strengthening, endurance, and functional retraining.

Although early studies suggested isolation of the pubococcygeus muscle was the optimal way to increase its strength, recent studies have reported that overflow contractions through lower extremity and abdominal muscle contractions may enhance pelvic floor muscle training.

28. What interventions can physical therapists use to address pelvic pain?

Manual therapy, use of vaginal dilators, pelvic floor surface electromyography (sEMG) biofeedback, pelvic floor exercise, relaxation techniques, instruction for dietary changes, hip stretching, abdominal and pelvic floor strengthening, and electrical stimulation to the pelvic floor may be used by physical therapists to address pelvic pain. Patient education including sexual activity, other activities of daily living, and instruction in a home exercise program, are also prescribed as part of the physical therapy treatment plan.

29. Define delayed menarche and why this is relevant to physical therapy.

Delayed menarche is lack of menstruation by the age of 16. Studies have shown that there is a strong association between delayed menarche and increased risk of scoliosis and stress fractures with girls involved in high-impact sports (runners, ballet dancers).

30. Define oligomenorrhea and amenorrhea.

Oligomenorrhea is scanty menstruation that can occur with a sudden weight loss of 10 lb with no regard to the woman’s original weight. Amenorrhea is an abnormal cessation of the menses for 3 or more months after menarche has already started.

31. What are some of the causes of oligomenorrhea and amenorrhea?

- Strong emotional disturbance
- Exercise induced—increased endorphins inhibiting hypothalamic function
- Pathologic secondary to disease process
- Dietary—severe weight loss or gain. Typically menses cease when a young woman loses weight to the point at which she is about 85% of her ideal body weight for age and height. Women and girls with a history of anorexia nervosa and/or long-standing amenorrhea are hypoestrogenemic and at high risk for osteopenia/osteoporosis.

32. Define lymphedema.

Lymphedema is the chronic unilateral or bilateral swelling of extremities caused by obstruction, disease, or removal of the lymphatic vessels or nodes. Lymphedema may occur after breast surgery, which may involve removal of part of the breast (lumpectomy), one fourth of the breast (quadrectomy), or the whole breast (mastectomy).
33. Describe the treatment for lymphedema.  
Physical therapy involves a combination of range of motion exercises, compression, and various systems of manual lymphatic drainage massage. Compression is achieved with intermittent pneumatic pump, compression garments, or wrapping the extremity with bandages. Ultrasound and myofascial release of the chest wall also promote healing of scars and adhesions. Despite their many variations, all manual lymphatic drainage techniques focus on rate of movement, depth of pressure, area of the body treated, desired direction of lymph flow, and scar tissue.

34. What is the female athletic triad?  
The female athletic triad is defined by disordered eating, amenorrhea (defined as an absence of menstrual periods for 3 to 6 consecutive cycles), and osteoporosis. Females participating in endurance sports or those emphasizing esthetic components, particularly a thin frame, are most susceptible. Those with an already low body weight or that participate in athletic activities at an elite level are also at an increased risk. Inadequate nutrition caused by inadequate intake or excessive exercise is common. Although the presence of anorexia nervosa (inability to maintain healthy body weight, weight below 85% of ideal body weight) or bulimia (recurrent binge eating combined with inappropriate compensatory behaviors) had previously been considered as a prerequisite for a diagnosis of female athletic triad, eating disorders not otherwise specified (EDNOS) are now considered to be related to the female athletic triad. EDNOS are defined as subclinical anorexia nervosa or bulimia or when some but not all symptoms of anorexia nervosa or bulimia are present.

35. How can the female athletic triad be addressed?  
Correction of hormonal deficiencies through prescription of oral contraceptives was at one time the focus of treatment. However, resolving nutritional deficiencies, addressing any additional metabolic and hormonal deficiencies, identifying individuals at risk for low bone mineral density, and cognitive behavioral therapy to address other contributing factors are also components of treatment. Graduated return to exercise with supervision of a sound exercise program is suggested.

36. What specific nutritional deficiencies should be considered in cases of female athletic triad?  
Ensuring adequate calcium and vitamin D intake can address the detrimental effects amenorrhea and disordered eating can have on bone health. Young women experiencing normal menstrual cycles require 1000 mg of calcium daily and those with scanty (oligomenorrhea) or absent (amenorrhea) cycles require 1500 mg of calcium daily.

37. Define osteoporosis.  
Osteoporosis is a disease of the bones caused by a thinning of the bone matrix, which results in overall bone loss. Osteoporosis reduces the thickness and strength of the bones and makes them more susceptible to fracture. It is most common in postmenopausal women.

38. How can a female athlete develop osteoporosis?  
The female athlete is often under intense pressure to have a low body fat percentage to improve performance. The athlete may develop eating disorders to obtain low body fat and, as a result of decreased estrogen levels, then develop amenorrhea and the consequences of osteoporosis (as in postmenopausal women).

39. Describe the common causes of osteoporosis.  
Decreased weight bearing, as in decreased activity, reduces stress on the bones, which triggers calcium resorption and results in bone loss. Risk factors for primary osteoporosis include female sex, Caucasian or Asian descent, early menopause (before age 45), no history of pregnancy, low body weight, family history of osteoporosis, use of steroids and high dosages of thyroid hormones, smoking, excessive alcohol consumption, deficient intake of calcium as a child, and inactivity.

40. What methods are used for the diagnosis of osteoporosis?  
Osteoporosis is diagnosed through bone density screening methods: dual-energy x-ray absorptiometry (DXA), which is 90% to 99% accurate; peripheral dual-energy x-ray absorptiometry (pDXA), which is 90% to 99% accurate; single-energy x-ray absorptiometry (SXA), which is 98% to 99% accurate; quantitative ultrasound (QUS); quantitative computed tomography (QCT), which is 85% to 97% accurate; and peripheral quantitative computed tomography (pQCT). These methods measure bone mineral density (BMD) at different sites of the body. Choice of test depends on the anatomic sites available for the study, cost, and accessibility of the technology. All of these tests measure the bone absorption of radiation or
high-frequency sound waves, but each uses a different method of measuring energy absorbed by the tissue. The results are expressed as grams of calcium hydroxyapatite per square centimeter of bone cross section. Normal bone mass is defined by the World Health Organization (WHO) as a T-score above −1; low bone mass as a T-score between −1 and −2.5; and osteoporosis as a T-score at or below −2.5.

41. What are the three types of osteoporosis?
   1. Idiopathic osteoporosis
   2. Type I or postmenopausal osteoporosis (in women around ages 51 to 75; mostly caused by endocrine changes but also occurs in men)
   3. Type II or involutional osteoporosis (may be related to a decrease in vitamin D synthesis within the body; occurs in people over 70, although it begins in the third decade; female-to-male ratio = 2:1)

42. Are radiographs useful in the diagnosis of osteoporosis?
   Radiographs are not sufficient for proper diagnosis of osteoporosis. Bone loss of up to 40% can occur before the loss is visible on radiographs.

43. Why is exercise important in the treatment of osteoporosis?
   Weight-bearing exercise stimulates increased bone density and bone growth. Exercise must be maintained, or the positive results of exercise are lost with a return to baseline bone mass. Exercise also improves muscle strength, joint flexibility, function, endurance, balance, gait, and posture. Adults who engage in physical exercise have a faster reaction time than sedentary adults. Reaction time is important in response to loss of balance and prevention of falls. Often falls caused by poor balance result in fractures to the distal radius, vertebrae, or proximal femur. Hip fractures have a high mortality rate.

44. Which bones are most commonly affected?
   Type I (postmenopausal) osteoporosis involves excessive and disproportionate trabecular bone loss. It is associated with vertebrae and distal radius fractures. Compression fractures may occur in any vertebra but usually involve those in the lower thoracic and upper lumbar area.
   In type II osteoporosis, fractures occur in the upper femur and femoral neck. Type II osteoporosis also may result in multiple wedge-type vertebral fractures, which are not as painful as the crush-type vertebral fractures associated with type I.

45. What type exercise is ideal for individuals with osteoporosis?
   A variety of resistance exercises designed to increase load-bearing and muscle forces throughout the skeleton have been determined as beneficial to bone health. A program consisting of 8 to 12 repetitions of 6 to 10 exercises performed three times per week has been shown to improve bone health among osteoporotic subjects that target weight-bearing structures; other studies recommend performance of weight-bearing exercise three to five times per week for a minimum of 30 to 45 minutes per session. However, these exercises must be continued in order for gains to be maintained.

46. What other types of activities are effective for individuals with osteoporosis?
   Fast-paced walking and low-impact standing exercises, such as heel raises, designed to deliver load bearing through the spine and extremities are effective. Plyometric activities, such as jumping or throwing, also increase load bearing and muscle forces across the skeleton. Activities such as tandem walking, toe walking, heel walking, standing postural activities, and single leg-stance activities can also reduce fall risk by addressing balance and coordination deficits.

47. Why does the risk of heart attack and stroke begin to increase after menopause?
   Lower estrogen levels found in postmenopausal women have a negative effect on the cardiovascular system for two reasons: 1) estrogen acts to increase the levels of high-density lipoproteins (HDLs) and decrease the levels of low-density lipoproteins (LDLs), and 2) estrogen also helps in inhibiting the deposition of atherosclerotic plaques in the intima of arteries. Consequently, a sedentary lifestyle, poor diet, and poor physical fitness can lead to a higher risk of cardiovascular disease, especially coronary artery disease. Myocardial infarction is the leading cause of death among postmenopausal women.

48. What is postprostatectomy incontinence?
   Postprostatectomy incontinence can occur after surgical procedures involving the prostate as a result of potential damage to the bladder neck and surrounding structures. Improvements in surgical technique have resulted in a decreased incidence of urinary incontinence after prostatectomy. However,
detrusor weakness or instability, sphincter weakness, or decreased urethral closure pressure can also result in urinary incontinence after prostatectomy.

49. **What physical therapy interventions can address postprostatectomy incontinence?**

Pelvic floor exercise and patient education are both used in the treatment of postprostatectomy incontinence. In addition, instruction in and performance of pelvic floor strengthening exercises before prostatectomy have also been found to be more effective in decreasing incontinence episodes and urinary urgency compared with exercise initiated after surgery.

50. **What is male pelvic pain?**

Male pelvic pain, or chronic pelvic pain syndrome (CPPS), is characterized by persistent abdominal, pelvic, genital, or perineal pain. Pain may also be accompanied by bladder symptoms including dysuria, urinary frequency, incomplete voiding, sexual dysfunction, or pain after ejaculation. Male pelvic pain can be caused by acute perineal trauma, entrapment of the pudendal nerve, or prostatitis.

51. **What physical therapy interventions can address male pelvic pain?**

Manual therapy, especially of the obturator internus, can address myofascial restrictions. Biofeedback can also be used to facilitate voluntary relaxation of the pelvic floor. Ischemic pressure techniques and contraction and relaxation of the pelvic floor have also been described as techniques effective in the treatment of male pelvic pain. A home program of self-relaxation to increase alpha motor input is also recommended, along with instruction in proper bowel and bladder habits.

**Bibliography**


CHAPTER 26 QUESTIONS

1. Diastasis rectus abdominus is:
   a. A decrease in the width of the linea alba
   b. Measured during a pelvic floor contraction
   c. Most accurate when measured by palpation
   d. Treated with transverse abdominus activation

2. Which of the following is associated with meralgia paresthetica?
   a. Lower motor deficits, sensory changes in the anterolateral thigh
   b. Sensory changes in the medial thigh, symptoms may be reproduced after sitting
   c. Sensory changes in the anterolateral thigh, reduction of symptoms with prolonged standing
   d. Symptom reproduction along with deep palpation along the inguinal ligament, entrapment of the lateral femoral cutaneous nerve

3. Which of the following is an effective treatment for pubic symphysis pain?
   a. Hip adductor stretching
   b. Iliopsoas strengthening
   c. Single leg stance training
   d. Transverse abdominus activation
1. What is the functional movement screen (FMS)?

The FMS is used to establish standards for movement and identify painful movement patterns. It can be performed by health care and fitness professionals who have taken the certification course. It takes on average 10 to 12 minutes to complete. It is comprised of seven tests scored on a scale of 0 to 3. Its role is only to rank and rate movement patterns and not diagnose or measure isolated joint movement. If any pain is present with the FMS a referral to a medical professional is warranted.

- **Deep squat**: purpose is to look at coordinated extremity mobility and core stability with the hips and core functioning symmetrically.
- **Hurdle step**: purpose is to look at step and stride mechanics, while looking at stability in the stance limb. The hurdle step challenges bilateral mobility and stability of the hips, knees, and ankles. The test also challenges stability and control of the pelvis and core as it offers an opportunity to observe functional symmetry.
- **Inline lunge**: purpose is to test stability and dynamic control of the hips and core. The narrow base requires appropriate starting stability and continued dynamic control of the pelvis and core when in an asymmetrical hip position. This test also challenges hip, knee, ankle, and foot mobility and stability and simultaneously challenges the flexibility of multiarticular muscles such as the latissimus dorsi and the rectus femoris.
- **Shoulder mobility**: purpose is to assess scapulothoracic rhythm and glenohumeral and thoracic mobility. This pattern observes bilateral shoulder range of motion, combining extension, internal rotation and adduction in one shoulder, flexion, external rotation, and abduction of the other.
- **Straight leg raise**: purpose is to assess hip mobility and core stabilization. This pattern challenges the ability to dissociate the lower extremities while maintaining stability in the pelvis and core.
- **Push-up trunk stability**: purpose is to assess core stabilization when initiating movement. It is used as a basic observation of core stabilization and is not a test or measure of upper-body strength.
- **Rotary stability**: purpose is to assess global stability in multiple planes and neuromuscular coordination.

2. Are there any additional tests associated with the FMS?

Yes. The following screens are performed and simply given a positive or negative result for pain:

- The shoulder clearing test is performed by resting the hand on the opposite shoulder and raising the elbow toward the forehead. This is performed after the shoulder mobility test. It is a test to determine the presence of rotator cuff pathology.
- The flexion clearing test is performed by rocking back onto the heels, flexing the spine, hips, knees, and assessing for any pain. This is performed after the rotary stability test.
- The extension clearing test is performed by doing a prone press-up movement, extending the spine and hips and assessing for pain. This is performed at the end of the push-up trunk stability test.
- For each of the clearing tests, if pain is noted the associated FMS test is given a score of zero. Both the movement score and the painful additional test are noted for future reference.

3. When should you use the FMS?

The FMS is ideal to use before sport participation or starting a fitness routine. The FMS can also be used as a component of a return to sport assessment and programing. Identifying asymmetries and dysfunctional...
movement patterns, and addressing them before a fitness routine or sport participation, could limit injury or dysfunction.

4. What is the most important test in the FMS?
All of the tests in the FMS are important as they evaluate the entire motor system. It is crucial to examine each of the individual movements apart from the composite score and know what comprises the movements to identify limitations.
   How is the FMS scored?
   Score on a scale of 0 to 3:
   0 – Pain or unable to complete the test
   1 – Severe compensation, incomplete movement, or total loss of alignment
   2 – Compensation, poor form, or loss of alignment
   3 – Movement is functional and complete
   For bilateral tasks (five of the seven) both right and left sides are scored, and if different, the lower of the two scores is used for the composite score. Clearing tests are scored as positive or negative for pain.

5. How reliable is the FMS?
- Interclass Correlation Coefficient (ICC) for the FMS “composite score” have been reported as intrarater 0.74–0.95, interrater 0.38–0.99, and intersession 0.74–0.92.
- Minick et al. demonstrated excellent or substantial agreement when both novice and expert raters assessed healthy individuals.
- Gulgin and Hoogenboom found that total FMS scores were similar among the raters, and the interrater reliability for a majority of the individual tests had a strong agreement despite the various levels of experience of the raters scoring the FMS tests.
- Teyhan et al. found that among novice raters, the FMS composite score demonstrated moderate to good intrarater and interrater reliability, with acceptable levels of measurement error.
- Elias concluded that “the results of the test indicate that the FMS is a reliable screening tool when used by ‘untrained’ practitioners in determining faulty movement patterns and that clinical experience level does not affect the reliability, therefore it may be a useful tool in the screening of athletic populations.”
- Tests with the lowest reliability included the hurdle step, rotary stability, and in-line lunge.

6. Is the FMS able to predict injury?
The FMS was not designed to predict injury. There have been studies that have shown a correlation between lower FMS scores and injury risk (<14). For example, Kiesel found that professional football players with an FMS score of <14 were more likely to sustain an injury and players with a score >17 were less likely to sustain an injury. An average FMS score is considered to be between 14 and 16 out of 21. Chorba collected FMS scores on 38 Division 2 NCAA female athletes in the sports of soccer, basketball, and volleyball before the start of their season. Sixty-nine percent of athletes in the study who scored under 14 sustained an injury during their season. These findings should not be generalized to the public at large.

7. Can a guided rehabilitation that is influenced by the findings of the FMS reduce injuries?
Yes. But it needs to be specific to the movement impairments identified by the FMS. Peate had 433 firefighters perform a guided rehabilitation program that was developed to address impairments in strength and flexibility found during FMS testing. Lost work time as a result of injury reduced 62%, and the number of absolute injuries reduced 42% compared with a historical control group. In contrast, Frost had 60 firefighters perform a general movement training program (unrelated to specific FMS findings) for 12 weeks (during which a control group performed no training). There was no difference between the general movement group’s FMS scores and the control group.

8. What is the selective functional movement screen (SFMA)?
The SFMA is a movement-based diagnostic system designed to clinically assess seven fundamental movement patterns in those with known musculoskeletal pain. Certification can only be obtained by licensed healthcare professionals. It takes between 2 and 3 minutes to conduct the initial top tier movement screen to identify dysfunctional movements. The full assessment varies based on the individual’s findings on the initial top tier screen. The practitioner is obligated to “break out” all dysfunctional movements, beginning with the most dysfunctional, nonpainful patterns. The concepts of regional interdependence, altered motor control, and the neurodevelopmental perspective drive the assessment process.
The movements of the top tier SFMA screen include:

- Active cervical flexion
- Active cervical extension
- Cervical rotation
- Upper extremity pattern 1 (extension/adduction/internal rotation)
- Upper extremity pattern 2 (flexion/abduction/external rotation)
- Multi-segmental flexion
- Multi-segmental extension
- Multi-segmental rotation
- Single leg stance
- Overhead deep squat

9. What is the theory behind the SFMA?
The theory behind the SFMA is to utilize an algorithmic approach to the evaluation of functional movement and to determine what impairments are the cause(s) of the dysfunctional movement. When a dysfunctional pattern is identified in the initial top tier screening, the SFMA is designed to advance with breakout patterns and isolate the root cause of the dysfunction as either a mobility or stability impairment.

10. What is the difference between the SFMA and FMS?

- The FMS is simply a screening tool that identifies dysfunctional movement patterns and movement asymmetries that could lead to injury, pain, or dysfunction. The FMS neither identifies specific impairment causes nor provides corrective strategies. The FMS can be performed by all fitness or medical professionals who have taken and passed a certification course.
- The SFMA is for licensed health care professionals to establish a movement diagnosis. It takes the initial screening process further into algorithmic breakouts that can identify precisely the impairments leading to dysfunctional movement, which can then be addressed through therapeutic means. Individuals who take the SFMA certification course also must pass a test to become certified practitioners.

11. What are the key concepts of the SFMA?

a. Regional interdependence: the concept that all movements require the entire motor system to operate in synergy. Regional interdependence suggests that impairments distant of the area of pain/dysfunction affect the region of concern.

b. Neurodevelopmental perspective: movement is developed in patterns and not as individual muscles; therefore the assessments within the SFMA were derived from basic developmental movement patterns.

c. Altered motor control: motor control is a process in which a neuromuscular system is responsible for coordinated movement of the body when performing a motor skill. The absence of normal motor control can lead to compensated movement patterns, pain, and dysfunction.

12. Provide an example of an SFMA breakout.
SFMA breakouts are designed to further assess dysfunctional movement. Ultimately dysfunctional movement will result from a stability or mobility impairment. The breakouts are designed to algorithmically take apart the movement patterns to determine which type of dysfunction is present and where. The algorithms are built using the following principles, and it is suggested that movement patterns are evaluated in this sequence:

- Bilateral to unilateral (or removal of a body part)
- Loaded to unloaded
- Active to passive

Example 1:
1. Multisegmental flexion (MSF) is limited in the top tier (standing/weight-bearing) screen; it is also limited on both the right and left when the patient performs it with one knee bent (bilateral to unilateral).
2. The patient then performs the long sit test, reaching for the toes and unable to touch the toes (unloaded hips/loaded spine)
3. They then perform an active straight leg raise, which is limited, followed by a passive straight leg raise (active to passive), again limited.
4. The consistency seen here identifies a mobility impairment. However this breakout is taken further to look at the hip and spine mobility.
5. Double knees to chest is then performed and found to be limited, incriminating the hip joint mobility as impairment. The hamstrings, or posterior chain tissue extensibility, may also be limited, but the joint mobility would be a priority.

6. Finally, the patient assumes a prone rock position looking at the curvature of the lumbar spine in an unloaded position (another component of the ability to perform multisegmental flexion in a weight-bearing position). If limited, this would be a lumbar joint mobility dysfunction or tissue extensibility dysfunction, and if normal, it would be considered a normal finding in this example.

Example 2:

1. MSF is limited in the top tier (standing/weight-bearing) screen; it is also limited on both the right and left when the patient performs it with one knee bent (bilateral to unilateral).

2. The patient then performs the long sit test, reaching for the toes and able to touch with a uniform spinal curve and a normal sacral angle.

3. Patients have now shown the full mobility required to perform MSF in an unloaded posture, and therefore, this is considered a stability and/or motor control dysfunction.

13. How is the SFMA scored?

Each of the top tier movements has specific criteria that must be met to consider the pattern “functional.” If any one of the criteria is not met for a given movement, it is labeled as “dysfunctional.” In addition, if the movement creates or reproduces pain, it is labeled as “painful.”

a. Functional nonpainful (FN) – a normal movement pattern and therefore no need for further analysis

b. Functional painful (FP) – address the pain as needed that is associated with the functional movement. No need to perform the breakout. Use these movements as measures of success after intervention for the painful movement patterns.

c. Dysfunctional painful (DP) – proceed with caution. Address pain before or while performing breakouts to identify the impairments leading to the dysfunctional movement and pain.

d. Dysfunctional nonpainful (DN) – proceed with breakout patterns to isolate the impairment leading to the dysfunctional movement.

14. What is the SMFA philosophy regarding the treatment of painful movements?

The SFMA approach suggests treating chemical pain first. The SFMA approach believes that pain will lead to altered motor control and dysfunctional movement. If dysfunctional nonpainful patterns are present those should be addressed and then painful patterns reassessed to determine whether pain has decreased or resolved. If there are no DNs present, DPs should cautiously be assessed and broken down. In each breakout, if a DP or FP end point is reached, the pain should be addressed and the patient reassessed.

15. What is mobility-based impairment?

Mobility impairments are defined by SFMA as being a JMD (joint mobility dysfunction) and/or TED (tissue extensibility dysfunction). A tissue extensibility dysfunction is limitation of a joint as a result of active or passive muscle insufficiency, neural tension, facial adhesions, lack of muscle lengthening, hypertrophy, trigger points, or fibrotic tissue. Joint mobility dysfunction is a limitation of or within the joint as a result of conditions such as osteoarthritis, osteoarthrosis, fusion, or adhesive capsulitis. A tissue extensibility dysfunction is an extraarticular limitation of a joint or region such as active or passive muscle insufficiency, neural tension, facial adhesions, lack of muscle lengthening, hypertrophy, trigger points, or fibrotic tissue.

16. What is a stability-based impairment?

A stability impairment is defined by SFMA as an SMCD. Instability could be caused by impaired static and/or dynamic stabilizers. In addition, instability may be caused by poor motor control. The inability to coordinate muscle control or asymmetry because of a lack of stabilization leads to dysfunctional movement. An example of this would be reduced active cervical rotation in sitting but full active cervical rotation in supine. Unloading of the spine, and stabilization of the scapulothoracic complex in supine, allows for full active range of motion, which is not achieved in sitting.

17. What is the purpose of the fundamental rolling assessments used in the SFMA?

When looking at the developmental sequence, the first large movement pattern humans develop is that of fundamental rolling from supine to prone and prone to supine. This unloaded task requires coordinated sequencing of segmental stabilizers in order to perform the activity correctly and is oftentimes
dysfunctional in those individuals suffering from various injuries and/or pain. The activity of rolling would be considered a motor control issue if it is dysfunctional. The SFMA is used to “grade” the severity of SMCD impairments—fundamental versus weight-bearing, or higher level SMCD. The SFMA requires the fundamental patterns of rolling be present and established before progressing to any higher level exercise or corrective.

Example: if a person is unable to perform the multisegmental flexion pattern, and in the breakout was found to have an “SMCD,” he or she would then be assessed with the supine to prone rolling patterns (flexion biased) to determine whether this was a fundamental SMCD or a higher level problem.

18. What are the fundamental rolling assessments used in the SFMA?

There are eight total patterns of rolling utilized in the SFMA:
- Supine to prone with upper extremity lead right
- Supine to prone with lower extremity lead right
- Supine to prone with lower extremity lead left
- Prone to supine with upper extremity lead right
- Prone to supine with upper extremity lead left
- Prone to supine with lower extremity lead right
- Prone to supine with lower extremity lead left
- The supine to prone patterns are flexion biased, the prone to supine patterns being more extension biased, and all are rotational in nature.

Each pattern is performed by keeping the nonleading extremities completely relaxed. Upper extremity lead patterns also make use of the head and neck throughout the pattern.

19. What is the intra- and interrater reliability of the SFMA?

Intrarater reliability is excellent when using a categorical scoring tool. Intrarater reliability ranges from good to poor when using a criterion checklist scoring tool. Interrater reliability was slight to substantial when using a categorical scoring tool and unacceptable when using a criterion checklist scoring tool.

BIBLIOGRAPHY


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1. **What is the cost of chronic pain?**
   The economic cost secondary to lost productivity and health care expenses for all chronic pain approaches $100 billion annually in the United States.

2. **Can chronic pain be prevented?**
   The quality of acute pain management is an important factor in the subsequent development or prevention of chronic pain. Persistent postsurgical pain may be seen in patients in whom lower doses of analgesics were initially prescribed, resulting in ineffective analgesia in the early postoperative days. Nerve block and spinal analgesic techniques can hasten the rehabilitation of orthopedic patients. Patients receiving multimodal analgesia that includes spinal local anesthetics and spinal opiates have improved health-related quality of life measures for months after surgery compared with patients receiving intravenous analgesic regimens.

3. **Define preemptive analgesia.**
   Preemptive analgesia may refer to the administration of agents before injury to prevent the ensuing cascade of events that leads to the development of chronic pain.

4. **How does the response of the central nervous system contribute to the genesis of chronic pain?**
   High-intensity noxious stimulation alters central processing of afferent neural information. Studies elucidating the mechanisms for central hypersensitivity have documented a host of neurochemical changes, including enhancement of dorsal horn neuronal activity after repetitive C-fiber barrage (wind-up); receptive field expansion with decreased dorsal horn threshold, resulting in both temporal and spatial summation; and increases in immediate gene and dynorphin expression. Resultant increases in the synthesis of nitric oxide (NO), a highly diffusible gas that freely disperses to surrounding regions of the spinal cord, induce a positive feedback cycle with clinical pain on light touch (allodynia). Spinal cord sensitization leads to increased sensitivity in wide areas surrounding the site of injury (secondary hyperalgesia). This sensitivity interferes with movement and rehabilitation. Further, considerable evidence supports a heritable basis for some neurologic conditions, including neuropathic pain. Susceptible people may be predisposed to the development of chronic pain after trauma, especially in the presence of unrelied acute pain, where spinal mechanisms (including constitutive cyclooxygenase-2 pathways) participate in the development of a “memory” for pain.

5. **Do continuous analgesic infusions prevent early recognition of posttraumatic compartment syndromes?**
   The pain associated with acute compartment syndrome typically breaks through properly regulated analgesia during brachial plexus infusion or lumbar epidural infusion. Furthermore, if weakness develops during the infusion, the local anesthetic can be withheld to facilitate prompt assessment.

6. **If no pain relief is obtained by sympathetic block, can the diagnosis still be sympathetically maintained pain?**
   Sympathetically maintained pain (SMP) retains clinical utility for its therapeutic implications. It applies to a multitude of posttraumatic pain conditions with both burning pain and alldynia, which are, by definition, relieved by sympathetic block. Dystrophic changes, neural injury, and vasomotor or sudomotor changes are often present but are not required for the diagnosis. Complex regional pain syndrome (CRPS type I, also known as reflex sympathetic dystrophy [RSD]; or CRPS type II, also known as causalgia) may be either sympathetically maintained (SMP) or sympathetically independent (SIP).
7. Is chronic neuropathic pain peripheral or central in origin?
Neural injury can alter the tonic level of conduction from the dorsal root ganglia and thus sensitize the nociceptors subserving the cutaneous distributions of the affected nerve root. The spread of sensitization to areas surrounding the injury appears to be mediated in part via wide dynamic range (WDR) neurons in the spinal cord. WDR neurons also appear to be the mediators of SMP. Sensitization of WDR neurons in the spinal cord is termed wind-up. Any low-threshold myelinated mechanoreceptor afferent activity converging on the same WDR neurons results in an exaggerated response, such as allodynia. Continuous pain results from sympathetic efferent sensitization of the peripheral sensory receptors, which in turn produces tonic firing of the low-threshold myelinated mechanoreceptors, projecting onto previously sensitized WDR neurons. Thus a painful cycle involving both peripheral and central components maintains neuropathic pain.

8. Why do muscles ache?
Although muscle pain and deep hyperalgesia are associated with a number of conditions as secondary phenomena, they may also be the primary source of pain. Primary nociceptors from muscle tissue are nerve fibers that, unlike rapidly transmitting “sharp” pain pathways, transmit afferent information slowly, thus giving rise to dull, aching pain. A-delta polymodal nociceptors responding to mechanical stimulation (group III) and unmyelinated C-fibers responding to ischemia and chemical stimuli (group IV) give rise to poorly localized, cramping muscle pain. The referred pain from muscle likely represents the extensive involvement of reflex mechanisms in the central nervous system. Hyperalgesia caused by central sensitization may result from activation of N-methyl-D-aspartate (NMDA) or other mechanisms of modulation of central synaptic processing.

9. How do trigger points differ from chronic muscle tenderness secondary to fibromyalgia?
Histologic changes associated with trigger points include atrophy of type II muscle fibers, a characteristic “moth-eaten” appearance of type I fibers, and segmental muscle fiber necrosis. Some investigators have noted elastic projections constricting affected muscle fibers. Lipid and glycogen deposition and abnormal mitochondrial accumulations are seen and result in muscular bands that are often clinically palpable. Pain from deep somatic structures is typically dull and diffuse. The ability to localize precise trigger areas decreases with increasing tissue depth. Diffusion and radiation can be indicators of severity. Muscle spasm and tenderness in zones of reference (as distinguished from trigger points) often appear at sites distant from the lesion.

10. What causes trigger points?
The precise mechanism of trigger point generation and perpetuation after trauma is not known. Transient overload of a muscle may cause damage to the sarcoplasmic reticulum. Once the t-tubule system is focally disrupted, localized zones within a muscle remain in a perpetually contracted position because of impaired calcium reuptake.

11. Why are trigger points painful?
After damage to the t-tubule system, stored calcium ions are released into the area of injury. Adenosine triphosphate (ATP) may activate the actin-myosin contractile mechanism focally in the absence of action potentials. A palpable band of electrically silent muscle may result. With calcium reuptake limited, unabated focal contractile activity persists. High levels of metabolic activity, documented by ATP depletion, produce the “hot spots” seen on infrared thermography. Further, because ATP is required for the calcium pump to retrieve calcium into the sarcoplasmic reticulum, depletion of ATP further enhances calcium availability and thus perpetuates contractile activity. Accumulation of metabolic byproducts results in local acidosis, which sensitizes adjacent nociceptors. Likewise, increased calcium may act as a second messenger to induce nociceptive neuronal hypersensitivity. Increased vascular permeability, local vasoconstriction, and reduced tissue oxygenation also contribute to the elaboration of algesic substances, which sensitize peripheral nociceptors. In addition, sensitized dorsal horn cells may cause enlargement of the receptive field, resulting in spreading dysesthesia.

12. Can physical manipulation affect the healing process after muscle injury?
Perpetuation of trigger point activity can be expected until the integrity of the sarcoplasmic reticulum is reestablished or the band is physically lengthened to prevent further interaction of the actin-myosin complex. If the taut muscular band comprising the trigger point can be stretched
effectively without inducing reflexive contraction secondary to pain, the reparative process is facilitated.

13. **How can trigger points induce sympathetic overactivity?**
Sensitized muscle nociceptors may evoke sympathetic hyperactivity. Sympathetic activation may sensitize nociceptors, inducing cyclical reflex mechanisms. The progression from acute posttraumatic muscular pain to chronic myofascial pain probably involves peripheral sensitization of high-threshold mechanoreceptors, recruitment of low-threshold mechanoreceptors, and central sensitization of dorsal horn neurons. Increased sensitivity of muscle vasculature to sympathetic transmitter substances may contribute. Clinically, persistence of trigger point activity can result in sympathetically mediated vasomotor changes. In such cases, sympathetic blockade can assist the manipulative therapy.

14. **When does the inflammatory cascade cease to be useful after musculoskeletal injury?**
The response to connective tissue injury is divided into stages. The first stage in the healing process is the inflammatory response, which typically extends through the first 2 days. During this phase chemotactic mechanisms induce cell mobilization and infiltration. The second stage lasts from the third day through the fifth and is characterized by ground substance proliferation in preparation for collagen deposition. Collagen formation begins in the third proliferative phase and lasts through the second week after injury. The final stage is the formative stage: from 14 days onward, the cross-linked collagen organizes into functional fibrils in the healed tissue. Persistence of inflammation beyond the initial period is not helpful and contributes to persistence of pain.

15. **Can corticosteroids interfere with healing?**
The timing of therapeutic intervention affects the quality of the reparative process. Corticosteroid administration in the first 2 weeks can inhibit prostaglandin synthesis, thus interfering with the initial proliferative phases of healing. Corticosteroids should be used with caution in acute strains and only when the joint is to be placed at rest. Furthermore, corticosteroid-induced fluid retention may contribute to tissue swelling. This effect may be most damaging after crush or blunt trauma to an extremity, where additional swelling may predispose to development of a compartment syndrome.

16. **Can exercise targeted at specific defects be effective in the treatment of chronic low back pain?**
Specific programs designed to strengthen the abdominal and lumbar multifidus muscles proximal to the specific defect as a stabilizing maneuver for symptomatic spondylosis or spondylolisthesis are effective.

17. **Which patients with low back pain derive the greatest long-term benefits from physical therapy?**
Mechanical low back pain responds favorably to physical therapy interventions, whereas radicular pain does not. The finding that multifidus muscle recovery is not necessarily spontaneous after remission of pain may explain a high level of recurrence of low back pain. Specific physical therapy interventions should include manual therapy techniques based on motion-provoked symptoms.

18. **Does evidence support physical therapy for acute low back pain?**
According to the Agency for Health Care Policy and Research, physical therapy seems helpful for acute low back problems without radiculopathy when used within the first month of symptoms. If the symptoms persist and no functional improvement has been noted after 1 month, therapy should be stopped and the patient reevaluated. An early return to function is favored over traditional bed rest. Bed rest longer than 4 days is not helpful and may hasten debilitation.

19. **Are exercise programs helpful?**
Stretching programs that progress to a home training and conditioning program after 3 months of therapy have been successful in reducing pain and disability as well as increasing optimism and self-control. Massage may reduce the overall costs of health care in persistent low back pain. Exercise in association with cognitive retraining, although typically failing to improve the number of patients returning to work, may reduce symptoms and improve coping in patients with chronic spinal pain.

20. **Should nerve blocks be used to facilitate physical therapy in patients with chronic pain?**
Neural blockade immediately before manipulation of the spine enhances the efficacy of treatment used either alone or sequentially. Denervation of receptive fields related to the innervation of the facet joints may allow improved manipulation by prevention of reflex muscle spasm and guarding during
treatment. Precision in blockade is essential to avoid total sensory loss, which may permit dangerous overstretching of the tissues. Cervical epidural hematoma and subluxation with quadriplegia have been reported. Widespread nonspecific blockade may permit stretch beyond safe limits. Careful and specific physical interventions within the physiologic range, combined with blockade limited to specific target elements, are designed to minimize such risks. A prospective, double-blind, placebo-controlled study of patients with whiplash found dramatic long-term relief with radiofrequency lesioning of the cervical facets.

21. When are physical measures needed after trigger point injections?
All trigger point injections should be followed by effective stretch of the treated muscles. Moist heat immediately after treatments helps to minimize local soreness and reflex muscle spasm. Best results are obtained when injections are reserved for patients with acute trigger points that, on physical examination, reproduce the patient’s pain and are ineffectively stretched by physical means alone. Injections also should be used in conjunction with a home stretching program to facilitate therapeutic exercises. If more than three injections are required, the search for underlying precipitating factors should be intensified.

22. Why does stretching promote healing of trigger points?
Physical therapy directed specifically at stretch of the trigger points to normal resting length is crucial. This prevents actin-myosin interaction, thereby reducing metabolic activity, and improves local blood flow and tissue oxygenation.

23. What measures effectively facilitate trigger point stretching?
Specific techniques are used to permit passive stretching, including vapocoolant spray, ischemic compression, acupuncture, acupressure, dry-needling, or, most effectively, infiltration with local anesthetic (trigger point injection). Injection relieves pain, relaxes muscles (by blocking ongoing reflex activity), and physically flushes away excessive extracellular calcium, hydrogen ions, and algesic substances. Relaxation and electromyogram biofeedback should be considered adjunctive measures.

24. How can trigger point injections abolish pain at sites distal to the injection?
Afferent pain signals secondary to activation of nociceptors enter the spinal cord through the dorsal root, where communication via internuncial neurons leads to hyperactivity in the anterior and anterolateral horn cells. Hyperactivity results in efferent traffic, causing intensified muscle spasm, vasoconstriction, and referred pain. Neural blockade interrupts this reflex arc. Resultant alterations in central nervous system processing of input from the receptive field may be responsible for the spreading tenderness after injury. This response is terminated by local anesthetic application.

25. What circumstances require the application of regional local anesthetic blockade?
Somatic regional block is used when multiple trigger points in a contiguous region make individual injections impractical or when simultaneous antisympathetic effect is required to increase blood flow or reduce sympathetic activity. Regional sympathetic blockade blocks perpetuating sympathetic activity and improves microcirculation, thus decreasing focal ischemia.

26. Discuss the role of sympathetic blocks.
Sympathetic block, by definition, relieves the pain of SMP. Although repeated sympathetic blockade may reduce or permanently eliminate clinical findings, most neuropathic pains are not sympathetically maintained. In fact, not even all cases of CRPS type I are amenable to sympathectomy. However, when a positive response from sympathetic blockade is obtained, the effect of the block often significantly outlasts the action of the local anesthetic, especially when repeated. Of interest, neural blockade distal to the sympathetic chain is also effective in reducing SMP, presumably because neurogenic block of the affected receptive field reduces the low-threshold mechanoreceptor activity that is stimulated by sympathetic outflow. With time, the plasticity of the central nervous system permits enhanced transmission over previously quiescent pathways. Enhanced transmission contributes to the clinical impression that, in the most chronic cases, peripheral measures are ineffective.

27. What physical therapy treatments are helpful in conjunction with invasive therapy for chronic pain?
Manipulation after facet or medial branch blocks provides a more dramatic result than either treatment used in isolation for the treatment of mechanical spine pain. After initial recovery and application of moist heat, provocative maneuvers are repeated to ensure appropriate blockade. The affected segments are then mobilized manually. Subsequent strengthening, especially of the
multifidi when treating pain in the lumbar region, is important after permanent medial branch neurolysis. The use of spinal cord stimulation in conjunction with physical therapy in the treatment of CRPS results in significantly improved pain and economic outcomes.

28. Is physical therapy important after intradiscal electrothermal therapy? The relatively new technique of intradiscal electrothermal therapy heats the annular disc in an effort to destroy nociceptors and reorganize collagen fibrils, thus sealing fissures within the disc. Because the collagen helices are rearranged, care must be taken to avoid placing stress on the treated disc until its structural integrity is reestablished. Although only patients with annular tears meeting strictly defined criteria are likely to benefit from this procedure, in properly selected candidates the number needed to treat (NNT) for 75% pain relief was 5, with 40% of all patients achieving at least 50% relief. Physical therapy, beginning at 6 weeks after the procedure, must be introduced gradually and designed to enhance lower extremity flexibility and truncal stabilization.

29. What exercise programs are effective in chronic pain patients? Exercises designed for specific impairments, behavioral techniques that promote wellness behaviors and extinguish pain behaviors, and administration of nonsteroidal antiinflammatory drugs and antidepressants are appropriate treatments for chronic pain. Success has been reported with the following paradigm:

- Week 1: comprehensive evaluation, home stretching program, and orientation to program
- Weeks 2 and 3: 2 hours of physical therapy three times per week (1 hour of stretching and 1 hour of strengthening)
- Weeks 4 to 8: 45 minutes of stretching, 1 hour of strengthening, and 1 hour of aerobic training three times per week.

Quantification of function biweekly and at program completion is recommended. Compliance, behavioral problems, and treatment goals should be discussed biweekly at case conference. Individualized written recommendations for exercises at home or at a fitness facility should be provided at the end of the program.

30. What are the essential elements in the physical therapist’s evaluation of patients with chronic pain? Serious underlying spinal conditions, such as fracture, tumor, infection, or cauda equina syndrome, must be ruled out. Features that should raise the index of suspicion include presentation under age 20 or over age 55; violent trauma; constant, progressive, nonmechanical pain; thoracic pain; past history of carcinoma; use of systemic steroids; drug abuse; HIV infection; systemic unhealthiness; precipitous or unexplained weight loss; persisting severe restriction of lumbar flexion; and widespread neurologic symptoms. Cauda equina syndrome may present with difficulty with micturition; loss of anal sphincter tone or fecal incontinence; saddle anesthesia about the anus, perineum, or genitals; widespread or progressive motor weakness in the legs; gait disturbance; or a sensory level of hypoesthesia.

31. Can a disparity between self-report and objective measures be documented? Inconsistencies in the evaluation should be documented. Waddell signs, which are associated with inconsistent and unreliable self-reports, include superficial nonanatomic tenderness; pain with axial loading on top of the head; increasing back pain or twisting torso as a unit; discrepancy between straight leg raising in sitting and lying positions; nonphysiologic regional disturbances in sensation, pain distribution, or weakness; and excessive verbalization, facial grimacing, and other pain behaviors out of proportion to test stimulus and physical findings. The presence of three or more Waddell signs predicts treatment failure. Although Waddell signs predict poor outcome acutely, they are not necessarily negative predictors in an interdisciplinary context.

32. Which medications are appropriate for chronic pain? Medications for neuropathic pain are administered by oral, topical/transdermal, IV regional, intraspinal, and nerve-blocking techniques. The oral route is by far the most common. Oral tricyclic antidepressants are standard therapy for chronic pain.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Indication</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tricyclic antidepressants (eg, amitriptyline [Elavil])</td>
<td>Neuropathic pain, depression, myofascial pain</td>
<td>Multiple, including central inhibition of 5-HT and NE reuptake</td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td>Neuropathic pain</td>
<td>Multiple, including NA channel inhibition, reduced spontaneous depolarization</td>
</tr>
</tbody>
</table>
Gabapentin (Neurontin)  
Clonazepam (Klonopin)  
Neuropathic pain, panic attacks, anxiety states  
GABA agonist

Capsaicinoids  
(eg, capsaicin [Zostrix])  
Neuropathic pain after severe nerve injury  
Depletion of substance P

Muscle relaxants  
(eg, baclofen [Lioresal])  
Spasticity after spinal cord injury; myofascial component  
GABA agonist

NMDA receptor antagonists  
(eg, ketamine [Ketalar])  
Posttraumatic, myofascial neuropathic pain; opioid tolerance  
Glutamate receptor antagonism

Local anesthetic derivatives  
(eg, mexiletine [Mexitil])  
Neuropathic pain with C-fiber hyperactivity, neural injury or neuroma with mechanical hypersensitivity, alldynia  
Na channel blockade, reduce spontaneous depolarization

Antihypertensives  
(eg, clonidine [Catapres TTS])  
Neuropathic pain with hyperpathia/alldynia  
α₂-adrenergic agonist

Opioids  
(eg, oxycodone [Oxycontin])  
Nociceptive pain uncontrolled by other measures in patients at low psychological risk for addiction  
Opiate receptor agonists

Nonsteroidal antiinflammatory drugs  
(eg, celecoxib [Celebrex])  
Neuropathic and myofascial pain  
Cyclooxygenase inhibition blocks prostaglandin synthesis

5-HT, 5-Hydroxytryptamine; NE, norepinephrine; Na, sodium; GABA, gamma-aminobutyric acid.

33. Discuss the role of perineural steroids in pain management.
Epidural steroid injections are an option for short-term relief of radicular pain after failure of conservative treatment and as a means of avoiding surgery. Evidence-based meta-analysis of epidural steroid studies provides conflicting results. Although the Oxford group reported efficacy with an NNT of 7.3 for greater than 75% relief at up to 60 days and an NNT of 13 for greater than 50% relief at up to 12 months, the Cochrane group found no such evidence. In a randomized, controlled trial, the transforaminal approach to epidural steroid injection has been reported to eliminate the need for surgery in 50% of patients with radicular pain emanating from one to two spinal levels.

34. What is chronic regional pain syndrome (CRPS)?
CRPS, previously known as reflex sympathetic dystrophy, is a syndrome characterized by pain, vasomotor instability, trophic changes, bony changes, and sensory changes.

35. What is the difference between CRPS types I and II?
Type I does not have peripheral nerve involvement. Type II is characterized by peripheral nerve entrapment that spreads to regional involvement.

36. What is the treatment for CRPS II?
CRPS type II should be treated by decompression of the involved nerve and a postoperative therapy program.

37. What is the treatment for CRPS type I?
Treatment should be a multimodal approach including sympathetic blocks, antiinflammatory and pain-modulating medications, physical therapy, and psychological counseling.

38. What radiographic changes are seen with CRPS?
Patchy demineralization is followed by periarticular osteopenia.

BIBLIOGRAPHY
1. Corticosteroids can interfere with the healing process by:
   a. Increasing DNA synthesis
   b. Interrupting the initial proliferative phase of healing
   c. Stimulating mitosis
   d. Increasing fibrosis

2. Trigger points:
   a. Are caused by t-tubule disruption
   b. Have low metabolic activity
   c. Have decreased vascular permeability
   d. Desensitize dorsal horn cells

3. Chronic regional pain syndrome:
   a. Is self-limiting and leaves no permanent disability
   b. Type II is treated by nerve decompression and therapy
   c. Can result in joint laxity
   d. Results in increased cortical density

 CHAPTER 28 QUESTIONS

1. Corticosteroids can interfere with the healing process by:
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   a. Is self-limiting and leaves no permanent disability
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   c. Can result in joint laxity
   d. Results in increased cortical density
1. Describe the basic categories of headache and their clinical presentation.

The International Headache Society (IHS) classification defines and categorizes each headache clearly with diagnostic criteria. However, because of considerable symptomatic overlap and common features, the differential diagnosis is difficult (see table).

### Four Types of Headaches

<table>
<thead>
<tr>
<th></th>
<th><strong>MIGRAINE</strong></th>
<th><strong>TENSION</strong></th>
<th><strong>CLUSTER</strong></th>
<th><strong>CERVICAL/ CERVICOGENIC</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender and age</strong></td>
<td>Women &gt; men</td>
<td>Women &gt; men</td>
<td>Men &gt; women</td>
<td>Women &gt; men</td>
</tr>
<tr>
<td><strong>Area of symptoms</strong></td>
<td>Unilateral, temporal, frontal, or retroorbital</td>
<td>Muscle of head, periorbital, temporal, and occipital; cervical symptoms may be present</td>
<td>Frontal, retroorbital, temporal, occipital Possible neck symptoms, but mild compared with head pain Unilateral; may change sides</td>
<td>Unilateral pain usually starting in suboccipital neck region and radiating to frontal, retroorbital, temporal, and occipital regions May be bilateral Does not change sides</td>
</tr>
<tr>
<td><strong>Quality of symptoms</strong></td>
<td>Throbbing, pounding, Moderate to severe intensity</td>
<td>Dull, aching quality Tight band or heavy weight on head Moderate to severe intensity</td>
<td>Severe, intense, burning, piercing, nonthrobbing; ocular symptoms and pressure retroorbitally Typically excruciating</td>
<td>Dull ache or boring pain; stabbing, shooting deep pain may be present At times may be throbbing Can reach moderate to severe intensity</td>
</tr>
<tr>
<td><strong>Associated symptoms</strong></td>
<td>Nausea, vomiting, photophobia, phonophobia No specificity to side of pain and neurologic symptoms</td>
<td>Nausea, vomiting, and photophobia</td>
<td>Nausea, vomiting, photophobia, lacrimation, rhinorrhea, ptosis, miosis, nasal congestion, flushed face, bradycardia</td>
<td>Nausea, vomiting, phonophobia, photophobia, blurred vision, difficulty with swallowing ipsilateral to side of pain</td>
</tr>
<tr>
<td><strong>Frequency and duration</strong></td>
<td>4–72 hr, generally &lt;24 hr 1 per year–several per week</td>
<td>May occur daily (few hr to few days) Chronic (semi-continuous or 2–3/week</td>
<td>15 min–2 hr 1–8/day for 1/2 to 3 mo; chronic up to 1 yr Remission for 6 mo–2 yr</td>
<td>Daily or at least 2–3 times/week 3–24 hr</td>
</tr>
</tbody>
</table>
2. What is the epidemiology of cervicogenic headache?

The reported prevalence of cervicogenic headache varies in the literature from 2.5%–13.8%. Cervicogenic headache affects patients with a mean age of 43, tends to be chronic in nature, and has a 4:1 female disposition. The IHS describes 14 different types of headaches with subcategories/classifications. Cervicogenic headache is classified as a secondary headache.

<table>
<thead>
<tr>
<th>Diagnostic Criteria for Cervicogenic Headache</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Pain, referred from a source in the neck and perceived in one or more regions of the head and/or face, fulfilling criteria 3 and 4</td>
</tr>
<tr>
<td>2. Clinical, laboratory, and/or imaging evidence of a disorder or lesion within the cervical spine or soft tissues of the neck known to be, or generally accepted as, a valid cause of headache</td>
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<tr>
<td>3. Evidence that the pain can be attributed to the neck disorder or lesion based on at least one of the following:</td>
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<tr>
<td>a. Demonstration of clinical signs that implicate a source of pain in the neck</td>
</tr>
<tr>
<td>b. Abolition of headache following diagnostic blockade of a cervical structure or its nerve supply using placebo or other adequate controls</td>
</tr>
<tr>
<td>4. Pain resolves within 3 months after successful treatment of the causative disorder or lesion</td>
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3. Describe the symptoms of cervical headache.

Although symptom location may vary widely, prevalent sites of pain are retroorbital, frontal, temporal, and occipital areas of the head. Usually suboccipital pain and neck pain also are present. These symptoms have strong tendencies to be unilateral with no changing of sides. Other studies report bilateral symptoms, although they are rare. Pain is described as an ache or dull, boring pain that varies in intensity from low grade to severe. At times throbbing or pulsing pain may be reported, but is typically in...
migraine headaches with the throbbing coinciding with pulse. Associated symptoms include nausea, vomiting, phonophobia and photophobia, visual disturbances (blurred vision, spots, flashing lights), difficulty in swallowing, dizziness, light-headedness, general irritability, and inability to concentrate. Symptoms are ipsilateral with pain. Headache pain is often present on awakening and may worsen progressively with increased activity levels. Other patients may have an onset of symptoms during or toward the end of the day, with neck pain as a warning sign.

4. How are cervical headaches precipitated?
Cervical headache commonly is precipitated or intensified mechanically by sustained neck flexion while working at a desk, typing, studying, driving a car, or reading. Often patients have difficulty in identifying specific aggravating factors. Although stress, tension, anxiety, and depression also may be provocative factors, they are common to other headache types. Cervical spine pathology may trigger muscle contraction (tension), or, conversely, tension may provoke existing pathology to produce headache. It is easy for patients to blame stress in their life as a causative factor, but millions of people with significant stress or tension in their lives are symptom-free. Thus musculoskeletal causes of headache should not be ruled out.

5. Discuss the neuroanatomic basis for cervicogenic headache.
Afferent fibers from the trigeminal nerve (cranial nerve V), which carries pain and temperature information for the head region, descend through the medulla oblongata and into the gray matter of the spinal cord as far as C3 and occasionally C4. Afferent fibers from the C1-C3 spinal nerves synapse at the segment at which they enter the spinal cord and send collateral branches to superior and inferior segments. Within this column of the spinal cord, the gray matter that receives both trigeminal and cervical afferents is called the trigeminocervical nucleus. This combined nucleus is essentially the nociceptive nucleus of the head, throat, and upper neck. The convergence of afferents constitutes the basis for referred pain in the head and upper neck. If afferents in the trigeminocervical nucleus that otherwise innervate the back portions of the head also receive upper cervical vertebral afferents, nociceptive upper cervical stimulation may be interpreted as arising in the head. All afferents converging on the trigeminocervical nucleus may refer pain to other structures that also synapse in the same nucleus.

![Trigeminocervical Nucleus Diagram](https://example.com/trigeminocervical.png)

6. Which structures facilitate synapsis of afferent information to the trigeminocervical nucleus?
Structures include all of the articular, muscular, and neural structures of the cervical spine from C0 to C3; the upper portion of the vertebral artery; the temporomandibular joint; the posterior cranial fossa/upper spinal cord dura mater; and cranial nerves V, VII, IX, and X.

7. Describe the anatomy of the posterior neck musculature, C2 sensory nerve root, and occipital notch.
Seven layers of muscles attach to the cervical vertebrae and the skull in the posterior neck region. From superficial to deepest, they are the trapezius, splenius capitis, longissimus capitis, semispinalis capitis, obliquus capitis, splenius cervicis, and multifidus. The dorsal root of C2-C3 courses under the obliquus capitis and through the splenius capitis and trapezius muscles before traversing the occipital notch and onto the scalp. The occipital nerve and the deep cervical artery and vein course through the muscles approximately 2 to 3 cm lateral to the midline at the level of the free edge of the posterior skull. The palpated notch on the skull edge is called the occipital notch.

8. What do cervical radiographs show in patients with headache?
Conventional radiographic studies comparing patients with cervical headache and controls found no significant differences. However, one study using computer-based analysis of median tomograms in maximal cervical flexion and extension found significant segmental hypomobility of the craniocervical joints from C0 to C2—most pronounced at C0/C1. In addition, the study found impaired overall mobility of the superior cervical spine from C0 to C5.

9. What is the gold standard for diagnosis of cervical headache?
A C2 nerve block or joint block on the symptomatic side can be used for diagnosis as well as therapeutic purposes. Patients generally report reduction of pain or complete resolution of symptoms if the block was successfully targeted. However, studies report no long-lasting therapeutic effect or even remission of pain. The pain cycle has been broken, but the underlying functional problem still exists, whether it be posture, cervical strength, cervical mobility, or myofascial problems.

10. List important differential diagnoses of cervicogenic headache.
- Chiari I malformation
- Posterior fossa tumors
- Vascular cerebral aneurysm
- Paroxysmal hemicrania
- Hemicrania continua
- Rheumatoid arthritis
- Syringomyelia
- Temporomandibular joint dysfunction (TMD)
- Neck-tongue syndrome
- Whiplash injury
- Migraine, occipital neuralgia, tension-type headache

11. How do poor posture and muscle impairment contribute to cervical headache?
Faulty postural habits can lead to abnormal stresses in the cervical and upper thoracic spine. In particular, forward head posture affects the biomechanics of the head and neck region, putting greater stress on muscles that function as stabilizers of the head. If forward head posture is maintained, it becomes fixed through adaptive shortening in upper cervical joints and posterior superficial and deep myofascial structures. Studies have shown that headache patients exhibit abnormal responses to passive stretching of the upper trapezius, levator scapulae, and short upper cervical extensor muscles. In addition, isometric strength and endurance tests have shown that the upper cervical flexors are significantly weaker in patients with headache compared with asymptomatic controls.

12. What types of physical therapy are useful in reduction of cervical headache?
The goal of physical therapy is to address objective findings of the evaluation. If faulty posture patterns are found, the therapist most likely will find impaired mobility in the upper cervical spine and subsequent forward shoulders with general weakness in the posterior shoulder girdle musculature. Initially, the therapist must correct myofascial and joint restrictions in the cervical and thoracic regions, generally with mobilization and manipulation of affected areas. Other important aspects are postural correction and reeducation by encouraging axial extension and shoulder retraction. Reinforce the importance of posture maintenance to reverse the pain cycle that results from strain on joints and various soft tissues of the cervical spine.
13. What exercises are believed to be of most benefit for the headache patient?
Stretching and exercise should target muscles of the upper quadrant with extensibility losses and weakness. Stretching should focus on posterior neck superficial and deep muscles, including the upper trapezius, levator scapulae, musculus scalenus, sternocleidomastoid, suboccipitals, and pectorals. Strengthening exercises should help to maintain gains in joint mobility after mobilization and stretching by focusing on the trapezius, rhomboids, and deep cervical flexors.

14. What does the evidence illustrate regarding manipulative therapy and/or therapeutic exercise for cervicogenic headache?
There is evidence that both specific therapeutic exercise and manipulative therapy are effective for cervicogenic headache. Benefits included a reduction in all of the following: headache frequency and intensity, neck pain, disability, and medication intake. Jull et al. provided evidence of a long-term treatment effect over a 12-month period. Their multicenter, randomized controlled study used a manipulative regimen described by Maitland, including low-velocity cervical joint mobilizations and/or high-velocity manipulations. The exercise program involved low load exercise directed to reeducate muscle control of the cervicoscapular region specifically targeting the deep neck flexors, postural correction exercises, and muscle lengthening as needed. It is believed that long-term effectiveness is concurrent with consistent use of a home exercise program and postural pattern awareness.

15. Are there predictors of responsiveness to physical therapy treatment on cervicogenic headache?
No. In the previously mentioned study, 25% of patients did not achieve a clinically acceptable outcome of 50% reduction in headache frequency. Analysis of identifying predictors from variables in subject demographics and headache history revealed no consistent pattern to minimize against a successful outcome from physical therapy intervention.

16. What other nontherapy treatments are there for cervicogenic headache?
- Nerve blocks – diagnostically and therapeutically
- Cryo procedures
- Radiofrequency neurotomy
- Pulsed radiofrequency
- Intraarticular Z-joint corticosteroid injections
- Greater occipital nerve (GON) decompression

17. What do systematic reviews reveal in management of cervicogenic headache with physical therapy and/or manual therapies?
Systematic reviews indicate physical therapy and cervical spinal manipulation to be effective in the management of cervicogenic headaches with regard to reducing headache intensity, frequency, duration, and neck pain. In addition, the most effective intervention seen was a combination of mobilization, manipulation, and cervicoscapular strengthening exercises.

18. List similarities/differences in distinguishing cervicogenic headache from migraine with aura.
Similarities:
- Unilaterality of the headache
- Occurrence predominantly in women
- Possible occurrence of nausea and vomiting
Differences:
- Unilaterality without “side shift” in cervicogenic headache (HA). Migraine without aura can shift sides during the same headache attack and between individual headache attacks.
- Migraine usually begins in the frontotemporal region; cervicogenic usually begins in the cervical spine.
- Cervicogenic headache can be provoked by manual pressure in the upper lateral cervical spine on the symptomatic side. This generally does not occur with migraine.
- Cervicogenic headache often presents with limitation of cervical ROM. In migraine, this is not a characteristic.

BIBLIOGRAPHY
CHAPTER 29 QUESTIONS

1. Which structure does not synapse with the trigeminocervical nucleus?
   a. Cranial nerve III
   b. Cranial nerve V
   c. Cranial nerve VII
   d. Cranial nerve IX

2. Who is at greatest risk for cervical headache?
   a. Women
   b. Men
   c. Professional occupations
   d. a and c

3. What headache pattern effects men more often than women?
   a. Migraine
   b. Cluster
   c. Tension
   d. Cervical
CHAPTER 30
FUNCTIONAL CAPACITY TESTING
AND INDUSTRIAL INJURY
TREATMENT
S.J. Isernhagen, PT

1. What are the types of functional evaluations used for assessing work capacity?
   The functional capacity evaluation (FCE) was the first type of test used by therapists to evaluate work function. It is defined by the American Physical Therapy Association (APTA) as an “objective measure of a client’s safe, functional abilities, compared with the physical demands of work.” The FCE should be standardized, thus being able to be evaluated for reliability. Therefore it is a generic test of work function, not one specific to any job.
   To define the ability of a person to do a specific job, a job-specific test (JST) is also a type of functional evaluation. The JST, to be accurate, must start with an objective job function evaluation. From the measured job functions (also termed essential functions), a test is developed that assesses functional capacity to perform that specific job. If the test replicates the job, it has content validity.

2. When should FCEs be performed?
   Because FCEs are long and relatively expensive (usually $500–$2000), and because JSTs are available for earlier testing, are more accurate for specific jobs, are shorter, and less costly, the FCE is used most often at later stages. Indications are when the worker is stable, has not resumed regular employment, and the need for clear physical abilities and restrictions is present. In return to work, best outcomes are gained with referral as soon as medical stability has been reached to produce the lowest likelihood of disability.

3. How is a functional capacity examination (FCE) used?
   An FCE provides the decision maker specific information about return to work decisions, specific job placement, job modification, disability evaluation, and determination of work capability. Often it is used to aid in the permanent impairment ratings that must be made when a workers’ compensation case is closed. It also is used as an entrance examination for work rehabilitation and provides excellent information for case management and case closure.

4. What are the typical components of an FCE?
   The components most often used follow the U.S. Department of Labor definitions and include lifting, carrying, pushing, pulling, gripping, pinching, hand coordination, reaching, bending, climbing, walking, standing, sitting, and balancing. Some referrers may require a general category of workload such as sedentary, light, medium, heavy, and very heavy. These are defined by state code or the Dictionary of Occupational Titles.

5. What other areas are covered?
   For chronic cases, referrers are also interested in the level of effort or cooperation. Consistency of performance is often rated. Many try to indicate that inconsistency is an indication of poor effort, but inability to reproduce a test score can sometimes be from physical problems. Information regarding whether pain interferes with the effort levels is often included. All of these lead to the importance of identifying the medical/physical reasons behind the functional limitation.

6. Does an FCE have a role in legal disability cases?
   The FCE plays a pivotal role, because it is the only definitive test that measures actual work function. In a typical court hearing, the physician testifies first about the medical status, diagnosis, and prognosis of the worker. The physical therapist then testifies about the outcome of impairments. In other words, what functional capacity does the worker retain and how does it relate to work activities?
Often the third expert is the vocational evaluator, who identifies what jobs are within the person’s safe physical capabilities.

7. **How should pain be reported in an FCE?**
The FCE is a test of function, but safety is also a prime factor. Pain itself is not a contraindication to testing. The most relevant aspect of the pain report is change in the initial level or area of discomfort during a test. The therapist, with a background in pathology and kinesiology, can determine whether and when a test item should be stopped. Because most persons who take an FCE do have chronic conditions, pain is often present. The importance of the patient’s history and physical relates to the ability of the therapist to know when to stop a test because of a physical/medical problem that affects safety.

8. **Could a client stop performing in an FCE if he or she did not want to participate?**
Consent forms and instructions should indicate that the client is aware of his or her ability to refuse a test or test completion. Although effort should be made to make the client feel safe and informed, it remains the right of the client to decline a test or test completion. The documentation would merely state that the test was not completed. Clients are more likely to use full effort when there is a good rapport between the evaluator and the client. The client must trust that the therapist is knowledgeable and objective.

9. **Are FCEs and JSTs medical tests?**
Yes. When the test is performed by a medical professional, it is considered a medical test. Also, APTA guidelines state that a history and physical must be performed before the test, thus adding these medical components.

10. **What reliability and validity measures should be applied to functional evaluations?**
Because an FCE is a standardized test, it can be subject to interrater reliability and intrarater reliability studies. This is critical and most often relates to the training and procedures of a test structure being followed exactly.

    The most important validity measure for work-related tests, where the evaluee will be working in the real world, is “content” validity. This is the measure that says that what the test finds will hold true in the real world. Although FCEs can have this attribute, it may be more in the level of work than job specificity (as the FCE is not job specific). The JST, developed properly and given accurately, should have a high level of content validity. Outcome studies are the methods of finding used most often if functional testing did result in successful return to work.

11. **What type of functional evaluation is best for hiring and placing workers at their job?**
The JST is the correct test for placing people at specific jobs. The Americans with Disabilities Act and its Amendment Act (ADAAA), specifically require that any decision on hiring and placing be done by identifying if the person can do the essential functions of the job. The ADAAA also states that a person who falls under the broad definition of disability must be offered a reasonable accommodation. The JST can often pinpoint the functional area where an accommodation is necessary. In hiring, the JST is used after an offer of employment is given. The therapist performs the tests, and the employer makes the hiring decision.

12. **Can a JST be used in return to work?**
Yes. In return to work, a JST can be used very early in the process or at any point where the original job or a specific job is targeted. It is a strong step in keeping the worker in an employment mode and with the work team. In the beginning, most, but not all, of the essential job functions can be performed. Those that cannot be performed become a goal for therapeutic intervention. Retesting until all the functions can be done safely allows early return to work and safe progression to full duty.

    The employer must receive the job ability information for proper placement back at work to occur, and the worker must be confident in his or her abilities in order to agree to return to work. The focus is on the employer-worker connection as return to work is dependent on both parties agreeing on the work assignment.

13. **What are the therapeutic interventions that either keep the injured/ill worker at work or can move the worker toward the return to work goal?**
   a. Therapists working onsite in industry provide excellent ability to place the person at the job functions of which they are capable. For the functions that require increased strength, coordination, endurance, or aerobic capacity, the therapists can design an exercise program. The therapist then is able to add job functions when capability has been established. The onsite therapist is increasingly being used in industry.
b. For clinical programs work conditioning or work hardening can be used. The APTA states that work conditioning is a specific work-related, intensive, goal-oriented treatment that focuses on strength, endurance, movement, flexibility, motor control, and cardiopulmonary functions. Work hardening is a broader rehabilitation program, which is interdisciplinary in nature. In addition to what is covered in work conditioning, behavioral and vocational functions also are addressed. In all cases, employer and employment goals are the target.

14. What are the eligibility requirements for work conditioning or work hardening?
According to the APTA guidelines, the worker must have the following:
- A job goal
- Stated or demonstrated willingness to participate
- A physical or functional deficit that interferes with work
- A point of resolution after the initial injury at which time participation in the program would not create harm

15. How does a therapist obtain cooperation from a client who is not working toward the program goals?
This common issue is best prevented by educating the client during the admission process. The preceding “rules” are discussed. The worker/client often signs a contract that indicates he or she will work toward the program goals. The program is the client’s “job,” and the client must often clock in and out. The client completes the daily work with self-responsibility, although the therapist and supportive staff are available to help. The focus is on the worker getting better and not on the “treatment.” Progress must be made, or the program will be terminated. The goals are reached when the worker/client is work-ready.

16. If a worker cannot meet the physical demands of work after an FCE or work rehabilitation program, what are the options?
A strong FCE can be given to identify areas of work ability. Case managers and employers who need productive workers then look at their abilities and can match the worker with a job within those capabilities. If there is still a functional discrepancy, job modifying with adaptive equipment, assistive devices, or teamwork is also an option. Because the employer and the worker are the ultimate decision makers, both parties must agree that the final work placement or job modification will be accomplished.

17. What are the major outcome measures for work rehabilitation?
The worker returning to work for a willing employer is the desired outcome. Outcome measures include return to work information and demographic and performance information gained from the test or program. Outcome data include return to work information such as:
- Same or different employer
- Previous or different job
- Full time or part time
- Time of safe return to work

18. How should a therapist evaluate the advantages and disadvantages of proprietary FCEs and JSTs?
- Is the FCE standardized? Are JSTs based on the measured and objective functions of the job? (Many functional testing vendors also teach job analysis and the development of job function descriptions.)
- Does it explain full policies and procedures for performing the tests?
- Is training in the system part of the purchase of the program?
- Have outcome studies been done to verify the real-world test effectiveness?
- Have reliability studies been done on the test or important test components?
- Has the predictive validity been established by identifying whether the tested capacities hold true in actual return to work?
- Does the FCE meet the requirements of disability insurance companies?
- Will the medical/legal credibility and history of the FCE and JSTs stand up in court?
- Is the FCE reliant on dynamic work tests rather than static isometric tests?
- Is the FCE infused with safety parameters?
- Is the report format clear and easy to read?

**CHAPTER 30 QUESTIONS**

1. Who are the primary decision makers as it relates to returning an injured worker back to work?
   a. The doctor and the therapist
   b. The employer and the worker
   c. The case manager and the doctor
   d. The insurer and the employer

2. What type of test would be best used to determine ability to do a specific job?
   a. A Job Specific Test (JST)
   b. A functional capacity evaluation (FCE)
   c. A history and a physical performed by the therapist
   d. A history and physical performed by the physician

3. In a work conditioning program (onsite or at a clinic), what brings the best results?
   a. Therapist deep tissue massage and ultrasound
   b. Exercise focused on highly repetitive large leg muscle exercise to build lower extremity strength
   c. Therapist closely monitoring each and every part of the program
   d. The patient-worker being self-motivated for program compliance and their own progress
1. What is a mnemonic?
Named after Mnemosyne, the Greek goddess of memory, mnemonics simply means “memory aid.” It is a learning device in which we relate a collection of hard facts to a known word, sequence of letters or numbers, or a rhyme in an effort to recall the facts accurately and sequentially. In human anatomy, there are thousands of facts to learn, and it is the volume of such facts that becomes the challenge and hence the beauty of mnemonics.

2. Can I make up my own mnemonics?
Yes. You have poetic license to construct your own mnemonics based on things you encounter in your own life.

3. What is the military saying for shoulder muscles?
“Lady between two majors.” “Lady” is actually “lati” because we are referring to latissimus dorsi. The majors are pectoralis major and teres major. The proximal end of the humerus presents crests for its two tubercles, the greater and lesser. Inserting onto the crest of the greater tubercle is the pectoralis major. Inserting onto the crest of the lesser tubercle is the teres major. Latissimus dorsi inserts into the intertubercular groove between the two tubercles, hence “lady (lati) between two majors—one lady lying on the floor between two majors, with the floor being the floor of the bicipital groove or intertubercular sulcus.

4. What is SALSAP?
The axillary artery is the continuation of the subclavian artery as it passes the lateral edge of the first rib. It courses obliquely through the axilla behind the pectoralis minor, which divides it into three parts (as blood flows, before-behind-after the muscle for parts one-two-three, respectively). At the lower border of the teres major, it becomes the brachial artery. Of its six branches, the first comes from part one, two branches from part two, and three branches from part three (as easy as 1, 2, 3!). SALSAP reminds us of these six branches:

- Supreme thoracic
- Acromiothoracic (or thoracoacromial) trunk
- Lateral thoracic
- Subscapular
- Anterior circumflex humeral
- Posterior circumflex humeral

5. How do elephants serve as a memory tool?
An elephant has a trunk, and the thoracoacromial trunk is a true trunk—a short vessel that quickly divides into three or more branches. In addition, an elephant is a pachyderm, which can help you remember this arterial trunk’s four branches:

- Pectoral
- Acromial
- Clavicular
- Deltoid

6. What does B+B = A mean?
This formula describes the fact that, although there is a defined point where the axillary artery becomes the brachial artery (lower border of teres major), no such similar landmark exists at a point where the axillary vein begins. The origin and termination of blood vessels in a limb are always based on blood flow; therefore veins will begin distally and terminate proximally. Wherever a basilic vein (B) joins a brachial vein (B), the axillary vein (A) begins.
7. How can the arrangement of structures in the cubital fossa be remembered?
This triangular fossa in front of the elbow is bounded by the brachioradialis, pronator teres, and a
line through the humeral epicondyles. Within this fossa from lateral to medial are TAN:
- T—Tendon of biceps brachii as it inserts into the radius
- A—Artery, specifically the termination of the brachial artery as it bifurcates into the radial and
  ulnar arteries
- N—Nerve, the median nerve, which within the fossa gives rise to the anterior
  interosseous nerve
  The direction from lateral to medial, if forgotten, is recalled easily because the tendon and
  artery are both palpable, and feeling them will indicate the direction. The most medial structure is
  the median nerve; this is a common site for stimulating the median nerve in nerve conduction
  studies relative to carpal tunnel syndrome.

8. What is the area code for carpal country?
The number ("area code") 921 reminds us of the carpal canal contents:
- 9 tendons—four tendons from flexor digitorum profundus and four tendons from flexor
digitum superficiais, plus the lone tendon from flexor pollicis longus.
- 2 bursae—one large bursa called the ulnar bursa surrounds the eight digitorum tendons and
  is thus sometimes called the common synovial sheath. The smaller radial bursa surrounds only
  the flexor pollicis longus. Bursae are small fascial sacs elongated along tendons to minimize
  friction when the tendons slide.
- 1 nerve—median nerve; this is the nerve compressed in carpal tunnel syndrome.

9. Is it true that the most risqué mnemonics relate to the carpal bones?
The mnemonics are:
- Send Lucy To Paris To Tame Carnal Hunger
- Some Lovers Try Positions That They Can’t Handle
  The carpal bones are arranged in two rows of four bones. In the proximal row, from lateral to
  medial they are:
- Scaphoid
- Lunate
- Triangular (or Triquetral)
- Pisiform
  From lateral to medial in the distal row they are:
- Trapezium
- Trapezoid
- Capitate
- Hamate

10. Moving on to the thorax, if I go cruising in my VAN, where would I be?
The arteries, veins, and nerves of the thoracic wall share the name intercostal. The arteries branch
off the aorta, the veins return blood to the inferior vena cava via the azygous system of veins, and
the nerves are the ventral rami of the thoracic spinal nerves (although T7-T11 are properly called
thoracoabdominal nerves and T12 the subcostal nerve). As these structures course forward on
the thoracic wall, they occupy a groove at the lower edge of the rib called a costal groove. Within
this groove the structures from superior to inferior are in the VAN arrangement—Vein, Artery, Nerve.

11. Is LARP a radio station in California?
LARP refers to the twisting of the right and left vagus nerves as they course onto the esophagus
after passing the heart. The anterior and posterior vagal trunks come from the left and right vagi,
respectively; hence LARP—Left Anterior Right Posterior.

12. How many birds reside in the (thoracic) cage?
The thoracic wall, with its 24 ribs and sternum, has been likened to a birdcage. One can see inside
the cage through the ribs like one can view the inside of a birdcage. With a stretch of the imagination
and slight mispronunciation of the named structures, there are four birds of the thoracic cage.
Remember the duck lies between two gooses (azygos and esophagus).
- Esophagus, or esoph-a-goose
- Vagus (nerve), or va-goose
- Azygos (system of veins), or azy-goose
- Thoracic duct, or thoracic duck
13. What does the formula $S + S = P$ mean?
The large portal vein that carries nutrient-rich blood from the intestines to the liver is formed by two veins that both begin with “s.” Hence this formula states that when the splenic vein joins the superior mesenteric vein, the portal vein is formed.

14. What does SCALP tell you about the head and neck?
SCALP can be used to remember the scalp’s five layers, which from superficial to deep are:
- **Skin**—covered with hairs, the follicles of which extend to deeper layers.
- Close subcutaneous tissue—called “close” because of its tightness and the fact that it binds skin to the aponeurosis.
- Aponeurosis, specifically the galea aponeurotica—a flat tendon between the frontalis muscle in the forehead and the occipitalis muscle posteriorly (the term epicranius can be used for this entire layer).
- Loose subaponeurotic layer—layer of loose connective tissue that allows for the first three layers to move as a group. It is also called the “dangerous layer” because infections can spread through it.
- Pericranium—the periosteum on the outside of the cranial bone.

15. Is there an easy way to remember the terminal branches of the facial nerve?
Two Zebras Bit My Cat. The five terminal branches of the facial nerve originate from the facial plexus embedded within the parotid gland:
- Temporal—to muscles of the eye and forehead
- Zygomatic—to muscles of the eye and upper lip
- Buccal—to muscles of the cheek and upper lip
- Marginal mandibular—to muscles of the lower lip
- Cervical—to the neck muscle, platysma

16. What can help me remember the cranial nerves?
On Old Olympus’ Towering Top, A Finn And German Viewed Some Hops. This is a classic mnemonic for the 12 cranial nerves (usually indicated by Roman numerals), and they match up as follows:
- I. Olfactory, sensory to the nasal mucosa
- II. Optic, sensory to the eye
- III. Oculomotor, motor to the eye
- IV. Trochlear, motor to the superior oblique muscle
- V. Trigeminal, sensory and motor to the face through its three divisions (ophthalmic, maxillary, and mandibular)
- VI. Abducent, motor to the lateral rectus muscle
- VII. Facial, ends up in the parotid gland (see question 15)
- VIII. Auditory or acoustic (or vestibulocochlear), sensory to the ear
- IX. Glossopharyngeal, sensory to the tongue and motor to the stylo-phyaryngeus
- X. Vagus, sensory and parasympathetic to head, neck, thoracic, and abdomen
- XI. Spinal accessory, both sensory and motor to trapezius and sternomastoid muscles
- XII. Hypoglossal, motor to the tongue

Regarding fiber content, the 12 cranial nerves follow the saying, with $S \equiv$ sensory, $M \equiv$ motor, and $B \equiv$ both sensory and motor (again, the capital letters are the 12 nerves in sequence): Some Say Marry Money. But My Brother Say Marry Money, Bad Business (Some—Olfactor-Sensory, Say—Optic-Sensory, Marry—Oculomotor-Motor, etc.).

17. What is the formula for remembering the nerve supply to the seven muscles of the orbit?
For the cranial nerves to the muscles that move the eyeball, the formula is: $LR6(SO4)3$
The lateral rectus (LR) is supplied by the sixth nerve—abducens; the superior oblique (SO) by the fourth nerve—trochlear; and the remaining five muscles (superior rectus, medial rectus, inferior rectus, inferior oblique, and levator labii superioris) by the third nerve—oculomotor.

18. Are there any slick mnemonics for the back and lower limbs?
Not slick, but SLIC. The largest deep back muscle that is concerned with posture is termed the erector spinae or sacrospinalis. This muscle consists of three longitudinal columns of muscle that, from medial to lateral, are the spinalis, longissimus, and iliocostalis.
19. Is poetry ever used to assist in recall of anatomic facts?
Mnemonics can on occasion be in the form of poems. The intervertebral discs that separate vertebral bodies help bind the vertebral canal anteriorly. Each disc consists of the outer, tough fibrous annulus fibrosus and the inner, semigelatinous nucleus pulposus. Hence the poem:

Said the nucleus pulposus to the annulus fibrosus,
"Why do you hold me so tight?"
"If I didn’t, you would fall into the vertebral canal,
And then you would be out of sight.”

20. What does the phrase “say grace before tea” stand for?
The pes anserina (“foot of the goose”) on the medial side of the knee is formed by three tendons that insert from anterior to posterior in this order: sartorius, gracilis, and semitendinosus. This arrangement can be recalled by the letters in the mnemonic Say Grace before Tea for sartorius, gracilis, and semitendinosus. That is the order in which they insert proximally to distally as well as superficial to deep. “Before” reminds us that there is a bursa situated deep to the pes anserine and can be upset by muscles imbalances or direct trauma to the area.

21. Who are Tom, Dick, and A Very Nervous Harry?
On the medial side of the ankle lies the flexor retinaculum, which with the tarsal bones form the tarsal tunnel. Through this tunnel will pass three tendons (tibialis posterior, flexor digitorum longus, and the flexor hallucis longus), change of order and vessels and nerves (posterior tibial artery and tibial nerve) that can be recalled by Tom, Dick, and Harry. The association from anterior to posterior is Tibialis posterior, flexor Digitorum longus, posterior tibial Artery and Vein, tibial Nerve, and flexor Hallucis longus, respectively.

22. What are the branches of the brachial plexus from lateral to medial? Remember, “My Aunt Ravaged My Uncle.”
- Musculocutaneous
- Axillary
- Radial
- Median
- Ulnar

23. What nerve roots comprise the long thoracic nerve that innervates the serratus anterior?
C5, C6, and C7—raise your arms to heaven.

24. What is the innervation of the pectoral muscles? Remember, “lateral is less and medial is more.”
The lateral pectoral nerve innervates the pectoralis major only, and the medial pectoral nerve innervates both the pectoralis major and pectoralis minor. Remember, these are named for the cord from which they are derived.

25. How do you remember the results of peroneal and tibial nerve injury? Remember “PED and TIP.”
- Peroneal—everts and dorsiflexes; loss = drop foot
- Tibial—inverts and plantar flexes; loss = cannot walk on TIP toes

26. What is the relationship of the suprascapular artery and nerve at the suprascapular notch?
The Army (artery) travels over the bridge, and the Navy (nerve) travels under.

27. Remembering the formation of the brachial plexus:
Rod Taylor Drinks Cold Beers. Corresponding with Roots, Trunks, Divisions, Cords, Branches.
- Roots – C5, C6, C7, C8, and T1, C5, and C6 come together and form the upper trunk.
- C7 stays by himself and forms the middle trunk.

C8 and T1 come together and form the lower trunk.
Hence we have three Trunks, each of which gives off anterior and posterior divisions. Anterior division of the upper trunk and the anterior division of the middle trunk form the lateral cord. Posterior divisions of all trunks form the posterior cord, as it is located posterior to the axillary artery.
Anterior division of the lower trunk continues and forms the medial cord. So now we have lateral, posterior, and medial cords as they relate to the axillary artery. Branches of the brachial plexus are classified as supraclavicular and infraclavicular. Supraclavicular branches arise off the roots and trunks. Infraclavicular branches arise off the cords.

Rod Taylor, Australian actor, played male lead role in Alfred Hitchcock’s “The Birds.”

28. Remembering the infraclavicular branches arising off the brachial plexus:
Let the Marine Corp ARM U
- Lateral pectoral nerve – off lateral cord
- Marine Corp = Musculocutaneous nerve – off the lateral cord
- A = Axillary nerve – off the posterior cord
- R = Radial nerve – off or a continuation of the posterior cord
- U = Ulna nerve – off or a continuation of the medial cord

29. Remembering nerve supply to sternocleidomastoid and trapezius:
- Trapezius is so named because of its shape, roughly diamond shaped.
- Diamonds are a girl’s best friend and make great accessories.
- These accessories are continually checked on an outstretched hand and by a glancing look with a turn of the head.
- A main head turning muscle is sternocleidomastoid.
- So there you have an easy way to remember that both sternocleidomastoid and trapezius are supplied by the accessory nerve.

30. You would rather eat lamb before mutton.
This reminds us about the superficial to deep arrangement of the adductor muscles of the thigh.
- Lamb = Adductor Longus – most superficial
- Before = Adductor Brevis – in between
- Mutton = Adductor Magnus – deepest
We know that these muscle are supplied by branches of the obturator nerve.
The obturator nerve divides into anterior and posterior branches about the obturator foramen.
The anterior branch passes between the adductor longus and adductor brevis.
The posterior branch passes between the adductor brevis and adductor magnus.
- So we can see that adductor brevis is in the prime position to be supplied by both anterior and posterior branches of the obturator nerve.
- Although the longus is supplied by the anterior branch, it is the posterior branch of the obturator nerve that supplies the magnus, along with the tibial division of the sciatic nerve.

31. Remembering the actions of palmer and dorsal interossei:
- Palmer interossei ADDuct = PADs
- Dorsal interossei ABDuct = DABs

32. Remembering the segmental innervation of sciatic, tibial, and common fibular (peroneal) nerves:
The sacral plexus arises from the spinal nerve levels L4, L5, S1, S2, and S3. Consider it as an elevator with the top floor being L4 and the ground floor being S3.
The common fibular nerve (CFN) supplies the anterior leg group (deep branch of CFN), and the lateral leg group (superficial branch of CFN). So if we consider that the anterior leg muscles insert onto the dorsal surface of the foot, then the nerve stops one level above ground, so the common fibular nerve arises from L4, L5, S1, and S2.
The tibial nerve goes all the way into the plantar aspect of the foot, where it divides into the medial and plantar nerves. So this nerve goes to the ground floor and uses ALL levels that form the sacral plexus L4, L5, S1, S2, and S3.

33. Remember AchilleS only had one weak spot.
S1 is the level that is tested with an Achilles tendon reflex test.
CHAPTER 31 QUESTIONS

1. What nerve roots innervate the serratus anterior?
   a. C4,5,6
   b. C5,6,7
   c. C6,7,8
   d. C7, 8, T1

2. What is action of the palmar interossei?
   a. adduction
   b. abduction
   c. opposition
   d. circumduction

3. The spinal level evaluated by the Achilles reflex is?
   a. L3
   b. L4
   c. L5
   d. S1
1. What are the dietary guidelines for Americans?
Concerned by the escalating incidence of obesity and related health issues in the United States, the Department of Agriculture and the Department of Health and Human Services created a set of recommendations designed to promote general health. These recommendations may be applied to anyone in the general population over the age of two. Adoption of these guidelines hopefully will improve overall health by promoting healthy body weight, reducing the incidence of type 2 diabetes, and reducing the risk of cardiovascular disease. Major guidelines include:
1. Balance calories with activity to manage weight
2. Choose more whole grains
3. Reduce sodium intake by choosing fewer processed foods
4. Reduce intake of foods containing solid fat
5. Reduce cholesterol intake
6. Increase fruit and vegetable intake
7. Consume low fat or reduced fat dairy products
8. Choose lean meats as a protein source
9. Increase physical activity
10. If alcohol is consumed it should be consumed in moderation

2. Describe the Ornish low-fat diet. What does it claim to do?
The Ornish diet is a vegetarian diet based mainly on vegetables, fruits, whole grains, and beans. No animal products are eaten except moderate amounts of egg whites and nonfat dairy. It consists of 10% fat, mainly polyunsaturated fat and monounsaturated fat; 70% to 75% carbohydrates, mainly complex; 15% to 20% protein; and 5 mg cholesterol per day. According to Ornish, people lose weight on his diet for several reasons: 1) it takes more calories to metabolize complex carbohydrates than simple carbohydrates; 2) metabolic rate may increase on the diet and; 3) people consume fewer calories when eating complex carbohydrates because they are more filling. Meat and animal products contain protein, but they also contain saturated fats and cholesterol. Ornish claims that his diet is the most effective diet for lowering cholesterol, preventing heart disease, reducing symptoms of type 2 diabetes, and decreasing the risk of developing many cancers.

3. What are the possible problems that may result from being on a very low-fat diet?
Very low-fat diets, (approximately 16 gm fat, 10% of calories from fat) may lead to insufficient amounts of essential fatty acids. Individuals with low HDL, high triglyceride, and high insulin levels may have these abnormalities amplified with these diets. Some studies have found these diets to be low in vitamins E, B₁₂, and zinc, but these reports are inconsistent.

4. Briefly describe the Atkins diet. What does it claim to do?
The Atkins diet is a low-carbohydrate, high-protein, ketotic diet, divided into four stages. The most restrictive stage limits carbohydrate consumption to 20 gm/day. Other stages allow between 25 and 90 gm per day. Most nutritionists recommend about 300 gm/day. The diet does not restrict protein, fat, or calories, but many dieters have suppressed appetite and decrease their caloric intake. Several dietary supplements are included, such as vitamins and minerals, especially antioxidants, trace minerals, and essential fatty acids.
Atkins claims that his diet mobilizes fat more than any other diet, is the easiest diet for maintenance of weight loss, and is a high-energy diet that makes people feel good. He believes that most obesity is caused by metabolic imbalances from carbohydrate consumption.

5. According to most traditional nutritional professionals, why do high-protein and high fat diets cause weight loss?
Fewer calories are consumed on high protein and high fat diets because proteins and fats are more filling than simple carbohydrates. The fewer calories you consume, the more weight you lose. Much of
the initial weight loss is from water loss from natriuresis. Additional water loss occurs when glycogen is converted to glucose. This conversion must occur to maintain blood sugar levels. In subsequent weeks, weight loss is from body fat, at a rate of 1 to 2 lbs per week. This rate is similar to that obtained with other types of low caloric diets.

6. What are the possible side effects of a high protein, high fat diet?
Some authors report few side effects of a high protein, high fat diet, although others report several significant side effects. The following side effects have been reported by some authors: High-protein, high-fat diets cause the liver and kidneys to work harder to metabolize and excrete excessive nitrogen. This may result in organ failure. Excessive water loss may result in dehydration and orthostatic hypotension. Dosage of certain medications may need to be adjusted to compensate for diuresis. There may be an increased risk of osteoporosis caused by calcium loss that occurs with excess water loss. Evidence suggests that high-protein diets are associated with certain cancers and heart disease. Vitamins and minerals found in carbohydrates may be deficient unless supplements are taken. Lowered glycogen stores may cause problems for long-distance runners.

7. Is there a difference in the adherence rates between the Atkins, Ornish, and Weight Watchers?
Not many studies have compared adherence rates of various diets. Dansinger et al have shown no significant difference in the adherence rates of the more extreme Atkins and Ornish diets compared with the moderate Weight Watchers diet after 1 year, but that there was a trend toward better adherence in the moderate diet. The average adherence rate for all of the diets combined was only 58%. This rather low adherence rate is the major problem for lack of long-term success with all of these diets.

8. When would the recommendation for bariatric surgery be appropriate?
Bariatric surgery promotes rapid weight loss in individuals who have been unsuccessful in weight loss attempts and who are morbidly obese (body mass index [BMI] > 40). Individuals with a BMI of 35 or greater with comorbidities such as type 2 diabetes, hypertension, heart disease, or sleep apnea would also be candidates for surgery. The gastric bypass procedure entails stapling off the stomach to a quarter cup pouch and attaching the jejunum to the pouch. The lap band procedure places a ring around the stomach making it smaller. Two thirds of the stomach is surgically removed in the gastric sleeve procedure. In each case stomach capacity is significantly reduced resulting in limited calorie intake. Weight loss may be significant during the first year after the surgery. Potential complications would include hernia risk, numerous vitamin and mineral deficiencies, and malabsorption.

9. What does a typical American diet consist of?
A typical American diet consists of 35% fat, 50% carbohydrates, and 15% protein.

10. What type of diet is most effective for long-term weight loss?
There are widely varying opinions on which diet is most effective for long-term weight loss. Most scientifically controlled studies indicate diets that reduce caloric intake are most effective for long term weight loss and body fat reduction regardless of the macronutrient composition. Dansinger et al found no significant difference in weight loss after 1 year between individuals on the Atkins, Ornish, or Weight Watchers diets. Weight loss will occur if the number of calories consumed is less than the number of calories expended. For most people the caloric deficit should be about 1000 kcal per day. If physical activity is not increased a diet of approximately 1400–1500 kcal/d seems to be optimal.

11. Why lower sodium intake?
Research has shown that sodium consumption increases fluid retention in the body. This added fluid increases blood pressure resulting in damaged blood vessel walls. The damage promotes atherosclerosis leading to a heart attack or stroke. Recommended sodium intake is 1500 to 2300 mg/day. One teaspoon of salt contains 2300 mg of sodium. The average adult’s consumption is well above the recommendation. Incorporating more fresh foods in the diet; eating out less often; and cutting back on processed foods will lower sodium intake.

12. Does soy protein decrease the risk of developing cardiovascular disease?
A meta-analysis concluded that consumption of soy protein in place of animal protein significantly lowers blood levels of total cholesterol, LDL, and triglycerides without affecting HDL. This is especially true in subjects with baseline cholesterol levels greater than 240 mg/dL. The FDA has approved labeling of food that contains greater than 6.25 g of soy protein per serving and to state that these foods reduce the risk of heart disease, assuming 25 g of soy protein intake daily.
13. Do antioxidant supplements decrease the risk of developing cardiovascular disease?
There is insufficient evidence for recommending the use of antioxidant supplements for decreasing the
risk of developing cardiovascular disease. Observational studies involving consumption of foods rich
in vitamin E have shown an association with lower disease risk. Similar studies using foods rich in
vitamin C have not been as consistent. However direct evidence that the decrease in disease was as
a result of antioxidant activity has not been shown for either vitamin. A few observational studies
using vitamin E supplements have reported inconsistent results. No randomized trial studies have been
done. Trials using β-carotene supplements have not shown any benefits and in some cases caused
increased risk of cancer.

14. Do folic acid, vitamin B₁₂, and vitamin B₁₂ decrease the risk of developing
cardiovascular disease?
Case-control and prospective studies have shown that lower levels of folic acid and vitamin B₉ have
been associated with coronary artery disease, but that low levels of vitamin B₁₂ have not been associated
with vascular disease. However randomized trial studies have not been done to determine a cause
and effect relationship between high folic acid and vitamin B₉ and decreased risk of cardiovascular
disease.

15. Do omega-3 fatty acids alter mortality rate, incidence of a cardiovascular events,
or cancer?
Several studies have reported beneficial effects of increased omega-3 fatty acid intake in patients
with coronary artery disease, including reduction in plasma triglyceride levels and a decrease in mortality
rates. However meta-analysis of several randomized control trials found no clear evidence that
dietary or supplemental omega-3 fatty acids from fish or plants alter mortality, cardiovascular events,
or cancers in individuals with cardiovascular disease, those at high risk of developing cardiovascular
disease, or the general population. These analyses also found no increased risks in mortality, cancer,
or stroke as a result of taking omega-3 supplements or increased omega-3 fatty acids in the diet.
Individuals who have previously had a myocardial infarction are therefore encouraged to consume
more omega-3 fatty acids. But people who have angina, but no previous myocardial infarction, and
the general public, are not advised to increase their consumption of omega-3 fatty acids.

16. Do folate supplements decrease the incidence of neural tube defects?
Yes, there is a significant reduction in the incidence of neural tube defects when folate supplements
are taken before and during the first 2 months of pregnancy.

17. Do folic acid supplements with or without vitamin B₁₂ supplements improve
cognitive function or mood?
Although studies are limited, there is no evidence that folic acid with or without vitamin B₁₂ improves
cognitive function or mood in normal or cognitively impaired older adults. Folic acid with vitamin B₁₂
has been shown to reduce serum levels of the amino acid homocysteine. Elevated homocysteine has
been linked to an increased risk of developing dementia.

18. What are the health benefits of adding fiber to the diet?
There are two forms of fiber that provide significant health benefits when incorporated into the diet.
The first, insoluble fiber, promotes regularity. Found in whole grains, bran, brown rice, and the peelings
of fruit and vegetables, it initially creates a sense of fullness (satiety). Feeling full should reduce total
caloric intake and insoluble fiber may facilitate the weight loss process. It also keeps water in the colon
thus reducing the risk of constipation and diverticulitis. The second, soluble fiber, promotes heart health
by lowering cholesterol. Found in oats, flax seed, and fruit it also creates a sense of satiety. It too
may play a role in weight reduction. Adults should incorporate 25 to 38 g of fiber into their daily diet.

19. Do calcium supplements increase bone density in postmenopausal women?
Calcium supplements appear to increase bone density between 1.6% and 2%. There is a trend toward
reduction in vertebral fractures associated with this increase, but the evidence is not clear regarding
a reduction in nonvertebral fractures.

20. A Mediterranean diet may be helpful in managing which medical condition?
To promote heart health and reduce the risk of cardiovascular disease many health care providers
recommend the Mediterranean diet. Consumption of fresh fruits, fresh vegetables, and whole grains
are the centerpiece of the diet. Red meat and high fat dairy is limited, but seafood is encouraged. Nuts are
a daily source of protein and fat. Food preparation utilizes olive or canola oil along with spices in place
of salt. Baking, broiling, and grilling are preferred cooking techniques. Red wine consumption, a component of the diet, has also demonstrated heart health benefits.

21. How should the daily recommended percentages of carbohydrate, fat, and protein intake be altered during heavy training?
In a training athlete, the percentage of carbohydrates should be higher, the percentage of fats should be lower, and the percentage of protein should be the same as for a sedentary person. Carbohydrates are the primary nutrient used during prolonged, moderate-to-high intensity exercise.

22. Should athletes consume additional protein when they are in training?
The current recommended daily allowance for protein in sedentary people is 0.8 gm protein/kg body weight per day. Several investigators have shown that athletes require more protein. Recommended amounts range from 1.2–1.8 gm/kg per day for aerobic and resistance training athletes. People just beginning an exercise program should use the upper end of this range. Because the average North American diet consists of 1.9 gm/kg per day, additional protein usually is not necessary.

23. Does carbohydrate consumption affect the amount of muscle growth?
Yes, carbohydrate consumption causes an increase in the release of insulin, which stimulates muscle synthesis. Testosterone levels, which also stimulate muscle synthesis, appear to be highest when the ratio of carbohydrate to protein intake is 4:1. Maximal muscle growth seems to occur when protein intake is 1.7–1.8 gm protein/kg body weight a day, energy intake is sufficient to prevent weight loss, and carbohydrate intake is 60% to 65% of nutrient intake. Consuming a carbohydrate with protein beverage after resistance exercise may enhance recovery or reduce muscle breakdown.

24. What is the primary factor that determines whether carbohydrates, fats, or proteins are metabolized during a bout of exercise?
The availability of oxygen is the main factor that determines whether fats or carbohydrates are metabolized. The more limited the supply of oxygen, the more carbohydrates will be metabolized. Less oxygen is needed for carbohydrate metabolism than for fat metabolism. More calories per liter of oxygen are produced from carbohydrates, and oxidation of carbohydrates occurs more quickly. Therefore during high intensity exercise carbohydrates are the prominent fuel source. As exercise intensity decreases, oxygen becomes more readily available, carbohydrate metabolism decreases, and fat metabolism increases. However, duration of exercise also contributes to the type of fuel used. The longer the duration of exercise, the greater the contribution of fat. Under normal circumstances proteins provide only 5% to 10% of the fuel source during exercise. The contribution is directly proportional to the intensity and duration of exercise. The increase in protein utilization with prolonged exercise seems to be related to glycogen stores. As glycogen stores are depleted, the body depends more on protein for energy production.

25. Do creatine supplements improve an athlete's performance?
Most studies agree that creatine supplements are beneficial for short-duration, repetitive bursts of intense exercise. Kreider has shown that short-term creatine supplementation (15–25 gm/day for 5 to 7 days) improves maximal power and strength by 5% to 15%, work performed during sets of maximal effort muscle contractions by 5% to 15%, single-effort sprint performance by 1% to 5%, and work performed during repetitive sprint performance by 5% to 15%. Long-term supplementation (15–25 gm/day for 5 to 7 days and 2–25 gm/day for 7 to 84 days) also results in significantly greater gains in strength, sprint performance, and fat-free mass. The most popular dosage is a loading phase of 0.3 gm/kg per day for 5 to 7 days and a maintenance dose of 0.03 gm/kg per day. Creatine supplements do not appear to improve longer-duration, aerobic exercise performance.

26. What are the side effects of creatine supplementation?
The only negative side effect reported in scientific studies is weight gain. When creatine supplements are taken, endogenous synthesis decreases; it returns when creatine is removed from the diet. Supplements may increase stress on the liver and kidneys, but this theory has not been confirmed. Anecdotal evidence suggests an increased incidence of muscle cramps and strains, minor gastrointestinal distress, and nausea, but no scientific studies validate such reports. Further research clearly is needed.

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**CHAPTER 32 QUESTIONS**

1. Which of the following is a potential health benefit attributed to adding soluble fiber to the diet?
   a. Improvement in gutt symptoms
   b. Improved kidney function
   c. Reduced serum sodium levels
   d. Reduced serum cholesterol levels
2. Adherence to a Mediterranean diet would incorporate which of the following fats in the diet?
   a. Butter
   b. Olive oil
   c. Corn oil
   d. Coconut oil

3. An individual engaged in heavy training for an endurance event should increase the percentage of which nutrient in his/her diet?
   a. Carbohydrate
   b. Cholesterol
   c. Fat
   d. Protein
1. What is dry needling (DN)?
DN is a skilled intervention using a thin filiform needle to penetrate the skin and stimulate underlying myofascial trigger points and muscular and connective tissues for the management of neuromusculoskeletal pain and movement impairments.

2. How does DN differ from wet needling?
DN and wet needling differ primarily in the type of needle used and also the intention of the insertion of the needle. DN utilizes a solid filament needle to stimulate neuromuscular tissue, and wet needling utilizes a hollow hypodermic needle to inject pain relievers, corticosteroids, or Botox into neuromuscular tissue. Needles utilized for DN are typically much smaller than hypodermic needles and are referred to in terms of diameter and length rather than “gauge.” They are solid rather than hollow and have a rounded rather than the beveled, cutting-edge tip of a hypodermic needle and so are generally more comfortable and carry less risk of introducing infection.

3. What is the difference between DN and acupuncture?
This is quite likely one of the most frequently asked questions. Both acupuncture and DN use a solid filament needle; however, the uses of a solid filament needle for acupuncture and DN are very different. The differences are evident in the evaluative tools used by the practitioner, the assessment, the application, and the overall intended goal. Acupuncture is a treatment based on Eastern medical diagnosis requiring training in traditional Chinese medicine (TCM). The TCM practitioner inserts needles into specific points that lie along meridians (channels) of the body through which the life force, or “qi” (pronounced “chee”), flows. The overall goal of TCM acupuncture is to restore normal flow of the life force. In contrast, DN is rooted in Western medical philosophy. DN revolves around a practitioner using a thin filiform needle to penetrate the skin and stimulate underlying myofascial trigger points and muscular and connective tissues for the management of neuromusculoskeletal pain and movement impairments. Although these two practices are distinctly different, research suggests there is correlation between acupuncture and trigger points (approximately 20%).

4. What are the proposed theories of how DN works?
   Increased blood flow
   - Needling of the trapezius and Achilles tendon results in increased local blood flow to the needled site.
   Decreased banding
   - It is hypothesized that following DN there is decreased banding of the target tissue and a restoration of sarcomere length and endomysium spacing.
Decreased spontaneous electrical activity
- Eliciting a local twitch response of a trigger point results in decreased electrical activity at that site. This change in electrical activity may reflect a normalization of the neuromuscular junction.

Biochemical changes
- Following DN of an active trigger point, elevated levels of hydrogen ions, neurotransmitters (bradykinin, 5HT, NE, CGRP, and substance P), cytokines, and chemokines (TNF-α, IL-1b, IL-6, and IL-8) are reduced locally and remotely.

Pain gate
- It is hypothesized that sensory and proprioceptive stimuli from needling may also drive the gate control mechanisms of pain reduction.

5. What is the current evidence regarding the effectiveness of DN?
Numerous studies indicate support for DN in cases of myofascial pain syndromes, spasticity as a result of stroke, shoulder pain, lateral epicondylalgia, heel pain, temporomandibular dysfunction, and pain.

6. What is a trigger point?
Travell and Simons define a trigger point as a hyperirritable spot in a taut band of skeletal muscle or the fascia that is painful upon compression and produces characteristic pain, referred tenderness, motor dysfunction, and/or autonomic phenomena. This is also referred to as an active trigger point. A latent trigger point has all the components of an active trigger point but does not produce referred pain. Trigger points have been found to exist in an acidic environment. Nociceptive sensitizing agents and abnormal spontaneous electrical activity are found in the region of a trigger point, which are correlated with lower pain pressure thresholds.

7. What is a local twitch response (LTR)?
When firm mechanical pressure (via palpation) on or a needle is introduced to a trigger point, a visual twitch (contraction) of the trigger point will occur. The LTR is easily visualized and it appears as a quick twitch or dimpling of the skin overlying the muscle the trigger point resided within. An LTR is a spinally mediated reflexive twitch of that muscle fiber.

8. What is the reliability of finding a TP?
Interrater reliability of identifying trigger points varies from study to study. Generally speaking, at this time, interrater reliability of trigger point identification is poor. However, specific training of practitioners can improve interrater reliability.

9. What are the current practice requirements for a physical therapist to perform DN?
Practice requirements vary from state to state. Currently there are 6 states in which dry needling by a physical therapist is prohibited and 27 states in which the practice act or the Physical Therapy Board specifically allows it. In the remainder of the states there has not yet been a determination regarding physical therapists performing DN. Requisites for training/competency vary from state to state.

10. What is the incidence of adverse events (AEs) when performing DN?
A recent study reported that of 7629 DN interventions performed, 1463 (19%) AEs occurred. No significant AEs were reported. The following mild AEs were reported along with their corresponding percentage. Common AEs included bruising (7.55%), bleeding (4.65%), pain during treatment (3.01%), and pain after treatment (2.19%). Uncommon AEs were aggravation of symptoms (0.88%), drowsiness (0.26%), headache (0.14%), and nausea (0.13%). Rare AEs were fatigue (0.04%), altered emotions (0.04%), shaking, itching, claustrophobia, and numbness, all 0.01%.

11. What are the precautions to TDN?
- Needle phobia/aversion
- Significant cognitive impairment
- Communication barrier
- History of traumatic or spontaneous pneumothorax
- Hyperalgesia or allodynia
- Local skin lesions or infections
- Compromised immune system
- Metal allergies
- Abnormal bleeding tendency
- Vascular disease
12. What are the absolute contraindications to TDN?
Consent denied by patient
Inadequate knowledge of the practitioner (lack of training or proper training)
Compromised equipment
First trimester of pregnancy
Scalp area of infants
Nipples, umbilicus, and external genitalia
Uncontrolled anticoagulant usage
Local infection, skin lesion, or active tumor
Occipital region with Arnold-Chiari malformation
Over a cardiac pacemaker

13. Describe a general protocol when performing TDN.
a. Examination and identification of neuromuscular impairments
b. Absence of contraindications
c. Consent of patient disclosure of potential adverse effects
d. Adherence to clean needle technique and universal precautions (ie, donning of gloves, cleaning of skin, and single-use filiform needles)
e. Identification of the trigger point
f. Direct needling of the trigger point to elicit a twitch response (ie, various techniques exist [piston motion, etc.] and are at the discretion of the practitioner)
g. Removal and proper disposal of needle; check patient for adverse effects
h. Reevaluation of the patient’s impairments to determine effect of needling
i. Introduction of other necessary therapeutic interventions such as manual therapy, therapeutic, exercise, or modalities that would be indicated to further reduce the patient’s impairments and improve function

14. What types of needles/supplies are required to perform TDN?
Required supplies include:
- Sterile, single-use solid filiform needles of varying lengths and diameters; as the length increases so should the diameter for improved needle control. Lengths vary from 30 to 120 mm in length and 0.20 to 0.50 in diameter.
- Isopropyl alcohol, alcohol swab, or antiviral/antibacterial to clean skin of patient
- Firm-fitting treatment gloves
- Sharps container for disposal of used needles
- Recommended but not required: electrical stimulation unit that can be modified for motor or sensory TENS and applied through the needles

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CHAPTER 33 QUESTIONS

1. Which of the following AEs is most common after DN?
   a. Bruising
   b. Pain after treatment
   c. Headache
   d. Nausea

2. What is the interrater reliability of trigger point identification?
   a. Poor
   b. Fair
   c. Good
   d. Excellent

3. Which of the following is an absolute contraindication to dry needling?
   a. Hyperalgesia
   b. First trimester of pregnancy
   c. Radicular pain beyond the elbow or knee
   d. Needle phobia
1. What do isokinetic devices do?
Isokinetic devices provide a resistance that accommodates the torque (force multiplied by perpendicular distance) applied by an individual to maintain a constant, preselected angular velocity.

2. What are the advantages of isokinetic devices?
- Because of the accommodating resistance, a muscle can be challenged to its maximal capacity through an entire range of motion (physiologic Blix curve).
- Muscle groups can be isolated for testing and exercising.
- Resistance that accommodates to pain and fatigue provides an inherent safety factor.
- Reliable objective data may be obtained for documentation.
- Exercise is possible at different angular velocities through a velocity spectrum.
- It is possible to train at high angular velocities to increase muscle power, quickness of muscle force development, time rate of torque development, or torque acceleration energy. These muscle performance characteristics are important for functional activities.
- Computerized feedback allows an exerciser to improve torque control accuracy.
- The reciprocal innervation time of agonist and antagonist muscle contractions can be decreased.
- Joint compressive forces decrease with higher angular velocities; Bernoulli’s principle states that the faster the movement of a surface (articular surface) over a fluid (synovial fluid), the lower the surface pressure.
- There is a 30-deg/sec physiologic (strengthening) overflow to slower angular velocities with isokinetic resistance.
- There is a 30-degree range of motion strengthening overflow during performance of short-arc exercises.
- Real-time feedback is available to the patient for motivation during exercise.

3. What are the contraindications to isokinetic testing or exercising?

**ABSOLUTE CONTRAINDICATIONS**
- Acute strain (musculotendinous unit) or sprain (noncontractile tissue)
- Soft-tissue healing constraints (eg, immediately after surgery)
- Severe pain
- Extremely limited range of motion (ROM)
- Severe effusion
- Joint instability

**RELATIVE CONTRAINDICATIONS**
- Subacute strain or chronic third-degree sprain
- Pain
- Partially limited ROM
- Effusion
- Joint laxity

4. When is it safe to perform isokinetic testing after surgical repair?
Generally, isokinetic testing may be performed safely when postsurgical soft tissue healing is complete. The table lists approximate healing times and the recommended testing criteria for several common surgical repairs. However, we strongly recommend consulting with the referring surgeon to establish testing criteria.
5. What parameters are commonly used for the assessment of isokinetic data?

- Peak torque—maximal torque value on the parabolic torque curve (A)
- Angle-specific torque—torque value at a specific point (angle) in the range of motion (B)
- Time rate of torque development (TRTD) to peak torque—elapsed time from the onset of torque production to the peak torque (C)
- TRTD to a predetermined torque value—elapsed time from the onset of torque production to a predetermined level of torque (D)
- TRTD to a specific point in the ROM—elapsed time to reach a specific point in the ROM
- Torque acceleration energy (TAE)—total work performed in the first 0.125 sec; a measure of the "explosiveness" of a muscle contraction
- ROM—measured in degrees with an electrogoniometer
- Reciprocal innervation time—time interval from cessation of the agonistic contraction to the initiation of the antagonistic contraction (E)
- Force decay rate—downslope of the torque curve; in general, the downslope should be straight or convex; if it is concave, the patient probably had difficulty with producing torque at the end ROM (F)
- Total work—total volume of work under the torque curve, regardless of speed, range of motion, or time (G)
- Average power—total work divided by the work time

6. How are isokinetic data commonly interpreted and analyzed?

- Bilateral comparison—analysis of torque values of one extremity relative to the other extremity; probably the most common comparison; differences >10% to 15% indicate significant asymmetry
- Unilateral ratios—comparison of agonist and antagonist muscle torque; this measure is particularly important to assess with velocity spectrum testing because the ratios change through different angular velocities in many muscle groups
- Torque to body weight—analysis of torque values relative to body weight; used to normalize muscle performance relative to size
- Total leg strength (TLS)/total arm strength (TAS)—summation of torque values for individual components of the leg or arm, respectively
- Comparison with normative data—analysis of torque values relative to published normative data for specific populations

Approximate Healing Times and Testing Criteria for Common Surgical Repairs

<table>
<thead>
<tr>
<th>SURGICAL REPAIR</th>
<th>APPROXIMATE HEALING TIME (WEEKS)</th>
<th>TESTING CRITERIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACL (patellar tendon graft)</td>
<td>8–12</td>
<td>Full AROM; KT scores WNL</td>
</tr>
<tr>
<td>ACL (semitendinosus graft)</td>
<td>12–16</td>
<td>Full AROM; KT scores WNL</td>
</tr>
<tr>
<td>PCL</td>
<td>12</td>
<td>Full AROM; KT scores WNL</td>
</tr>
<tr>
<td>Rotator cuff tear</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Small tear</td>
<td>12–16</td>
<td>Full AROM; healthy tissue</td>
</tr>
<tr>
<td>Medium tear</td>
<td>16–24</td>
<td>Full AROM; healthy tissue</td>
</tr>
<tr>
<td>Large tear</td>
<td>24</td>
<td>Full AROM; healthy tissue</td>
</tr>
<tr>
<td>Capsular shift</td>
<td>12</td>
<td>Full AROM</td>
</tr>
<tr>
<td>Lateral ankle reconstruction</td>
<td>8–12</td>
<td>Full AROM</td>
</tr>
<tr>
<td>Achilles tendon rupture</td>
<td>16–24</td>
<td>Full AROM</td>
</tr>
</tbody>
</table>

ACL, Anterior cruciate ligament; PCL, posterior cruciate ligament; AROM, active range of motion; WNL, within normal limits; KT, KT-1000 knee ligament arthrometer (MEDmetric Corp, San Diego, Calif.).
7. **Describe the evaluation of isokinetic data relative to normative data.**

Descriptive normative data for different populations may be used as another guideline for testing and rehabilitation. The table provides descriptive normative data for peak torques relative to body weight and unilateral agonist/antagonist ratios for several commonly tested muscle pairs. Normative data are particularly useful when a patient has bilateral injuries and bilateral comparison is not a useful measure. Examples of commonly used normative data are also included in the table.

### Normative Test Data on Cybex

<table>
<thead>
<tr>
<th>SPEED (DEGREES/SEC)</th>
<th>60</th>
<th>180</th>
<th>300</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Shoulder (modified neutral)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>External rotation (% BWT)</td>
<td>13</td>
<td>10</td>
<td>6</td>
</tr>
<tr>
<td>Internal rotation (% BWT)</td>
<td>22</td>
<td>18</td>
<td>14</td>
</tr>
<tr>
<td>ER/IR ratio (%)</td>
<td>59</td>
<td>56</td>
<td>43</td>
</tr>
<tr>
<td>Shoulder (modified neutral)</td>
<td>60</td>
<td>180</td>
<td>300</td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>External rotation (% BWT)</td>
<td>9</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Internal rotation (% BWT)</td>
<td>15</td>
<td>12</td>
<td>9</td>
</tr>
<tr>
<td>ER/IR ratio (%)</td>
<td>60</td>
<td>50</td>
<td>33</td>
</tr>
<tr>
<td><strong>Shoulder 90/90</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>External rotation (% BWT)</td>
<td>15–20</td>
<td>13–18</td>
<td>11–16</td>
</tr>
<tr>
<td>Internal rotation (% BWT)</td>
<td>25–30</td>
<td>22–27</td>
<td>19–24</td>
</tr>
<tr>
<td>ER/IR ratio (%)</td>
<td>60–69</td>
<td>60–69</td>
<td>60–69</td>
</tr>
</tbody>
</table>

Continued on following page
8. Is there value in using isokinetic testing to document strength and power in patients with ACL injuries before surgery?

Eitzen et al. performed objective isokinetic testing for preoperative ACL reconstructions and found it to demonstrate that if a patient has a 20% quadriceps deficit before the surgery, then he or she will have a residual deficit for up to 2 years postoperatively. Therefore this emphasizes the importance of prehabilitation for patients with ACL injuries and the importance of objective documentation using isokinetic testing.

9. Discuss the correlation between isokinetic testing and manual muscle testing.

Wilk and Andrews compared the results of knee extension manual muscle testing (MMT) and isokinetic open kinetic chain (OKC) knee extension/flexion in 175 patients after knee arthroscopy. All 175 patients had normal MMT scores, but isokinetic testing revealed bilateral deficits in 21% at 180 deg/sec and 16% at 300 deg/sec. Ellenbecker reported bilateral deficits of the shoulder internal and external rotators ranging from 13% to 28% with isokinetic testing in subjects with normal-grade (5/5) MMT. Isokinetic devices can measure subtle differences in strength that may not be evident with MMT.

### Normative Test Data on Cybex (Continued)

<table>
<thead>
<tr>
<th>SPEED (DEGREES/SEC)</th>
<th>60</th>
<th>180</th>
<th>300</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Knee extension/flexion</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quadriceps (% BWT)</td>
<td>100</td>
<td>75</td>
<td>50</td>
</tr>
<tr>
<td>Hamstrings (% BWT)</td>
<td>60–69</td>
<td>35–47</td>
<td>25–37</td>
</tr>
<tr>
<td>Quadriceps/hamstring ratio (%)</td>
<td>60–69</td>
<td>70–79</td>
<td>85–95</td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quadriceps (% BWT)</td>
<td>90</td>
<td>65</td>
<td>40</td>
</tr>
<tr>
<td>Hamstrings (% BWT)</td>
<td>60</td>
<td>35</td>
<td>25</td>
</tr>
<tr>
<td>Quadriceps/hamstring ratio (%)</td>
<td>60–69</td>
<td>70–79</td>
<td>85–95</td>
</tr>
<tr>
<td><strong>Ankle inversion/eversion</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inversion (% BWT)</td>
<td>12</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Eversion (% BWT)</td>
<td>11</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>Inversion/eversion ratio (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Ankle plantar flexion/ dorsiflexion</strong></td>
<td>30</td>
<td>60</td>
<td>90</td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plantar flexion (% BWT)</td>
<td>70–75</td>
<td>65</td>
<td>51</td>
</tr>
<tr>
<td>Dorsiflexion (% BWT)</td>
<td>16</td>
<td>12</td>
<td>9</td>
</tr>
<tr>
<td>PF/DF ratio (%)</td>
<td>20–25</td>
<td>25</td>
<td>33–40</td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plantar flexion (% BWT)</td>
<td>60–65</td>
<td>50</td>
<td>40</td>
</tr>
<tr>
<td>Dorsiflexion (% BWT)</td>
<td>14</td>
<td>12</td>
<td>8</td>
</tr>
<tr>
<td>PF/DF ratio (%)</td>
<td>20–25</td>
<td>25</td>
<td>33–40</td>
</tr>
</tbody>
</table>

ER, External rotation; IR, internal rotation; BWT, body weight; PF, plantar flexion; DF, dorsiflexion.
### Relationship Between Isokinetic Testing and Functional Performance

<table>
<thead>
<tr>
<th>REFERENCE</th>
<th>GROUPS COMPARED</th>
<th>ISOKINETIC TEST</th>
<th>FUNCTIONAL TEST(S)</th>
<th>SIGNIFICANCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barber et al.</td>
<td>Normals, ACL-deficient</td>
<td>60-deg/sec knee extension</td>
<td>Single-leg hop for distance</td>
<td>p ≤ 0.01</td>
</tr>
<tr>
<td>Noyes et al.</td>
<td>Normals, ACL-deficient</td>
<td>60-deg/sec knee extension, 300-deg/sec knee extension</td>
<td>Single-leg timed hop for distance</td>
<td>Statistical trend was found with 60-deg/sec quadriceps scores and hop tests; trends were not apparent at 300 deg/sec</td>
</tr>
<tr>
<td>Sachs et al.</td>
<td>Postoperative ACL</td>
<td>60-deg/sec knee extension and flexion</td>
<td>Single-leg hop for distance</td>
<td>Quadriceps and hamstring peak torque indices correlated with mean hop index</td>
</tr>
<tr>
<td>Wilk et al.</td>
<td>Postoperative ACL</td>
<td>180-, 300-, and 450-deg/sec knee extension and flexion</td>
<td>Single-leg hop for distance</td>
<td>Positive correlation was found between knee extension peak torque (180 and 300 deg/sec) and subjective knee scores of function and hop tests</td>
</tr>
</tbody>
</table>

### ACL, Anterior cruciate ligament.

10. **What is the correlation between isokinetic testing and functional performance?**
   The research is divided, although most studies indicate that a correlation exists. Only Anderson et al. and Greenberger and Paterno have reported that no correlation is evident. Several studies (Petsching et al., Baltaci et al., and Kong et al.) demonstrate there is a strong correlation between isokinetic testing and functional performance as measured with various performance tests.

11. **How can isokinetic testing be integrated in a rehabilitation functional testing algorithm?**
   Davies created the functional testing algorithm (FTA), which consists of a series of progressively challenging tests. The FTA can be used to assess patient progress and determine readiness to return to activity. With serial reassessments, the clinician can update and customize the clinical rehabilitation program and home exercise program and plan appropriately for discharge. Specific criteria have been established for testing progression within the FTA (see table).
DAVIES’ FUNCTIONAL TESTING ALGORITHM

- Basic measurements (e.g., visual analog pain scales, anthropometric measurements, goniometric measurements)
- KT 1000 testing for injuries of the anterior and posterior cruciate ligaments
- Kinesthetic, proprioceptive, and balance testing
- Closed kinetic chain (CKC) supine isokinetic testing
- OKC isokinetic testing
- CKC squat isokinetic testing
- Functional jump test
- Functional hop test
- Lower extremity functional test
- Specific testing for activities of daily living, vocation, and sports
- Discharge and return to activity

<table>
<thead>
<tr>
<th>TESTS</th>
<th>EMPIRIC GUIDELINES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sport-specific testing (SST)</td>
<td></td>
</tr>
<tr>
<td>Lower extremity function test (LEFT)</td>
<td>Female: 2:00 min</td>
</tr>
<tr>
<td></td>
<td>Male: 1:30 min</td>
</tr>
<tr>
<td>Functional hop test (FHT)</td>
<td>&lt;15% bilateral comparison and normals</td>
</tr>
<tr>
<td>Functional jump test (FJT)</td>
<td>&lt;20% compared with body height and normals</td>
</tr>
<tr>
<td>CKC isokinetic testing (standing)</td>
<td>&lt;20% bilateral comparison</td>
</tr>
<tr>
<td>OKC isokinetic testing</td>
<td>&lt;20% bilateral comparison</td>
</tr>
<tr>
<td>CKC isokinetic testing (supine)</td>
<td>&lt;30% bilateral comparison</td>
</tr>
<tr>
<td>Digital balance evaluation (DBE)</td>
<td>&lt;0.6</td>
</tr>
<tr>
<td>KT</td>
<td>&lt;3-mm bilateral comparison</td>
</tr>
<tr>
<td>Basic objective measurements</td>
<td>&lt;10% bilateral comparison</td>
</tr>
<tr>
<td>Subjective status</td>
<td>Pain &lt;3 (analog pain scale 0–10)</td>
</tr>
</tbody>
</table>

CKC, Closed kinetic chain; OKC, open kinetic chain; KT, KT-1000 knee ligament arthrometer.

12. Is isokinetic exercise beneficial?
Numerous published studies demonstrate the efficacy of isokinetic exercise in improving muscle performance. Timm conducted a comprehensive study of 5381 patients over a 5-year period to evaluate the effectiveness of rehabilitation programs after knee surgery. He found that people who performed isokinetic exercises were discharged to resume normal activity earlier than people who performed isometrics or isotonics. A classic study by Mikkelson et al. demonstrated no increased knee laxity, stronger quadriceps, and return to sports 2 months sooner using an integrated OKC and CKC rehabilitation program.

13. Discuss the use of a short-arc spectrum isokinetic rehabilitation program.
Short-arc exercise is used when full ROM is contraindicated. It is difficult to accelerate an isokinetic dynamometer to angular velocities in excess of 180 deg/sec in a short arc of motion; angular velocities <180 deg/sec avoid free-limb acceleration. Angular velocities slower than 60 deg/sec should be avoided because increased joint compressive forces, abnormally slow motor patterns, and pain inhibition may occur. Angular velocities in multiples of 30 deg/sec should be used because of the physiologic overflow to slower angular velocities.
14. How can the principle of physiologic overflow with isokinetic exercise be applied in rehabilitation?

Increases in strength are fairly velocity-specific, but with isokinetic exercise a 30-deg/sec physiologic overflow occurs at each angular velocity to slower velocities. Therefore it is not necessary to exercise at each angular velocity in a chosen velocity spectrum. Instead, incremental velocities of 30 deg/sec may be used. Isokinetic exercise also produces a physiologic ROM overflow of 30 degrees. For example, a patient with shoulder pain during 90 to 120 degrees of elevation can perform short-arc isokinetic exercises at 60 to 90 degrees and at 120 to 150 degrees and still experience strength gains within the painful arc as a result of the physiologic overflow.

15. Does isolated OKC isokinetic training improve functional performance?

Ellenbecker, Davies, and Rowinski found that performing isolated kinetic OKC rotator cuff muscle exercise increased tennis serve velocity. Mont et al. and Trieber reported similar results. These studies indicate that isolating individual components of the kinetic chain (particularly when they are critical functional components) can positively affect functional performance.

16. What does the evidence show regarding the use of open kinetic chain (isolated joint exercises) or isokinetics in regard to rehabilitation of patients with patellofemoral pain syndromes (PFPS)/anterior knee pain syndromes?

Witvrouw et al. have demonstrated that there is no statistically significant difference in most pain or functional measures between the OKC and CKC groups for rehabilitation of anterior knee pain.

17. What does the evidence demonstrate regarding the use of open kinetic chain (isolated joint exercises) or isokinetics and development of knee pain in patients during the rehabilitation program after ACL reconstructions?

Morrisey et al. divided patients who had ACL reconstructions into an OKC or a CKC rehabilitation program. Morrisey et al. concluded that “OKC and CKC leg extensor training in the early period after ACL reconstruction do not differ in their immediate effects on anterior knee pain.” Hooper et al. demonstrated no clinically significant differences in the functional improvement resulting from OKC or CKC exercises in the early period after ACL repair. Mikkelsen et al. demonstrated that there were no significant differences in hamstring torques between the OKC group versus the OKC combined with CKC group. The integrated rehabilitation group (OKC and CKC) did have statistically significant increases in the quadriceps torque. Furthermore, the OKC/CKC group had a significant number of patients returning to sports at the same level as before the surgery. The OKC/CKC group who returned to the same level of sports participation as before the surgery also returned 2 months earlier than those with CKC exercises only.

18. What does the recent evidence demonstrate in regard to the use of open kinetic chain (OKC) (isolated joint exercises) or isokinetics for rehabilitation of patients with ACL reconstructions? What are the effects of the exercises on the graft?

Bennyon et al. used a dynamic variable reluctance transducer (DVRT) that was actually sewn onto the ACL ligament. The subjects were then asked to perform a series of exercises, and the strain on the ACL was measured in vivo. OKC knee flexion (100 degrees) to extension (0 degrees) in an unloaded condition created a 3.5% strain on the ACL. CKC squats from extension (0 degrees) to flexion (100 degrees) produced a 3.6% strain on the ACL. CKC squats using a sport cord from extension (0 degrees) to flexion (100 degrees) produced a 4.0% strain on the ACL. In their conclusions, Beynnon et al. speculated that “it might not be valid to designate CKC or OKC activities as ‘safe’ or ‘unsafe’ with respect to rehabilitation of the injured ACL or healing graft.”

19. Do OKC exercises actually stress the graft where it could compromise long-term healing and maturation?

Additional research by Fleming et al. evaluated the strain on the ACL using a similar methodology as described in the previous study. The results demonstrated the following responses regarding the ACL strain:

- Isometric hamstrings at 30/60/90 degrees—0.0%
- Quadriceps and hamstrings (Q & H) co-contraction at 90 degrees—0.0%
- Q & H co-contraction at 60 degrees—0.0%
- Q isometrics at 90 degrees—0.0%
- Q isometrics at 60 degrees—0.0%
20. **Because of the regional interdependency concept, does isokinetic testing of the lower kinetic chain demonstrate particular deficits with specific pathologies?**

A popular topic today is the concept of regional interdependency. One of the first studies to demonstrate this concept with objective isokinetic testing was actually published 36 years ago by Nicholas et al. As an example, they demonstrated that patients who had ankle injuries also had statistically significant deficits in the ipsilateral hip abductors and hip adductors. Patients with patellofemoral pain syndrome demonstrated statistically significant deficits in the quadriceps, hamstrings, hip flexors, and trends toward weaknesses of the hip abductors and hip adductors. Again this demonstrates the importance of isolated testing in order to identify weak links in the kinematic chain. With CKC testing or functional testing, it does not demonstrate where the weaknesses are within the kinematic chain and therefore what needs to be addressed in rehabilitation.

**BIBLIOGRAPHY**


Morrissey, M. C., et al. (2002). Effects of distally fixed (CKC) vs nondistally fixed (OKC) leg extensor resistance training on knee pain in the early period after ACL reconstruction. *Physical Therapy*, 82, 35–43.


CHAPTER 34 QUESTIONS

1. Preoperative testing for patients with an ACL deficient knee demonstrate that a patient with a _____% deficit will continue to have a residual deficit 2 years later:
   a. 10%
   b. 20%
   c. 30%
   d. 40%

2. Mikkelson et al. demonstrated that patients with ACL reconstructions following OKC and CKC rehabilitation, versus CKC rehabilitation only, demonstrated which of the following?
   a. No increase in knee laxity, stronger quadriceps, and return to sports 2 months sooner
   b. Increase in knee laxity, stronger quadriceps, and return to sports 2 months sooner
   c. No increase in knee laxity, weaker quadriceps, and return to sports 2 months sooner
   d. No increase in knee laxity, stronger quadriceps, and delayed return to sports

3. Several studies (Wilk et al., Petsching et al., Baltaci et al., Artero et al., and Kong et al.) have demonstrated which of the following relationships?
   a. No correlation between isokinetic testing and functional performance testing
   b. No correlation between subjective knee scores and functional performance testing
   c. A correlation between isokinetic testing and functional performance testing
   d. None of the above

4. In a systematic review of ACL reconstruction rehabilitation programs by Kruse et al., the results indicated which of the following?
   a. CKC exercises only should be used.
   b. OKC exercises only should be used.
   c. Neuromuscular stability exercises only should be used in the rehabilitation program.
   d. Neuromuscular exercises should not be performed to the exclusion of strengthening and ROM exercises.

5. The concept of regional interdependency is a popular topic in the present literature. However, one of the first studies to demonstrate this concept used isokinetic testing by doing which of the following?
   a. Performing total leg isokinetic strength testing of lower extremity muscles and correlating to weaknesses throughout the lower extremity
   b. Performing total leg isokinetic strength testing of lower extremity muscles and correlating to weaknesses throughout the upper extremity
   c. Performing total leg isokinetic strength testing of lower extremity muscles and correlating to performance testing
   d. Correlating OKC with CKC testing
1. Summarize the critical demographics of aging in America and the effects on health care.

   Functional declines, physical disability, and greater use of health care resources are associated with aging. Health care costs are higher per capita among older Americans than any other age group. Older adults comprise the fastest growing segment of the US population. Estimates project that the population of persons aged 65 and older will double to 92 million by 2060 and the population of persons aged 85 and older will triple to 5.9 million by 2040. By 2056, the number of people aged 65 and older will be greater than those aged 18 and younger. By 2060, the older population will be more diverse. Projections indicate that non-Hispanic white adults will decline to 56% of the total population. The proportion of Asian Americans will double, and the proportion of Hispanics will nearly triple. The ratio of males per 100 females is projected to increase from 77.3 in 2012 to 84.4 in 2060. Males tend to have higher health care costs than females in the later decades of life.

2. Summarize the health status of older adults.

   At least 80% of older adults have at least one chronic illness and more than 50% have multiple chronic conditions. Hypertension, diabetes, cardiovascular disease, arthritis, chronic obstructive lung disease, and mental illness is increasing and contributes significantly to disability and reduced quality of life. Heart disease and cancer are the leading causes of death among all adults aged 65 and older, regardless of sex, race, and ethnicity. Other leading causes of death are chronic respiratory disease, stroke, Alzheimer’s disease, diabetes, influenza, and pneumonia.

3. What is the importance of fall risk assessment in older adults? What factors are associated with an increased incidence of falls?

   Falls are the leading cause of fatal and nonfatal injury among people over 65 years of age. One in every three older community-dwelling adults falls each year. A multifactorial fall risk assessment should be conducted on all persons who report falling in the previous year or on those who have gait and lower extremity muscle strength or balance abnormalities. Risk factors associated with falls include lower extremity muscle weakness, gait and balance impairments, impaired vision, variable blood pressure, poor vision, cognitive impairment, psychoactive medications or polypharmacy, footwear or foot problems, and environmental hazards.

4. Can exercise reduce the risk of falling?

   Yes, but it is difficult to determine the relative contribution or type of exercise for decreasing the risk of falls because many studies incorporate exercise into a multifaceted treatment approach. Individual and group exercise programs that include balance, coordination, and gait and strength training have been shown to reduce falls among community-dwelling older people. Training programs longer than 12 weeks are most effective. Caution should be used when initiating exercise among sedentary older persons with limited mobility, as exercise could increase fall rate.

5. What medications are associated with increased risk of falling?

   Antidepressants and sedatives are most strongly linked to increased risk of falls, but cardiovascular drugs to control hypertension and arrhythmias are also implicated. A significant number of falls are associated with postural hypotension, an adverse side effect of many cardiovascular medications.
6. What is orthostatic (postural) hypotension, and what are common signs and symptoms?
Orthostatic hypotension is defined as a drop in systolic blood pressure of > 20 mm Hg or a drop in diastolic blood pressure of 10 mm Hg with a concurrent rise in pulse rate within 3 minutes of moving from supine or sitting to a standing position. Associated signs and symptoms include dizziness, lightheadedness, blurred vision, and syncope or fainting. Orthostatic hypotension has been associated with increased falls among older adults.

7. Describe physical therapy interventions for orthostatic hypotension.
Treatment strategies include progressive elevation of the head of the bed, progressive sitting on the side of the bed while performing active leg exercises, and deep breathing. The use of lower extremity elastic stockings during physical activity and elevating the bed by 5 to 20 degrees during sleep is recommended.

8. Describe the musculoskeletal effects of aging.
Age-related decline in muscle mass (sarcopenia) begins in the third decade of life and accelerates after age 50. As slow-twitch type I fibers and fast-twitch type II fibers decrease in number, the cross-sectional area of muscle mass is reduced. Muscle strength decreases approximately 8% per decade, beginning in the third decade of life, with a total loss of 40% to 50% by age 80. Muscle weakness may be as a result of a reduction in the number and force-generating capacity of cross bridges between actin and myosin myofilaments. Power or speed of movement is also compromised because of a loss of fast-twitch fibers. The amount of collagen increases within soft tissues, but collagen becomes less extensible because of increased numbers of cross-links and loss of water content. Joint capsules and ligaments stiffen with age. By the seventh decade, joint motion may decrease 20% to 30% and can affect mobility. These musculoskeletal effects may lead to functional declines, frailty, and ultimately, loss of independent living. Bone mass declines with age regardless of sex, with the highest rates of loss occurring in postmenopausal women. Estrogen deficiency plays a role in reduced bone formation and increased bone loss in men and women.

9. What causes frailty in older adults?
Sarcopenia is a major contributor to frailty, a common syndrome particularly among persons older than 80 years of age. Frailty is associated with an increased risk of falling, disability, and death. Although there is disagreement regarding the definition of frailty, many consider a person frail when two or more of the following factors are present: unintended weight loss of 10 lb or more in a year, extreme exhaustion, muscle weakness, reduced gait speed, and low physical activity level. A vicious cycle of inactivity and functional decline ensues among persons who are frail, because a high percentage of energy reserves are used to perform simple activities. A multidisciplinary treatment approach that includes progressive resistance exercise and functional training has been shown to be of particular benefit.

10. What muscle groups are often weak in older adults?
In general, trunk and lower extremity muscles are affected to a greater extent than upper extremity muscles. With inactivity, the postural, antigravity muscles such as the quadriceps, gluteals, erector spinae, and gastrocnemius-soleus are affected the most. These muscle groups are important for upright posture, locomotion, and functional independence.

11. What musculoskeletal effects of aging can be reversed or attenuated with exercise?
Exercise positively affects flexibility, strength, power, and muscle mass in older adults. The age-related decline in bone mass may be offset by weight-bearing endurance and resistance exercise. Bone loading forces should be moderate to high to affect bone mineral density. In postmenopausal women, hormone therapy may be necessary to prevent osteoporosis, even among those who are physically active.

12. Summarize the recommended protocol for strength and power training in older adults.
For muscle adaptations to occur with strength training, an intensity of 60% to 80% of the muscle's maximum force-generating capacity (one repetition maximum [1-RM]) is recommended. The 1-RM threshold is considered the workload that can be lifted only once through the full range of motion while using excellent form. Percentages of the 1-RM can be determined using a rate of perceived exertion (RPE) scale or by determining the maximum number of repetitions achieved by an exercising muscle at near failure (demonstrated by a decline in form or lack of full motion for the last one to two repetitions). A maximum repetition capacity of 15 (RPE of fairly light to moderately hard) equates to a 60% overload.
stimulus, and an 80% workload equates to a maximum repetition capacity of 10 (RPE of hard to very hard). Emphasis should be placed on strengthening the major muscle groups, especially those of the hips, trunk, and lower extremities. Each major muscle group should be exercised two to three times a week with 1 to 2 days of rest before the next workout of the same muscle group. Strength gains can be achieved with a single set of repetitions per exercise maneuver, with greater strength gains realized with higher numbers of sets. Power training (generating muscle force quickly) should be incorporated into the resistance training routine once an individual can complete two sets of 10 to 15 maximum repetitions of an exercise maneuver. The concentric phase of an exercise should be performed quickly and the eccentric phase performed more slowly. The power training component should progress from 20% to 60% of the 1-RM.

13. When is exercise or exercise testing not recommended in older adults?
According to the American College of Sports Medicine (ACSM), exercise and/or exercise testing is not recommended if there are recent changes in an ECG suggesting myocardial ischemia, an acute cardiac event such as myocardial infarction, pulmonary embolism, severe aortic stenosis, symptomatic heart failure, or an aortic aneurysm. Other contraindications include severe shortness of breath, infection or inflammation of the heart, or any systemic infection.

14. When is heavy resistance training not recommended in older adults?
Porter and Vandervoort determined that participation in heavy resistance exercise should be avoided or limited in older adults with a history of hypertension, acute or “unstable” cardiovascular disease, unstable chronic conditions (e.g., uncontrolled diabetes mellitus), recent bone or joint injury, recent surgery, or any condition that prevents strong muscular contractions. Blood pressure and heart rate should be monitored before, during, and after exercise.

15. Summarize the recommendations for strength training in older adults with hypertension.
Aerobic exercise should be the first priority. Resting systolic and diastolic blood pressure ≥160 mm Hg and diastolic blood pressure ≥100 mm Hg are relative contraindications. Resistance should be of low to moderate weight load or 30% to 60% of one repetition maximum. The rate of perceived exertion should not be higher than 11 to 13 (fairly light to somewhat hard) on the 20-point Borg scale. Static hand-gripping and breath holding should be avoided. Increase resistance with each exercise only after 12 to 15 repetitions can be comfortably performed. Discontinue the exercise with the onset of abnormal signs or symptoms such as dizziness, unusual shortness of breath, angina-type discomfort, abnormal heart rhythm, cold sweat, confusion, excessive fatigue, or incoordination.

16. Summarize the recommendations for aerobic exercise in older adults.
The ACSM recommends physical activity at a moderate exertion level ≥5 days a week (for 30–60 minutes per day; total of 150–300 minutes per week) or vigorous exertion level ≥3 days a week (for 20–30 minutes per day; total of 75–100 minutes per week) or a combination of moderate to vigorous exertion levels 3 to 5 days a week. Older adults who are deconditioned should start at lower intensity levels and may need to exercise several times a day in 10-minute bouts to reach recommended exercise time frames. A progressive increase in time and/or intensity is necessary for improvement in aerobic fitness. Walking is one of the best types of aerobic activities for older adults because it is functional, provides weight-bearing stimulus to the lower extremities, and requires no special equipment. In persons with significant loss of muscle mass, a muscle strengthening program should be implemented before an aerobic training program.

17. Can older adults improve aerobic capacity with endurance training?
Yes; improvements of 10% to 30% can be seen, the same as in younger adults. The amount of improvement in aerobic capacity depends on baseline fitness level and training intensity.

18. Can exercise improve functional outcomes in older adults?
Exercise can improve lower extremity strength, power, and walking speed. Rising from a chair, the ability to climb steps, and cross the street are improved with high-velocity resistance training. An increase in walking speed has also been associated with a decreased risk of mortality in older adults.

19. Can exercise reduce mortality and increase life expectancy?
Moderate and vigorous physical activity has been associated with reduced all-cause mortality with greater relative risk reductions occurring with more vigorous exercise.
Compared with inactive individuals, those who exercise a minimum of 15 minutes per day or 90 minutes per week have a 3-year longer life expectancy.

20. What are the primary risk factors for cardiovascular disease? Why is this information important to orthopedic specialists?
Hypertension (blood pressure \( \geq 140/90 \text{ mm Hg} \)), cigarette smoking, hyperlipidemia (cholesterol \( >200 \text{ mg/dL} \)), diabetes mellitus, and positive family history of cardiovascular disease are primary risk factors. Other risk factors include age, male gender, obesity, a sedentary lifestyle, and stress. If the patient experiences pain above the waist that commences with activity and is relieved by rest, cardiac disease should be considered until ruled out. Patients with a primary diagnosis of orthopedic dysfunction should be screened for cardiovascular risk factors if exercise is a planned intervention.

21. What are appropriate cardiovascular responses to aerobic or dynamic exercise?
Heart rate and systolic and mean arterial blood pressure rise, and diastolic blood pressure remains the same, or falls slightly, with increasing workload. Failure of systolic blood pressure to rise with increasing workloads, or a drop \( >20 \text{ mm Hg} \), may indicate a decrease in cardiac output and correlate with myocardial ischemia or left ventricular dysfunction.

22. How should a person taking \( \beta \)-blocker medications be monitored during exercise?
Beta-blockers decrease the workload of the heart by decreasing heart rate and contractility and thus blood pressure at rest. During exercise, \( \beta \)-blockers blunt the heart rate and blood pressure responses; thus, heart rate and blood pressure measurements during exercise may not be a true measure of exercise effort. Patient symptoms and rating of perceived exertion are more helpful in evaluating tolerance to exercise. An exercise performance test is needed to prescribe exercise accurately. Calculating a target heart rate from the age-predicted maximal heart rate is inappropriate in patients taking \( \beta \)-blockers.

23. How should a person with a pacemaker or implantable cardioverter defibrillator (ICD) be monitored during exercise?
Warm-up and cool-down periods should be longer for people with fixed-rate pacemakers, and exercise intensity must be monitored by methods other than pulse counting (eg, patient symptoms, rating of perceived exertion, and blood pressure measurements). Rate-responsive pacemakers adapt the pacing rate to physical activity demands. Abnormal exercise response or unusual symptoms such as dyspnea, dizziness, or syncope should be reported immediately to a physician. During exercise, persons with an ICD should not exceed a heart rate that is within 10 beats per minute of the programmed antitachycardia pacing and defibrillation threshold.

24. How can general musculoskeletal chest pain be distinguished from cardiac ischemic pain?
The diffuse burning, squeezing, or crushing chest pain caused by cardiovascular disease should be differentiated from the more specific sharp, localized, stabbing, or burning chest pain of musculoskeletal origin. Musculoskeletal chest pain is generally unaffected by nitroglycerin, is accompanied by muscle and joint soreness, or tenderness to palpation and is not associated with electrocardiographic changes or constitutional symptoms. Cardiac chest pain (angina) is relieved by nitroglycerin and is associated with diaphoresis, shortness of breath, nausea, and/or ST-segment changes on the ECG. Anginal chest pain may occur anywhere above the waist, commence with exertion, and decrease with rest.

25. Can a patient experience a heart attack without the usual symptoms?
Yes. Sometimes people have no pain during an episode of critical loss of blood flow to the heart (ie, silent ischemia) but may complain of shortness of breath, weakness, fatigue, exhaustion, or flu-like symptoms. Persons with diabetes are at higher risk because of the autonomic neuropathy associated with the disease.

26. What are the exercise recommendations for patients with heart failure (HF)?
In the past, physical activity was restricted for patients with HF. However, recent studies have shown that exercise training can improve activity tolerance, symptoms, and quality of life without adversely affecting ventricular function. Exercise guidelines for persons with HF are difficult to implement because the patient’s condition often fluctuates, but exercise can be done safely in selected patients. Patients should be assessed thoroughly before exercise, and vital signs and symptoms should be closely monitored during exercise. A relative contraindication for exercise is uncompensated HF. Exercise can be initiated in patients with compensated HF, determined clinically by the ability to speak comfortably with a respiratory rate \(<30 \text{ breaths per minute} \), less than moderate fatigue, crackles in less than half of
the lungs, and a resting heart rate of less than 120 beats per minute. Exercise should be modified or terminated if the patient demonstrates extreme shortness of breath, marked fatigue, abnormal hemodynamic responses, development of abnormal heart sounds, arrhythmias, increase in pulmonary crackles, or evidence of myocardial ischemia. Because patients with HF are generally deconditioned, a low level of effort may be sufficient to induce positive physiological changes. Short walking sessions can be progressed to longer, less frequent bouts of activity. Dynamic light resistance training can improve muscle strength and endurance without adversely affecting left ventricular function.

27. What types of exercises are recommended for patients with chronic primary or secondary pulmonary disease?
Persons with pulmonary disease may benefit from an individually tailored program of breathing exercises, coughing techniques, cardiopulmonary endurance training, strength training, flexibility, respiratory muscle training, and relaxation exercises/techniques. Other components should include airway clearance techniques, ventilatory strategies, energy conservation, and patient education. Exercise training in persons with pulmonary disease may not directly improve lung function but, as demonstrated, may reduce hospital admissions and mortality and improve quality of life and function, particularly following acute exacerbations.

28. What types of exercises are recommended for people with osteoporosis?
Recommendations include moderate intensity weight-bearing aerobic exercise performed 3 to 5 days a week. Resistance training should be done 2 to 3 days a week and should include 8 to 12 repetitions of exercises of the major muscle groups. Exercise should emphasize hip and trunk stabilizing muscles, avoiding spinal flexion. High-impact loading and abrupt, ballistic movements should be avoided.

29. What are the most common causes of sport injuries in the older athlete?
Acute muscle injuries and overuse injuries in the lower extremities are the most common causes of sport injuries, particularly if the older adult suddenly increases the amount of physical activity involving repetitive movement. Older adult athletes performing a novel sport are at higher risk for injury than are younger persons.

30. Is exercise recommended for patients with cancer?
In general, yes. Cancer and the adverse effects of medical intervention can cause generalized weakness, fatigue, and impaired activity tolerance. The benefit of exercise varies with the type and stage of cancer and the effects of the treatment regime on immune system function. Because cancer treatment can affect the physiologic response to exercise, all patients should be medically screened before participation in an exercise program. Persons receiving chemotherapy or radiation may be at higher risk for peripheral neuropathy, cardiac dysfunction, and hematologic changes. Winningham’s contraindications for aerobic exercise in patients receiving chemotherapy are platelet counts < 50,000/mL, hemoglobin < 10 g/dL, white blood cell count < 3000/mL, and absolute granulocyte count < 500/mL. During exercise, persons with immunosuppression should be closely monitored for abnormal signs and symptoms of cardiopulmonary compromise. Patients who experience excessive fatigue with aerobic activity may better tolerate frequent short bouts of physical activity. Depending on the stage or severity of the disease, other therapeutic treatments or interventions such as functional training or energy conservation techniques may be warranted.

31. List the three most common fractures that older adults sustain:
- Vertebral (compression) fractures (≈ 750,000 annually)
- Hip fractures (≈ 300,000 annually)
- Distal radius fractures (≈ 250,000 annually)

BIBLIOGRAPHY
ACSM’s Guidelines for exercise testing and prescription. (2014) (9th ed.).
CHAPTER 35 QUESTIONS

1. It is estimated that by 2060, the population of adults aged 65 and older will:
   a. Remain the same
   b. Double
   c. Triple
   d. Quadruple

2. The leading causes of death among persons aged 65 and older are:
   a. **Heart disease and cancer**
   b. Cancer and chronic obstructive pulmonary disease
   c. Stroke and heart disease
   d. Alzheimer’s disease and pneumonia

3. The ACSM recommends that older adults should participate in aerobic exercise training at a moderate intensity of:
   a. 1 day a week
   b. 2 days a week
   c. 3 days a week
   d. **At least 5 days a week**
1. Is x-ray imaging dangerous?
In general, x-ray imaging is not dangerous. Radiation exposure from a single x-ray of an extremity is 0.01 millisievert (mSv), from a chest x-ray is 0.02 mSv, and from a lumbar spine/pelvic x-ray is 1.3 mSv. To put this in context, most people are exposed to a certain amount of radiation in the environment each day; an extremity x-ray is equivalent to one-half day of exposure, whereas chest and lumbar spine/pelvic x-rays are equivalent to 1 and 65 days of exposure, respectively.

2. How is an x-ray different from an arthrogram?
An arthrogram is an x-ray with a contrast material to examine soft tissue structure. The contrast material is commonly radiopaque iodine or gadolinium and is injected into the joint, typically to determine whether there is disruption of the joint capsule, thus evaluating the soft tissue structure of the joint.

3. What are the ABC’s of reading a radiograph?

A—Alignment: view the joint surfaces for congruency and alignment. For example, the shoulder, a ball-and-socket joint, should demonstrate the ball of the humeral head aligned within the cup of the glenoid fossa. Deviation from this normal anatomic alignment could indicate a minor subluxation or a major dislocation.

B—Bone density: observe the general bone density and look for distinct cortical edges. A loss of the distinct cortical edges may indicate loss of bone mass. Next, observe the local bone density. Look for areas of increased density that would indicate sclerosis. Also, observe texture abnormalities of the bone. When the mineralization of a bone is changed, the trabeculae can appear thin, delicate, coarsened, fluffy, or smudged.

C—Cartilage: although cartilage is not directly seen on an x-ray, inspecting the region in which cartilage lies can indicate possible problems with cartilage. Narrowed joint space (the area between articulating bones) can indicate arthritis, whereas widened joint space can be indicative of joint effusion or a genetic/metabolic condition such as acromegaly or chondrocalcinosis.

S—Soft tissue: typically swelling can be observed with an x-ray but is a nonspecific finding. Other soft tissue findings can be the presence of gas following surgery or trauma, calcification, and an abnormal soft tissue mass such as a hematoma, abscess, or tumor.

4. How many views are typically ordered to diagnose injuries?
The minimum number of films needed is usually two—the anteroposterior (AP) view and the lateral view. More often three views are obtained. An oblique view is often included because bones that overlap in either an AP or a lateral view can mask subtle fracture lines.

5. What is computed axial tomography (CAT/CT) scanning?
CT scans, also called CAT scans, were developed jointly in 1972 by Sir Godfrey Newbold Hounsfield in the United Kingdom and Dr. Allan Cormack in the United States and are based on mathematical reconstruction of multiple axial slices of x-rays surrounding the body part to be imaged. CT scans provide between 200 and 300 shades of gray compared with x-rays, which produce 20 to 30 shades of gray. All images are collected in the axial plane and then mathematically reconstructed to provide other views, such as coronal, sagittal, or 3D images.

6. What is diagnostic ultrasound?
Ultrasound images are generated from sound waves that bounce off of tissues back to the transducer, generating contrast between the different types of tissues. Originally developed in the 1950s to provide gray scale images of cardiac valve motion, ultrasound is now commonly used in obstetrics to image the developing fetus. Recent advances allow for greater resolution with reconstructed 3D and 4D images, creating videos of the moving fetus.
7. What is rehabilitation ultrasound imaging (RUI)?
RUI is a growing area in the rehabilitation field to visualize muscle structure and relate it to function. Most publications of RUI include measurement of lumbar or abdominal muscles statically or dynamically to provide feedback to patients. Ultrasound provides a method to quantify muscle function and is ideally suited to complement a rehabilitation program. Some clinicians have also reported using ultrasound imaging to visualize tissue prior to treatments. However, clinicians need to be cautioned regarding scope of practice before making statements regarding tissue integrity.

8. How is diagnostic ultrasound different from therapeutic ultrasound?
Therapeutic ultrasound is typically delivered at 1 to 2 W/cm², and diagnostic ultrasound is typically of much lower intensity, <0.1 W/cm². Therapeutic ultrasound is typically performed at 1 or 3 MHz, and diagnostic ultrasound is usually conducted at 5 to 10 MHz.

9. What are the advantages and disadvantages of ultrasound as an imaging modality?
Ultrasound is quick, readily available, and inexpensive and does not expose the patient to radiation. However ultrasound is highly operator-dependent, meaning that the skill of the technician administering the examination could enhance or degrade the accuracy of this type of imaging modality.

10. How does magnetic resonance imaging (MRI) work?
An MRI image is based on tissue response to multiple magnetic fields. The magnetic field knocks the tissue off its aligned position, which then responds based on its water content or, more specifically, hydrogen ion concentration. In other words, it is a matter of lining up the molecules (B0) and then knocking them down (B1).

The image is generated by how fast tissue responds to being knocked down. The time it takes to return to the upright position generates the T1 signal, also called the spin-lattice relaxation time, and the time it takes to return to moving at its natural frequency generates the T2 signal, or the spin-spin relaxation time.

11. What are the characteristics of the T1 image?
T1-weighted images show fat and blood as white, muscle tissue as gray, and edema, tumors, and cerebrospinal fluid (CSF) as black.

12. What are the characteristics of a T2 image?
T2-weighted images show CSF, edema, and tumors as white, muscle as gray, and fat, cartilage, and tendons as black.

13. Is exposure to the magnetic fields during MRI dangerous?
Magnetic fields of 1.5 and 3 T are generally not thought to be dangerous because of the relatively short exposure time experienced by patients. However, it is unknown at what exposure level (intensity or duration) magnetic fields become dangerous. Therefore in relatively healthy populations the only contraindication for MRI is pregnancy because of the unknown effects of magnetic fields on the developing fetus.

14. Should people with metal implants or electronic implants be excluded from MRI?
Metal implants can severely degrade an image, which renders MRI useless and warrants the use of other imaging studies for these types of patients. The farther away the metal is from the area being scanned, the less the effect. Therefore metal implants in the lower extremity should not affect MRI being conducted of the brain. Ferrous metal implants are considered a contraindication for MRI. Magnetic fields can also interfere with electronics and pose a risk to patients with electronic implants.

15. What is an MRI arthrogram?
It is similar to an x-ray and CT arthrogram, in that the contrast material (gadolinium) is injected to enhance the contrast between soft tissues.

16. What is the most valuable MRI sequence for assessing pathology?
A T2-weighted image is probably the most useful sequence to assess abnormalities on MRI. The reason for this is that fluid is bright and therefore stands out on T2-weighted images. Most pathologic processes (trauma, infection, tumors) lead to increased fluid content in tissues and therefore are bright on a T2-weighted image.

17. Is MRI best for evaluating soft tissue injuries?
Yes; however, there are circumstances where MRI might not be needed or warranted. In addition, multiple imaging planes increase scanning time and can cause patients to become claustrophobic. It is necessary for the radiologist to have an idea of what to look for so that the proper sequence can be conducted to image the correct anatomic region. For example, the ACL is best imaged in the sagittal plane where the full length of the ligament can be seen to assess its integrity.
18. What is the appearance of a normal ligament or tendon on MRI?
Both ligaments and tendons should be dark in all imaging sequences. Sometimes striations can be seen within these structures on MRI. The anterior cruciate ligament and the quadriceps and triceps tendons are examples where a striated appearance can be seen.

19. What is positron emission tomography (PET) scanning?
A PET scan is a diagnostic examination that is the acquisition of positrons from a radioactive substance administered to the patient. It is used most commonly to detect cancer and examine its effects by characterizing biochemical changes in the body. Patients are given a radioactive or tagged substance, usually glucose, before the examination. The tagged glucose will accumulate in areas representing biologic activity, differentiating it from areas that demonstrate greater uptake of glucose, such as a tumor. This is typically described as a hot spot. PET scans are also used to evaluate brain and heart function. Radiation doses are very small and short-lived and will not affect normal body processes. Pregnant women should not undergo this type of study.

20. What is a bone scan?
A bone scan or scintigraphy involves the injection of a slightly radioactive tracer (technetium-99m) into a vein to evaluate biophysiologic aspects of bone and disease. Bone scans are helpful in identifying stress fractures very early, before the onset of architectural changes in the bone. They are also valuable for detecting bone infections, arthritis, metabolic disorders such as Paget’s disease, and cancers that can spread to the bone. The amount of radioactive material is small and eliminated quickly. Complications are rare; however, pregnant women are not candidates for this procedure.

21. When will a stress fracture become visible on a plain film?
A stress fracture will not be visible on an x-ray film until approximately 7 to 14 days after the injury. An example of a second metatarsal stress fracture is provided with plain film radiographs.

AP view of the foot demonstrates callus formation along the medial aspect of the distal second metatarsal, compatible with a healing stress fracture.
22. What is the x-ray appearance of a stress fracture?
Radiographic abnormalities can include a subtle thin radiolucent line through the cortex, a focal band of sclerosis, or periosteal cortical thickening.

23. What is the appearance of a stress fracture on MRI?
On MRI, stress fractures appear as a linear zone of a dark signal (the fracture) with a broader and a poorly defined area of abnormal signal that represents edema in the bone.

24. What is patella alta and how is it diagnosed on radiographs?
Patella alta is a condition in which the patella is more superiorly displaced than normal. This can be seen with clinical observations or can also be examined radiographically with the Insall-Salvati ratio. The Insall-Salvati ratio is measured with the knee flexed 30 degrees on a lateral knee radiograph and is calculated as the length of the patella over the length of the patella tendon. Normally, this ratio is 1; a 20% deviation indicates patella alta.

25. What is a sulcus angle?
The sulcus angle is used to quantify the angle of the femoral sulcus, in which the patella sits. The sulcus angle is generated from a Merchant’s view radiograph, where the knee is flexed 30 degrees, and the image demonstrates the patella sitting in its femoral sulcus. The angle is defined by the highest and lowest point of the medial and lateral intercondylar sulci. A sulcus angle > 140 degrees indicates a shallower intercondylar sulcus and is suggestive of patellofemoral problems.

26. Is osteoporosis detectable by x-ray imaging, and if so, what is its appearance?
Yes; it is visible. However, it is visible only after a 30% to 50% loss of bone. Plain film radiographs typically show “picture framing,” where the cortex is sharp, but the trabeculae are decreased. The vertebral bodies appear as an “empty box,” because of increased density of the vertebral endplates. The vertebral bodies also show concavity and will demonstrate compression fractures with more severe cases of osteoporosis. Compression fractures are typically viewed as a wedge-shaped deformity of the vertebral body with loss of vertebral body height.

27. What are a delayed union and a nonunion?
Delayed fracture healing occurs when healing is slower than expected (16 to 18 weeks), and nonunion occurs when healing is delayed for longer than 6 months. Nonunion fractures may be broadly classified as atrophic or hypertrophic. Atrophic nonunions typically require stabilization and bone grafting, whereas hypertrophic nonunions may require stabilization only.

28. What is spondylolysis and how is it diagnosed radiographically?
Spondylolysis is a bony defect in the pars interarticularis caused by a chronic stress fracture; it is typically seen at the L5 vertebra in adolescent athletes. With oblique plain film radiographs, a collar...
or radiolucency is seen around the pars interarticularis, reminiscent of a dog collar. Spondylolisthesis occurs with a bilateral pars interarticularis defect, and there is slippage of one vertebral body on another because of the loss of stability provided by the bony architecture. See question 46 and figure associated with this.

29. **How is scoliosis measured radiographically?**
   It is measured on a posteroanterior (PA) film of the entire spine obtained with the patient in a standing position without shoes. The standard method of measuring scoliosis uses the Cobb method. Lines are drawn along the superior endplate and the inferior endplate of the highest and lowest vertebrae involved in the curvature. The angle subtended by lines drawn perpendicular to these two lines forms the Cobb angle.

![AP view of the thoracolumbar spine. The scoliosis in the lower spine is measured using the Cobb angle.](image)

30. **How is alignment of the cervical spine evaluated?**
   Three imaginary smooth curved lines can be drawn on a lateral view of the cervical spine to assess alignment. The lines are drawn along the anterior aspects of the vertebral bodies, the posterior aspects of the vertebral bodies, and the spinolaminar line. A fourth line can be drawn along the posterior aspects of the C2 to C7 spinous processes, though even in normal patients this line may not be smooth and contiguous. In the setting of trauma, any malalignment of the first three lines should be considered evidence of fracture or ligamentous injury.
31. When is the predental space considered abnormal?
The predental space (or atlantodental interval) is the space between the odontoid process and the anterior aspect of the ring of C1 (see figure). It is evaluated on the lateral radiograph of the cervical spine. The predental space is abnormal when it measures greater than 3 mm in adults and 5 mm in children. An increased atlantodental interval indicates atlantoaxial instability caused by rupture of the transverse ligament.

32. What is the normal thickness of the prevertebral soft tissues in the cervical spine?
Hematoma and edema of the soft tissues secondary to trauma can cause thickening of the soft tissues on the lateral radiograph of the cervical spine. Anterior to the C2 vertebral body, the soft tissues should not normally measure more than 7 mm. Anterior to C7, the prevertebral soft tissues can normally measure up to 22 mm.

33. What is ulnar variance?
Ulnar variance refers to the position of the distal articular surface of the ulna relative to the radius. Ulna neutral exists when the radius and ulna are of equal length.

In this situation, 80% of the axial load across the wrist is transmitted through the radius and 20% through the ulna. Ulnar variance is negative if the articular surface of the ulna is proximal to that of the radius.

Positive ulnar variance exists when the ulna is longer than the radius. With negative ulnar variance, less stress is borne by the ulna; conversely, with positive ulnar variance, the stress borne by the distal ulna increases. 80% of people are + or − 1 mm of ulnar neutral.

34. What are some of the common radiographic measurements made on wrist x-rays?
- Scapholunate angle—the angle formed by lines drawn through the long axis of the scaphoid and the axis of the lunate on a lateral view of the wrist. In normal individuals the scapholunate angle measures between 30 and 60 degrees. Ligament injuries and fractures can lead to carpal collapse patterns that result in an abnormally increased or decreased scapholunate angle.
Capitolunate angle—the intersection of lines drawn through the long axis of the capitate and the axis of the lunate on a lateral x-ray. Normally the capitolunate angles measures less than 20 degrees. An increase in this angle can be seen with carpal instability.

Radial inclination—drawn on a PA view of the wrist. This is the angle formed by a line drawn perpendicular to the long axis of the radius and a line drawn from medial to lateral along the distal edge of the radius. Normal radial inclination is approximately 23 degrees.

Palmar tilt—drawn similar to the angle of radial inclination except on a lateral view of the wrist. The first line is perpendicular to the long axis of the radius. The second line extends along the distal aspect of the radius, bridging the volar and dorsal edges. A measurement of 10 to 15 degrees is considered normal.
Lateral view of the wrist depicting the scapholunate angle. Line 1 is drawn along the long axis of the scaphoid while line 2 is drawn through the lunate. Normally, the angle measures between 30 and 60 degrees.

Lateral view of the wrist depicting the capitolunate angle. Line 1 is drawn along the long axis of the capitate while line 2 is drawn through the lunate. Normally, the angle measures less than 20 degrees.
PA view of the wrist demonstrating the normal angle of radial inclination.

Lateral view of the wrist demonstrating normal volar tilt of the distal radius.
35. What radiographic lines and angles can be used in the diagnosis of developmental dysplasia of the hip (DDH)?

- Hilgenreiner’s line—a horizontal line drawn through the triradiate cartilage
- Perkin’s line—drawn vertically along the lateral rim of the acetabulum
  
  The intersection of Hilgenreiner’s line and Perkin’s line divides the hip into four quadrants.

  The femoral head ossification center should normally be within the inner lower quadrant. With hip dislocation or DDH, the femoral ossific nucleus will be outside of this area.

- Shenton’s line—smooth curved line drawn between the medial femoral neck and the superior portion of the obturator foramen; may be broken or discontinuous in DDH or hip dislocation

- Acetabular index—a measure of the slope of the acetabular roof. The angle is formed by the intersection of Hilgenreiner’s line with a line drawn through the lateral margin of the acetabular roof. It varies with age; at birth, the angle normally measures between 18 and 36 degrees. Acetabular dysplasia is suggested when the acetabular index is increased.

- Wiberg’s center-edge angle (CE angle)—This angle is formed by a vertical line drawn superiorly from the center of the femoral head and a line drawn from the center of the femoral head to the lateral margin of the acetabular roof. It is an indication of acetabular depth. The CE angle is normal when it measures 20 to 40 degrees. The angle is decreased in dysplastic hips.

36. What is the femoral neck-shaft angle?

The femoral neck-shaft angle is the intersection of lines drawn through the axis of the femoral neck and femoral shaft. It measures approximately 150 degrees at birth and normally decreases with age. The neck-shaft angle measures 120 to 130 degrees in adults. A decrease in the neck-shaft angle is termed coxa vara, and an increase in this angle represents coxa valga.
37. How does an osteochondral lesion of the lateral femoral condyle appear on a radiograph?
38. How does an anterior dislocation of the shoulder appear on a radiograph?

AP view of the shoulder in a patient with an anterior dislocation. Although the anterior location of the humeral head (thin black arrow) relative to the glenoid (thick black arrow) cannot be detected on this view, the position of the humeral head below the coracoid process (thin white arrow) and medial and inferior to the glenoid is classic for this type of dislocation.

39. How does a normal ACL appear on MRI?

Sagittal proton density images demonstrating a normal ACL. A black signal is seen throughout the ACL as it extends from the femur to the proximal tibia (white arrows). The course of the ACL should parallel the posterior aspect of the intercondylar notch of the femur (black arrows). This is called Blumensaat’s line. Note the normal gray cartilage at the anterior aspect of the distal femur (open white arrow).
40. How does a ruptured ACL appear on MRI?

Sagittal proton density MRI of the knee. Normal tendons are black or low signal on MRI. Note the normal patella tendon anteriorly (thin black arrows). The posterior cruciate ligament (PCL) is also normal (thin white arrows). Only the inferior portion of the PCL is seen on this image. The anterior cruciate ligament (ACL) is abnormal. There is an amorphous intermediate or grayish signal where the normal ACL should be (open white arrows). Only the inferior aspect of the ACL contains the normal black signal.

41. How does a greater tuberosity fracture appear on MRI?

Coronal oblique T1-weighted MRI of the shoulder. The subcutaneous fat and the fat in the bone marrow are normally white or high signal on a T1-weighted image. In the region of the greater tuberosity, there is a gray signal in the bone marrow with black lines running through it (white arrows). This is compatible with a fracture and surrounding bone marrow edema, confirming the finding seen on the previous x-ray. The black line below the fracture is the normal physeal remnant (black arrows). Note the normal supraspinatus muscle and tendon just above the humeral head (open white arrows).
42. What are the MRI findings of a shoulder dislocation?
Hill-Sachs lesions of the humeral head are associated with as much as 75% of all shoulder dislocations and is an impaction of the humeral head on the glenoid as the humeral head tries to relocate into the glenoid fossa. Bankart tears occur when a piece of the labrum is torn off during an anterior shoulder dislocation. These lesions can also occur in up to 70% of anterior shoulder dislocations. Sometimes a bone chip from the glenoid rim is included, and this is called a bony Bankhart lesion.

![MRI Image of Shoulder Dislocation](image1)

43. What is the radiographic appearance of an acomioclavicular separation?
Acomioclavicular separations can appear as a minimal disruption that requires conservative or severe treatment that could necessitate surgical intervention to restore normal anatomy. Minor A-C separations, such as grade I and II, demonstrate widening of the AC joint but normal coracoclavicular distance and are treated conservatively. More severe AC separations will have marked widening of both the AC and coracoclavicular spaces. Right figure demonstrates grade III separation.

![Radiographic Image of Acomioclavicular Separation](image2)
44. What is the radiographic appearance of proper and improper total hip arthroplasty component positioning?

With aging populations, hip arthroplasty (“total hip” or “hip replacement”) is expected to rise for the foreseeable future. This procedure has been hailed as a major medical improvement over the past quarter century because of how it can restore pain-free lower extremity function. However, biomechanically correct placement of prostheses is necessary to restore function. Hip arthroplasty requires optimal placement of prosthetic components in order to maximize effectiveness. The acetabular cup should be anteverted 10 to 20 degrees and between 30 to 50 degrees of abduction in order to reduce the dislocation rate. Note the greater anteversion on the patient’s left side (right side of image) compared with the contralateral side.

45. Compare the appearance of a rotator cuff as viewed with ultrasound compared to MRI.

Musculoskeletal ultrasound is ideal for obtaining quality images of superficial (<4 cm) anatomy.

Full-thickness tear of the rotator cuff tendon.

(A) Transverse ultrasound image of the shoulder shows loss of the normal convex outer border of the rotator cuff tendon (arrows) at its insertion site onto the greater tuberosity (arrowhead). (B) Corresponding sagittal T2-weighted, spin-echo MRI of the shoulder shows a focal high signal intensity defect through the width of the rotator cuff tendon (arrow) at its insertion site onto the greater tuberosity.
46. What is the radiographic appearance of a pars interarticularis fracture?
Spondylolysis is a stress fracture of the pars interarticularis and appears radiographically as a “soft collar” or the broken neck of the “Scotty dog.” The Scotty dog is defined by the transverse process, being the nose of the dog, the super articular facet as the dog’s ear, the inferior articular facet as the foreleg of the dog, and the spinous process as the dog’s hind leg.

47. What is the radiographic appearance of a Jones fracture?
Jones fracture is typically at the base of the fifth metatarsal and needs to be distinguished from the avulsion fracture and from the peroneus brevis, sometimes called the pseudo-Jones fracture. This injury usually occurs with landing on an inverted foot. Note the oblique view gives better visualization of the fracture at the base of the fifth metatarsal.
48. What is the MRI appearance of a Triangle Fibrocartilage Complex (TFCC) tear?
Triangle fibrocartilage complex (TFCC) includes a cartilaginous disc and ligaments at the distal end of the ulna as it approximates the proximal carpals of the wrist. Tears can be classified as traumatic or degenerative and present with pain or discomfort ulnarily, near the styloid. Traumatic TFCC tears are associated with up to 50% of wrist dislocations and fractures. Note in the pictures a normal TFCC on the left and a TFCC tear indicated by fluid accumulation at the distal end of the ulna from a T2-weighted MRI.

![MRI images of TFCC tear](image)

49. What are the radiographic and MR appearances of avascular necrosis of the hip?

Between 5% to 40% of hip dislocations can lead to avascular necrosis (AVN) of the femoral head. However, it may take 3 to 4 months to show up radiographically. MRI can help identify bony changes earlier than conventional radiographs. Note that in part (A) the MRI can detect early changes. Radiographically a “crescent sign” can be seen at the tip of the femoral head.

![MRI images of AVN](image)
CHAPTER 36 QUESTIONS

1. Normal radiographic anatomy of the distal radius does not include:
   a. **Dorsal tilt of 10–15 degrees**
   b. Radial inclination of 23 degrees
   c. SL angle of 30–60 degrees
   d. + or − 1 mm of ulnar neutral variance

2. Normal femoral shaft angle in adult hips is approximately:
   a. 110 degrees
   b. **125 degrees**
   c. 145 degrees
   d. 160 degrees

3. Characteristics of T2 images include:
   a. fat as white
   b. CSF as black
   c. **Tendon as black**
   d. Edema as black

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BIBLIOGRAPHY


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Figure—cont’d  As destruction of the bone ensues, the femoral head will collapse as a result of weight bearing (B).
1. Name the origins, insertions, innervation, and actions of all muscles that attach to the scapula.

There are 17 muscles attached to the scapula, and the following table summarizes their origins, insertions, innervation, and action.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Origin</th>
<th>Insertion</th>
<th>Innervation</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subscapularis</td>
<td>Subscapularis fossa</td>
<td>Lesser tuberosity of humerus</td>
<td>Upper and lower subscapular nerve</td>
<td>Glenohumeral head depressor; extension, adduction, and medial rotation of humerus</td>
</tr>
<tr>
<td>Supraspinatus</td>
<td>Supraspinatus fossa</td>
<td>Superior facet on greater tuberosity</td>
<td>Suprascapular nerve</td>
<td>Abduction, stabilization of glenohumeral joint</td>
</tr>
<tr>
<td>Infraspinatus</td>
<td>Infraspinatus fossa</td>
<td>Middle facet on greater tuberosity</td>
<td>Suprascapular nerve</td>
<td>Extension and external rotation</td>
</tr>
<tr>
<td>Teres minor</td>
<td>Superior part of lateral border of scapula</td>
<td>Inferior facet on greater tuberosity</td>
<td>Axillary nerve</td>
<td>Extension and external rotation</td>
</tr>
<tr>
<td>Teres major</td>
<td>Dorsal surface of inferior angle of scapula</td>
<td>Medial lip of intertubercular groove of humerus</td>
<td>Lower subscapular nerve</td>
<td>Adducts and internally rotates arm</td>
</tr>
<tr>
<td>Serratus anterior</td>
<td>External surfaces of lateral parts of ribs 1–8</td>
<td>Anterior surface of medial border of scapula</td>
<td>Long thoracic nerve</td>
<td>Protracts and rotates scapula and holds it against thoracic wall</td>
</tr>
<tr>
<td>Deltoid</td>
<td>Lateral one third of clavicle, acromion, and spine of scapula</td>
<td>Deltoid tuberosity of humerus</td>
<td>Axillary nerve</td>
<td>Flexes, abducts, and extends arm</td>
</tr>
<tr>
<td>Trapezius</td>
<td>Spinous processes of cervical and thoracic vertebrae</td>
<td>Scapula and acromion</td>
<td>Spinal accessory nerve and branches of ansa cervicalis</td>
<td>Elevates, retracts, and rotates scapula</td>
</tr>
<tr>
<td>Levator scapula</td>
<td>Posterior tubercles of transverse processes of C1–C4 vertebrae</td>
<td>Superior part of medial border of scapula</td>
<td>Dorsal scapular nerve</td>
<td>Elevates scapula and tilts glenoid cavity inferiorly by rotating scapula</td>
</tr>
</tbody>
</table>
2. What is the normal scapulohumeral rhythm?
Normal scapulohumeral rhythm, as initially described by Codman in 1934, refers to the steady and continuous motion that occurs simultaneously at the scapulohumeral and scapulothoracic articulations during elevation of the arm. If the shoulder joint is abnormal, the scapula moves haltingly on the chest wall and not in concert with the glenohumeral joint. Although the relative motion of the glenohumeral joint to the scapulothoracic joint varies among individuals and at different ranges of the shoulder (1.25 to 1–4.3–1), the average is approximately 2 to 1.

3. Describe the gliding movements at the shoulder.
During rotational motion of the shoulder, obligate translation of the humeral head is a result of the asymmetric tightening and loosening of the capsuloligamentous structures. Anterior translation of the humeral head occurs with forward elevation beyond 55 degrees, and posterior translation occurs with extension >35 degrees. Surgical tightening of the posterior capsule or rotator interval tissue results in increased obligate anterior translation during forward elevation. Conversely, excessively tight anterior instability repairs shift the humeral head and joint contact point posteriorly. These findings illustrate that capsular restriction in one direction can lead to instability in the opposite direction. During elevation the humeral head moves superiorly 3 mm early in elevation then rotates in place with little translation.

4. How is glenohumeral joint stability maintained?
Stability of the glenohumeral joint depends on both static and dynamic stabilizers of the shoulder joint. The static or passive stabilizers of the shoulder joint include the glenohumeral joint capsule and ligaments. These structures are normally lax during the mid-range of motion but tighten at the extremes of motion, serving as passive checkreins to excessive glenohumeral translation. The dynamic stabilizers include primarily the rotator cuff and deltoid muscles, although all glenohumeral muscles contribute to stability to some degree. The dynamic stabilizers make the greatest contribution to stability within the functional mid-range of motion by actively contracting and keeping the humeral head centered in the
5. Which structure is the most important static restraint to anterior glenohumeral translation in the 90-degree abducted-externally rotated position?

Most traumatic shoulder dislocations are anterior and occur with the arm in the extreme abducted and externally rotated position. Cadaveric ligament-cutting studies have shown that different regions of the glenohumeral capsule and ligament complex are placed on stretch, depending on the position of the arm. The anterior band of the inferior glenohumeral ligament is the principal static restraint to the anterior translation of the humeral head with the arm in the 90-degree abducted-externally rotated position. The middle glenohumeral ligament is a significant restraint to anterior translation in the mid-range of shoulder elevation. The superior glenohumeral ligament appears to prevent excessive external rotation and inferior translation with the arm adducted at the side.

6. What are the normal strength ratios of the shoulder?

- Internal to external rotation: 3 to 2
- Adduction to abduction: 2 to 1
- Extension to flexion: 5 to 4

Women have approximately 45% to 65% of the shoulder strength of men.

7. What are the four parts of the proximal humerus?

The proximal humerus is composed of four distinct anatomic segments: 1) the shaft of the humerus, 2) the greater tuberosity, 3) the lesser tuberosity, and 4) the articular or head segment. These segments correspond to the four ossification centers of the proximal humerus. The shaft of the humerus connects with the proximal humerus at the surgical neck, just below the tuberosities. The anatomic neck is above the tuberosities, between the articular margin and the attachment of the articular capsule. The greater tuberosity has three facets for the attachment of the supraspinatus, infraspinatus, and teres minor muscles. The lesser tuberosity is the site of insertion of the subscapularis muscle. The four parts of the proximal humerus are common sites of fractures, especially in older patients with osteopenic bone, and form the basis for the Neer classification of proximal humerus fractures.

8. What is the normal shape of the human glenoid?

The normal anatomy of the bony glenoid is highly variable. The glenoid articular surface is often described as pear-shaped, with a larger diameter in the lower portion than in the upper portion. Although this is the most common shape seen, a cadaver study showed 29% of normal glenoids were ovoid in shape, with similar diameter in the upper and lower portions. Mean glenoid width is approximately 26.8 mm, with a normal range between 20 and 35 mm. Average glenoid height is 38 mm, with normal values ranging from 29.4 to 50.1 mm. Men typically have slightly larger glenoids than women in both of these dimensions. Inclination (or tilt) is the slope of the glenoid face in the superior-inferior direction. Average inclination is reported from −2.2 degrees (inferior tilt) to +4.2 degrees (superior tilt) but with normal ranges anywhere from −12 degrees to +15.8 degrees. Glenoid version is the direction of the glenoid surface in the anterior-posterior plane. Average glenoid version is about 2 degrees of retroversion, but normal version ranges between 12 degrees of anteroverision and 14 degrees of retroversion.

9. What is the rotator interval?

The rotator interval, as originally described by Neer, is the capsular tissue in the interval between the subscapularis and supraspinatus tendons. The rotator interval is composed of parts of the supraspinatus and subscapularis tendons, the coracohumeral ligament, and the superior glenohumeral ligament. These structures contribute to stability of the shoulder by limiting inferior translation and external rotation with the arm adducted, as well as posterior translation when the arm is forward flexed, adducted, and internally rotated. The more medial part of the interval primarily limits inferior translation and to a lesser extent external rotation, although the lateral part of the interval primarily limits external rotation in the adducted arm. Pathologic rotator interval tissue can play a significant role in limiting motion, particularly external rotation, in the setting of adhesive capsulitis. At the opposite end of the spectrum, deficient or attenuated rotator interval tissue may be associated with recurrent anteroinferior or multidirectional instability of the shoulder.

10. What are the basic biomechanical functions of the rotator cuff?

The rotator cuff acts to provide stability through force couples and aid in motion about the glenohumeral joint. The subscapularis, the strongest of the cuff muscles, makes up the anterior portion of this transverse force couple. This balances the posterior portion, made up of the supraspinatus,
The combined action of the anterior and posterior force couple creates compression of the humeral head into the glenoid and a humeral head–depressing effect that counteracts the superior pull of the deltoid muscle. Concavity compression refers to the stability obtained by compression of the humeral head into the concave glenoid fossa. This compressive load is primarily provided by dynamic muscle contraction of the rotator cuff muscles, enhancing glenohumeral stability during motion and maintaining proper position of the humeral head within the glenoid.

11. Describe the anatomy of the supraspinatus tendon and its clinical significance.

The supraspinatus muscle functions to initiate abduction and depress the humeral head against the upward pull of the deltoid. The muscle and tendon travel slightly obliquely from posterior to anterior, allowing it to contribute to external rotation as well. It has a broad attachment at the greater tuberosity, which is why supraspinatus strength testing is best performed in internal rotation, as this brings the tendon insertion into the plane of the scapula. There are two muscle bellies, anterior and posterior. The anterior muscle belly is larger and pulls through a smaller tendon area. Thus the anterior tendon stress is significantly greater than the posterior tendon stress, and rotator cuff tendon repairs should incorporate the anterior tendon whenever possible, as it acts as the primary contractile unit.

12. Describe the role of the long head of the biceps.

Opinions vary considerably. Some investigators suggest that it is a vestigial structure, whereas others believe that it plays a crucial role in shoulder stability. Dynamic cadaveric and in vivo electromyographic studies have shown that the long head of the biceps may contribute to anterior stability of the shoulder by decreasing translation of the humeral head and may have a humeral head–depressing effect in the presence of a large rotator cuff tear by restraining superior migration of the humeral head. Elbow flexion strength can decrease by as much as 30% after a tear of the long head of the biceps. Supination decreases by an average of 10% to 20%. Abduction strength may decrease 20% after a tear of the long head of the biceps secondary to the loss of its stabilizing function.

13. What is the role of the bicipital groove in anterosuperior shoulder pain?

The differential diagnosis of anterosuperior shoulder pain can include impingement syndrome, rotator cuff pathology, acromioclavicular joint pain, instability, and biceps tendon disease. Furthermore, there is a positive association between radiographic degenerative changes of the bicipital groove and anterosuperior shoulder pain. Studies have shown there is an increased incidence of bicipital tendon disease in patients with degenerative changes in the bicipital groove. These degenerative changes include stenosis and osteophyte formation, which has been correlated to biceps tendon disease via ultrasonography.

14. Describe the most common variations of the labral origin of the biceps anchor.

Forty to sixty percent of the biceps tendon origin is from the supraglenoid tubercle, although the remaining fibers originate from the superior glenoid labrum. There is considerable variability in the attachment to the superior labrum. The most common variation is an equal contribution of anterior and posterior labral attachment. The next most common is attachment mostly posterior but with a small contribution to the anterior labrum. The third most common variation consists of an entirely posterior attachment. Finally, the least common labral attachment is mostly anterior but with a small contribution to the posterior labrum.

15. Define the borders of the quadrangular space, triangular space, and triangular interval. Which structures pass through them respectively?

The quadrangular space (also known as the quadrilateral space) is an anatomic interval formed by the shaft of the humerus laterally, the long head of the triceps medially, the teres minor muscle superiorly, and the teres major muscle inferiorly. The axillary nerve and the posterior humeral circumflex artery pass through this space from anterior to posterior.

The triangular space is an anatomic interval medial to the quadrangular space. Its borders are formed by the long head of the triceps laterally, the teres minor superiorly, and the teres major inferiorly. The circumflex scapular artery, a branch of the scapular artery, passes through the triangular space.

The triangular interval is inferior to the quadrangular space, bordered by the teres major superiorly, the long head of the triceps medially, and the lateral head of the triceps laterally. The radial nerve and profunda brachii artery pass through the triangular interval.
16. Describe the three most common normal variations in anterior labral anatomy.

The three most common variations are the following: 1) the presence of a sublabral foramen, defined as the sulcus between a well-developed anterosuperior portion of the labrum and glenoid articular cartilage; 2) the presence of a sublabral foramen and a cordlike middle glenohumeral ligament; 3) the complete absence of labral tissue at the anterosuperior aspect of the labrum in association with a cordlike middle glenohumeral ligament attached to the superior part of the labrum at the base of the biceps (Buford complex).

17. What is a Bankart lesion?

A Bankart lesion represents a lesion of the glenoid labrum corresponding to the detachment of the anchoring point of the anterior band of the inferior glenohumeral ligament and middle glenohumeral ligament from the glenoid rim. It is the result of a traumatic anterior dislocation of the glenohumeral joint. The injury can be of soft tissue, diagnosed as a labral tear at the anterior glenoid. There can also be fractures of the anterior lip of the glenoid, termed “bony Bankart” lesions.

18. What is an HAGL lesion?

An HAGL lesion represents an avulsion of the humeral attachment of the inferior glenohumeral ligament as a result of glenohumeral dislocation. This lesion can predispose patients to recurrence of anterior glenohumeral instability. The HAGL lesion is analogous to an avulsion of the glenoid attachment of the inferior glenohumeral ligament that often accompanies a labral tear or Bankart lesion.

19. What is a Hill-Sachs lesion and how does it relate to recurrent anterior shoulder instability?

A Hill-Sachs lesion represents an impression fracture of the posterolateral margin of the humeral head caused by impaction on the anteroinferior rim of the glenoid during an anterior shoulder dislocation. Hill-Sachs lesions are felt to become clinically important if they engage around the anterior rim of the glenoid at a position of function. Large Hill-Sachs lesions involving more than 30% of the humeral articular surface often contribute to recurrent shoulder instability. Smaller Hill-Sachs lesions can also contribute to recurrence when combined with anterior glenoid bone loss or Bankart lesions.

20. What is the biomechanical function of the clavicle?

The clavicle attaches medially to the manubrium through the sternoclavicular articulation and laterally to the scapula through the acromioclavicular articulation and the coracoclavicular ligaments. The clavicle functions as a strut for the shoulder girdle, providing the only bony connection between the upper extremity and the axial skeleton. By maintaining the upper extremity away from the midline, the clavicle improves the biomechanical efficiency of the axiohumeral muscles. As a result, the muscles do not expend their energy pulling the shoulder medially but rather create motion at the glenohumeral joint.

21. What are the normal motions of the clavicle?

During full abduction of the arm, the clavicle rotates 50 degrees axially. This clavicular rotation permits the glenoid fossa to continue to elevate with increasing arm elevation. If the clavicle is prevented from rotating, arm abduction is limited to 120 degrees. With arm motion, the clavicle has also been noted to retract and elevate at the acromioclavicular joint with arm abduction in the scapular or coronal plane. Compared with the clavicle position at rest, its lateral elevation is estimated between 15 degrees and 20 degrees and posterior retraction approximately 30 degrees with arm motion. Clavicle depression and protraction are seen to lesser degrees with shoulder extension and adduction.

22. Describe the origin, insertion, innervation, and function of the subclavius muscle.

The subclavius muscle has a tendinous origin from the first rib and inserts on the inferior surface of the middle third of the clavicle. It receives innervation from the nerve to the subclavius, a branch of the superior trunk of the brachial plexus with contributions from C5 and C6. The function of the subclavius muscle is to stabilize the sternoclavicular joint during strenuous activity.

23. Name the primary arterial supply to the humeral head.

The arcuate artery, the terminal branch of the ascending branch of the anterior humeral circumflex artery, supplies most of the blood to the humeral head. This branch ascends the bicipital groove with the long head of the biceps tendon, entering the bone near the articular margin. The remainder of the blood supply to the head comes from branches of the posterior humeral circumflex artery and from branches within the rotator cuff tendon insertions. The primary blood supply to the head has been debated after one study showed the posterior circumflex humeral artery to provide 64% of humeral
head blood supply. These results have not been reproduced, and consensus remains that the anterior humeral circumflex artery is the dominant blood supply to the humeral head.

24. What is the average proximal humerus articular version relative to the transepicondylar axis of the distal humerus?
The proximal humeral articular surface is retroverted toward the face of the glenoid. The average proximal humerus retroversion is 30 degrees relative to the transepicondylar axis of the elbow.

25. Describe the course of the suprascapular nerve.
The suprascapular nerve arises from the upper trunk of the brachial plexus. It courses posteriorly to the suprascapular notch of the scapula, accompanied by the suprascapular artery. The nerve passes through the notch deep to the transverse scapular ligament, whereas the artery passes over the ligament. The suprascapular nerve then travels deep to the supraspinatus, which it innervates. Next, it passes through the spinoglenoid notch at the base of the spine of the scapula before it continues deep to the infraspinatus, which it also innervates. Articular sensory branches are given off to the acromioclavicular and glenohumeral joints along the course of the nerve. Compression of the suprascapular nerve can occur at the suprascapular or spinoglenoid notches, producing posterior shoulder pain and weakness.

26. Which neurovascular structure is at greatest risk during anterior shoulder surgery? Describe the course and branches of this structure.
The structure at greatest risk during this surgery is the axillary nerve, which traverses posteriorly from the posterior cord of the brachial plexus to innervate the deltoid and teres minor muscles, as well as the skin over the lateral aspect of the upper arm. With the posterior humeral circumflex artery, it passes below the inferior border of the subscapularis and travels along the inferior glenohumeral joint capsule, with which it is intimately associated. Although passing through the quadrangular space, the axillary nerve will divide into four branches—motor branches to the anterior and posterior portions of the deltoid muscle, a sensory branch (superior lateral brachial cutaneous nerve), and a motor branch to the teres minor muscle. Careless surgical dissection of the subscapularis or anterior/inferior capsule, as well as aggressive retraction, can result in injury to the axillary nerve or one of its branches.

27. Which nerve lies superficial in the posterior cervical triangle and is susceptible to injury?
Cranial nerve XI (spinal accessory nerve) travels through the posterior cervical triangle just below the cervical fascia. The posterior cervical triangle is bordered by the sternocleidomastoid anteriorly, the trapezius posteriorly, and the clavicle inferiorly. The spinal accessory nerve may be injured iatrogenically, most commonly during cervical lymph node biopsy, or by direct trauma. Injury to the spinal accessory nerve, which innervates the trapezius, leads to drooping of the shoulder, an asymmetric neckline, pain, and weakness in elevation of the arm.

28. Which nerve injury leads to primary medial scapular winging?
The direction of scapular winging is defined by the direction of the inferior angle of the scapula. Injury to the long thoracic nerve leads to paralysis of the serratus anterior muscle, which normally stabilizes the scapula laterally. Medial winging of the scapula results because the medial scapular stabilizers remain unopposed and the lateral border of the scapula is no longer closely held against the thoracic cage.

29. Which nerve injuries lead to lateral scapular winging?
Palsy of the spinal accessory nerve (cranial nerve XI) leads to weakness of the trapezius muscle, a medial scapular stabilizer. Injury to the dorsal scapular nerve leads to weakness of the rhomboid major and minor muscles, which also attach to the medial scapula. Weakness of medial muscles from injury to either of these nerves leads to lateral scapular winging. Weakness of the trapezius leads to more pronounced lateral winging than that seen with loss of the rhomboids.

30. Describe the course of the musculocutaneous nerve.
The musculocutaneous nerve is a terminal branch of the lateral cord of the brachial plexus with contributions from C5, C6, and C7. It penetrates the muscle belly of the coracobrachialis, providing innervation through small motor branches. It then travels into the brachium between the brachialis and biceps brachii muscles, innervating both. Its terminal sensory branch emerges between the brachialis and brachioradialis muscles and travels into the forearm as the lateral antebrachial cutaneous nerve. This nerve provides sensation to the lateral aspect of the forearm.
31. Describe the basic structure of the brachial plexus.
The brachial plexus, which provides sensory and motor innervation to the upper extremity and shoulder girdle, receives contributions from spinal nerves C5–C8 and T1. Inconstant innervation is received from C3 and C4. The five roots from the ventral rami of C5–T1 coalesce to form three trunks (superior, middle, and inferior). The three trunks divide to produce three anterior and three posterior divisions. The divisions combine into the three cords of the brachial plexus (lateral, posterior, and medial). Finally, the cords end in the terminal branches, which are the musculocutaneous, axillary, radial, median, and ulnar nerves. A helpful mnemonic to remember the order of the components of the brachial plexus (roots, trunks, divisions, cords, branches) is Rod Taylor Drinks Cold Beer.

32. Is there a relationship between glenoid inclination and rotator cuff tears/instability?
Yes. Preliminary studies have demonstrated that increasing superior inclination of the glenoid significantly reduces the amount of force required for superior humeral head migration. This suggests that more upward-facing glenoids may increase the risk for superior humeral translation, which has been shown to contribute to the development of rotator cuff disease. Increased glenoid retroversion has been shown to increase the risk for posterior instability.

33. Where is the center of rotation of the normal glenohumeral joint? Where is the center of rotation in the severely cuff-deficient glenohumeral joint?
The center of rotation in the normal glenohumeral joint is at the center of the humeral head at the mid-glenoid level. In a severely rotator cuff-deficient glenohumeral joint, the head of the humerus migrates superiorly and medially secondary to the unopposed pull of the deltoid and the loss of the humeral head-depressing function of the rotator cuff. As a result, the center of rotation migrates superiorly. When the humeral head is no longer centered in the glenoid cavity because of abnormal force couples and loss of the glenohumeral fulcrum, the deltoid is at a mechanical disadvantage and limited abduction results.

34. What are the glenoid erosion patterns typically seen in osteoarthritis and rotator cuff arthropathy?
The most common erosion pattern seen with primary osteoarthritis is posterior glenoid wear with a variable degree of posterior humeral head subluxation. Posterior glenoid wear in osteoarthritis is often accentuated by internal rotation contractures that develop with worsening disease. Posterior wear increases glenoid retroversion and, if severe, can lead to formation of a biconcave glenoid. Severe retroversion is usually secondary to glenoid dysplasia, which is often a deficient formation of the posterior and inferior glenoid. Central glenoid erosion can also occur with osteoarthritis but is more often seen with inflammatory arthritis.

Rotator cuff arthropathy is defined by worsening proximal humeral migration, which leads to superior glenoid erosion. The humeral head is no longer depressed by a functional rotator cuff, and the

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The brachial plexus. a, Dorsal scapular; b, long thoracic; c, suprascapular; d, nerve to subclavius; e, lateral pectoral; f, upper subscapular; g, thoracodorsal; h, lower subscapular; i, axillary; j, medial pectoral; k, medial brachial cutaneous; l, median antebrachial cutaneous; m, musculocutaneous; n, median; o, radial; and p, ulnar.
head eventually articulates superiorly with the acromion. Superior wear of the glenoid leads to increasing
glenoid inclination/tilt.

35. What are the most frequently occurring anatomic variations of the coracoacromial (CA) ligament?
Variations include quadrangular (48%), Y shape (42%), broader lateral and thinner medial band,
broad-banded (8%), and multiple bands (2%).

36. Are the acromial attachments of the coracoacromial ligament and anterior deltoid preserved during arthroscopic acromioplasty?
In most cases, the acromial attachment of the CA ligament is released during an arthroscopic
acromioplasty, as it can be an impinging structure and often has calcification or enthesopathy within it,
contributing to anterior acromial spur formation. Some surgeons attempt to preserve the anterior
attachment of the ligament, as it has a role in static restraint of the glenohumeral joint. The overlying
deltoid originating at the anterior part of the acromion is always left attached during acromioplasty
by a bridge of tissue composed of periosteum and deltoid tendon.

37. Describe the anatomy of the pectoralis major tendon including the insertion and
anatomy of the medial and lateral pectoral nerves as they relate to the insertion
of the tendon.
The width of the insertion of the pectoralis major is approximately 6 cm. The insertion is broad on the
undersurface of the tendon and small on the anterior surface. The sternal head spirals into its insertion to
form the posterior lamina, and the clavicular head remains anterior as it inserts into the humerus to form
the anterior lamina.

The medial pectoral nerve enters the pectoralis major approximately 12 cm from its lateral
humeral insertion and 2 cm from its inferior edge. The lateral pectoral nerve inserts approximately
12.5 cm from its humeral insertion. The medial pectoral nerve’s insertion into the pectoralis major
is inferior to the lateral pectoral nerve’s insertion. The lateral pectoral nerve passes medial to the
pectoralis minor before entering the pectoralis major, whereas the medial pectoral nerve passes through
or lateral to the pectoralis minor before entering the pectoralis major.

38. Describe the anatomy of the deltoid insertion.
The anterior, middle, and posterior deltoid muscle fibers enter into the deltoid insertion in a V-shaped
tendinous confluence. This consists of a broad posterior band and a narrow separate anterior band.
The anterior band accounts for approximately one fifth of the insertion. The insertion can be extremely
close to the pectoralis major insertion and in some cases nearly opposed to each other. The average
distance from the axillary nerve is approximately 5.6 cm anteriorly and 4.5 cm posteriorly.

39. What are the main stabilizers of the AC joint, and in which direction do they resist
displacement?
The acromioclavicular ligament and joint capsule acts as a primary constraint for posterior displacement
of the clavicle and posterior axial rotation. The conoid ligament plays a primary role in constraining
anterior and superior rotation, as well as anterior and superior displacement of the clavicle. The trapezoid
ligament contributes to constraint for both horizontal and vertical displacement primarily when the
clavicle moves in axial compression toward the acromion.

40. What direction is the most common with traumatic sternoclavicular dislocation?
What direction is the most dangerous?
Traumatic sternoclavicular joint dislocations are usually the result of high energy and can be either
anterior or posterior. Anterior dislocations are far more common and much less dangerous. Posterior
dislocations can potentially injure the mediastinal vessels and trachea, which can be life-threatening.
These injuries require more urgent reduction and may require assistance from a cardiothoracic surgeon. It
is also important to determine whether a medial clavicle physeal separation has occurred, rather
than a joint dislocation. The medial physis (growth plate) of the clavicle is the last to fuse in the body,
usually around 25 years of age.

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1. **What are the prevalence and natural history of rotator cuff disease?**

   Prevalence data deemed from cadaver investigations range from 7% to 40%. Recent MRI and ultrasound investigations of asymptomatic subjects demonstrated the prevalence of rotator cuff tears to range from 13% to 34%. The prevalence ranges from 51% to 54% in 60- to 80-year-old subjects. Patients with a rotator cuff tear who are asymptomatic have a 51% chance of becoming symptomatic in the future.

2. **Define os acromiale.**

   Os acromiale, or unfused acromial epiphysis, is the failure of the distal end of the acromion to ossify. Ossification usually occurs between 18 and 25 years of age. Os acromiale is often bilateral and may be seen in up to 8% of the normal population. The four presentations of os acromiale (pre, meso, meta, and basi) involve the acromion to greater or lesser degrees. An os acromiale may project into the rotator cuff outlet, decreasing its total area, and is thought to be associated with rotator cuff pathology.

3. **What are the three morphologic types of the acromion?**

   Bigliani classified the acromion according to its shape. Type I acromion is flat (17% incidence); type II (43% incidence) curves downward into the rotator cuff outlet; and type III (40% incidence) is hooked downward into the rotator cuff outlet. Of patients with rotator cuff tears, 70% have type III acromion, 27% have type II, and 3% have type I. Types II and III decrease the area of the rotator cuff outlet and can traumatize the superior surface of the rotator cuff tendons.

4. **Are types II and III acromia acquired or developmental?**

   It has been proposed that acromial hooks lie within the coracoacromial ligament and are actually traction spurs. Whenever the humeral head is pressed upward against the coracoacromial arch, it places a traction load on the distal lateral acromion, and a traction spur forms in response to the loading. It is similar to traction spurs that form on the calcaneus at the attachment of the plantar fascia.

5. **Describe Neer’s classification of rotator cuff pathology.**

   - **Stage I**—edema and hemorrhage. Patients usually are less than 25 years old and have pain with activity that often resolves with rest. The condition is reversible, and treatment is conservative (relative rest and medication).
   - **Stage II**—fibrosis and tendinitis. Patients typically are between 25 and 40 years old and experience recurrent pain with activity that does not always abate with rest. According to Neer, subacromial decompression should be considered if conservative treatment fails.
   - **Stage III**—bone spur and tendon rupture. Patients typically are older than 40 years and have a history of progressive disability that has led to a tear of the rotator cuff. Rotator cuff repair is advised.
   - **Stage IV**—cuff tear arthropathy. Patients typically are older than 60 years and have a history of progressive disability with a torn rotator cuff. Clinical management consists of rotator cuff repair, hemi-arthroplasty, or total shoulder replacement.

6. **Describe the coracoacromial arch and its clinical importance.**

   The coracoacromial arch consists of the coracoacromial ligament, which spans the distance between the coracoid and acromion of the scapula. The ligament provides a protective covering over the subacromial bursa and rotator cuff tendons and restricts excessive superior humeral head migration. Clinically, the coracoacromial ligament has been associated with rotator cuff pathology (especially in overhead athletes). During humeral elevation and internal rotation, the greater tuberosity and the attached rotator cuff tendons can be compressed against the arch. Repetitive compression may traumatize the rotator cuff tendons and lead to pathology.
7. What is a partial-thickness rotator cuff tear (tensile failure of the rotator cuff)?

The rotator cuff degenerates naturally with increasing age, especially after the third decade of life. Degeneration or tensile failure of the rotator cuff begins deep within the tissue near the undersurface attachment of the tuberosity. With time it may extend outward until it becomes a full-thickness tear. Partial-thickness tears of the rotator cuff also may occur on the bursal side of the cuff, most commonly near the insertion.

8. Do partial-thickness tears heal or progress to full-thickness tears?

Partial-thickness tears attempt to heal, but in most instances they progress to full-thickness tears. Matsen describes why partial-thickness rotator cuff tears eventually progress to full-thickness tears:

- Ruptured fibers can no longer sustain a load; thus increased loads are placed on neighboring fibers, making them more susceptible to rupture.
- Disruption of the tendon fibers also disrupts local blood supply within the tendon, thus inducing ischemia.
- Disrupted tendon fibers are exposed to joint fluid, which has a lytic effect on tendons that impairs the healing process.
- When a tendon heals, the scar tissue that replaces the ruptured tendon fibers does not have the same tensile strength as the original tissue; thus it is at increased risk of failure.
- Once the tear becomes full thickness, loads that normally are distributed through the entire intact tendon often are transmitted at the torn margins of the rotator cuff tendon; this process produces a “zipper effect” and extends or unzips the tendon from the tuberosity.

9. What is an undersurface rotator cuff tear?

Undersurface rotator cuff tears are caused by rupture of the deep tissues of the rotator cuff that attach to the tuberosity. Undersurface tears, in fact, are partial-thickness tears of the rotator cuff on the articular surface. They can result from the natural degenerative process that affects the shoulder, but often are noted in younger overhead athletes. Undersurface tearing in overhead athletes is thought to result from repetitive eccentric tensile loading (ie, deceleration of the throwing arm).

10. What is rotator cuff arthropathy?

With massive tearing of the rotator cuff, cuff tendons slide off the humeral head. These tendons, which once served as humeral head depressors, now act as humeral head elevators and promote superior translation of the humeral head. The result is excessive wear and degeneration on both the humeral head and the undersurface of the acromion. If allowed to progress, the degeneration of the glenohumeral joint can become so significant and painful that a hemi-arthroplasty or total shoulder replacement is indicated. In severe cases of rotator cuff arthropathy, radiographs can aid in the diagnosis before surgery. Radiographs reveal sclerosis of the undersurface of the acromion (“eyebrow sign”) secondary to prolonged bone-on-bone contact (humeral head in contact with undersurface of acromion) and cystic changes of the greater tuberosity.

11. When are acromioplasty and subacromial decompression required? What are the two types?

The typical patient requiring acromioplasty and decompression is between 25 and 40 years of age, experiences recurrent pain with activity that does not always abate with rest, and has failed conservative treatment (physical therapy, medications). The two types of acromioplasty and decompression are open and arthroscopic. Some surgeons believe that a more complete decompression is accomplished with the open technique. In addition, if a large rotator cuff tear is encountered during the open procedure, it can be repaired with relative ease, whereas arthroscopic repair of a large rotator cuff tear is difficult and technically demanding.

12. Should the coracoacromial ligament be released during subacromial decompression?

The coracoacromial ligament is a static stabilizer that limits superior humeral head translation. Release of the ligament contributes to increased superior humeral head migration and degenerative processes in shoulders with a massive rotator cuff tear. Thus some surgeons believe in retaining the coracoacromial ligament and preserving the arch to limit more severe superior humeral head migration, which may lead or contribute to rotator cuff arthropathy.

13. What is the Mumford procedure?

The Mumford procedure is an excision of the distal 2 cm of the clavicle. Mumford originally intended the surgery to provide pain relief for patients suffering from acromioclavicular dislocation. Distal clavicle
excision often is performed during acromioplasty and subacromial decompression to allow even greater rotator cuff decompression. The acromioclavicular joint no longer exists, however; distal stability of the scapula is maintained through the intact costoclavicular ligaments (conoid and trapezoid).

14. What are the primary rotator cuff exercises?
The primary or “core” rotator cuff exercises involve the SITS muscles:
- **Supraspinatus**—scaption is best described as abduction in the plane of the scapula. Another form of this exercise is prone scaption, in which the patient lies prone and performs scaption from 90 degrees of elevation to approximately 120 to 150 degrees.
- **Infraspinatus**—external rotation can be performed in many different positions, such as standing or side-lying.
- **Teres minor**—prone extension with external rotation is preferred. The teres minor is also an external rotator but seems to have greater electromyographic activity when external rotation is combined with glenohumeral extension. The patient lies prone with the arm hanging off the table and then extends the shoulder level with the horizon while maintaining the shoulder in external rotation.
- **Subscapularis**—internal rotation can be performed in many different positions, such as standing or side-lying.

Exercise of the SITS muscles alone does not include all muscles that contribute to optimal dynamic shoulder function. The therapist also should address the axioscapular (eg, serratus anterior) and the axiohumeral (eg, pectoralis major) muscle groups.

15. What rotator cuff exercises result in the greatest electromyographic (EMG) activity of the supraspinatus, infraspinatus, and teres minor?
Side-lying external rotation results in the greatest EMG activity of the infraspinatus and teres minor (62% maximum voluntary contraction [MVC]); supraspinatus activity was greatest during prone scaption with external rotation (82% MVC). However, prone scaption is considered an advanced exercise position for the supraspinatus, and judgment must be exercised when prescribing this exercise.

16. What is primary rotator cuff impingement?
Primary impingement is a mechanical impingement of the rotator cuff beneath the coracoacromial arch and typically results from subacromial overcrowding. Factors related to primary impingement involve abnormal structural characteristics (eg, congenital anomalies of the osseous structures of the acromioclavicular [AC] joint, coracoid process, or greater tuberosity of the humerus) or tendon thickening attributable to calcific deposits, trauma, or surgery.

17. What is secondary rotator cuff impingement?
Secondary rotator cuff impingement is a relative decrease in the subacromial space caused by microinstability of the glenohumeral joint or scapulothoracic instability. Attempts by the active restraints of the glenohumeral joint to compensate for the loss of the passive restraint function of the joint capsule and ligaments result in eventual fatigue and abnormal translation of the humeral head, leading to mechanical impingement of the rotator cuff by the coracoacromial arch.

18. What is posterior (internal) impingement?
Posterior impingement often is seen in overhead athletes, such as throwers, swimmers, and tennis players. It occurs when the arm is in an elevated and externally rotated position (similar to the cocking phase in throwing). The infraspinatus and supraspinatus muscles are pinched between the posterior superior aspect of the glenoid when the upper limb is in the cocked position. The lesion occurs on the undersurface rather than the bursal side of the rotator cuff. In addition, this form of impingement is thought to be associated with anterior instability.

19. What are the typical age, gender, and occupation of patients with rotator cuff tears?
The frequency of rotator cuff tears increases significantly with age. Tears become increasingly more common after the age of 40 years. Occupations or activities that predispose the rotator cuff to pathology require excessive and repetitive overhead motions. Sports that involve throwing or repetitive overhead motions (eg, baseball pitching, tennis, swimming) also have a high prevalence of rotator cuff injuries. However, most cuff defects have a degenerative etiology. Neer reported that 40% of patients with cuff defects never performed strenuous physical work, and many heavy laborers never develop cuff defects. Fifty percent of patients with rotator cuff tears had no recollection of shoulder trauma. A high incidence (70%) of rotator cuff defects occurs in sedentary people doing light work; two thirds of cases occur in males.
20. Do shoulder dislocations lead to rotator cuff tears?
Rotator cuff tears may occur with anterior and inferior glenohumeral dislocations. The frequency of rotator cuff tears accompanying glenohumeral dislocations increases with advancing age and has been reported to exceed 30% in patients over 40 and 80% in patients over 60 years of age.

21. What classification system is used to describe the extent or size of a rotator cuff tear?
According to the grading system adopted by the American Academy of Orthopedic Surgeons, a small tear is <1 cm, a medium tear is 1 to 3 cm, a large tear is 3 to 5 cm, and a massive tear is >5 cm.

22. Do full-thickness rotator cuff tears heal?
No. Although primary healing of a full-thickness tear is unlikely, the results of nonoperative management of patients with full-thickness rotator cuff defects have demonstrated various degrees of improvement (33%–90%) in pain and overall function. Partial-thickness tears may progress to full-thickness tears if left untreated, with deterioration in function over time.

23. Describe the typical physical therapy protocol for patients with rotator cuff repair.
Rehabilitation after rotator cuff repair depends on the following factors: size of the tear, quality of the tissue, method/type of surgical repair, age of the patient, chronicity of the condition, and occupation and/or desired activities. The typical acromioplasty and open cuff repair is followed by a short period of immobilization with or without an abduction pillow—1 to 6 weeks, depending on the size of the tear and quality of repair. Early passive motion exercises (flexion, abduction, external rotation), including pendulum exercises and pulleys, begin within the first few postoperative days to prevent adhesions and loss of motion. Scapulothoracic, cervical, and elbow, wrist, and hand range of motion (ROM) exercises should be incorporated immediately. Submaximal isometrics for shoulder internal/external rotators, flexors, and abductors may begin at 3 to 4 weeks. Active assisted ROM exercises should be progressed, delaying active abduction for up to 6 to 8 weeks. Care should be taken to ensure that exercises are performed in the scapular plane whenever possible. Full ROM should be restored by 8 to 10 weeks. Rhythmic stabilization of the scapulothoracic and glenohumeral joints is incorporated later and progresses as tolerated to promote dynamic stabilization. Strengthening typically progresses from supine to side-lying, sitting, and standing. Isotonic exercises via small handheld weights or elastic tubing typically begins in 4 to 6 weeks. Further progression and rehabilitation should be based on the needs of the individual patient.

24. How is the Neer impingement test performed?
With the patient sitting or standing, the examiner places one hand posteriorly over the scapula and grasps the patient’s elbow. With the patient’s scapula stabilized, the shoulder is maximally passively flexed overhead, compressing the greater tuberosity against the anteroinferior border of the acromion. Shoulder pain and apprehension indicate a positive sign—involvement most likely of the supraspinatus and possibly of the long head of the biceps tendon.

25. How is the Hawkins-Kennedy impingement test performed?
With the patient sitting or standing and the upper extremities relaxed, the examiner forward flexes the shoulder to 90 degrees and then forcibly internally rotates the shoulder. This action pushes the supraspinatus tendon against the anterior surface of the coracoacromial arch. A positive finding is denoted by pain and apprehension.

26. Describe the reverse impingement sign.
In the presence of a positive painful arc or pain with external rotation, the patient lies supine. The examiner pushes the humeral head inferiorly while simultaneously abducting and externally rotating the shoulder. The test is considered positive for mechanical impingement if the pain is decreased or abolished.

27. Describe the cross-over impingement test.
With the patient seated, the examiner places one hand over the posterior aspect of the scapula to stabilize the trunk and with the other hand grasps the patient’s elbow. With the trunk stabilized, the examiner maximally adducts the shoulder horizontally. Superior shoulder pain indicates acromioclavicular joint pathology, whereas anterior shoulder pain may indicate subscapularis, supraspinatus, and/or long head of the biceps tendon pathology. Posterior shoulder pain may indicate pathology of the infraspinatus, teres minor, and/or posterior joint capsule.

28. What is the painful arc sign?
A painful arc refers to a particular ROM that is painful. Usually it is preceded and followed by normal, pain-free ROM and indicates mechanical compression of pain-sensitive tissue such as the supraspinatus.
or infraspinatus tendon, subacromial bursa, or bicipital tendon. The most common range for a painful arc is 60 to 120 degrees of humeral elevation.

29. **How is the supraspinatus or empty can test performed?**

   The patient stands with both shoulders abducted to 90 degrees, in the scapular plane (horizontally adducted 30 degrees), and internally rotated in such a position that the thumbs point toward the floor. The examiner applies resistance against abduction. Pain and/or weakness indicates a tear of the supraspinatus or injury to the suprascapular nerve.

30. **Describe the drop-arm test.**

   With the patient standing or sitting, the examiner passively places the involved shoulder in 90 degrees of abduction, asking the patient to lower the arm slowly to the side. A positive sign, defined as inability to lower the arm slowly to the side or reproduction of significant pain, indicates a tear in the rotator cuff.

31. **What is the lift-off sign?**

   Standing with the dorsum of the hand placed against the back pocket, the patient lifts the hand away from the back. Inability to perform this task or pain may indicate a lesion of the subscapularis. This maneuver also can produce abnormal scapular motion, indicating scapular instability, and is used to assess rhomboid muscle strength.

32. **Describe the drop sign.**

   The examiner places the patient’s arm in 90 degrees of elbow flexion, 90 degrees of abduction, and almost full external rotation. When the arm is released, the patient is asked to maintain the same position. A drop or lag indicates infraspinatus attenuation or insufficiency related to weakness often related to tearing.

33. **What are the lag signs of the shoulder?**

   - **External rotation lag sign**—examiner places the patient’s arm passively in 90 degrees of elbow flexion, 20 degrees of shoulder elevation (in scapular plane), and nearly full external rotation. The examiner then lets go of the wrist support while supporting the elbow. The test is positive for supraspinatus or infraspinatus pathology if the patient cannot maintain the position. The lag is recorded to the nearest 5 degrees.
   
   - **Internal rotation lag sign**—patient’s elbow is passively flexed 90 degrees, the shoulder is held in 20 degrees of extension and 20 degrees of elevation, and the arm is placed behind the patient’s back. The hand is passively lifted off the back, and support is maintained on the elbow but released from the wrist. Lag is recorded to the nearest 5 degrees and indicates subscapularis tearing.

34. **Describe the sensitivity and specificity of the various tests used in rotator cuff pathology.**

<table>
<thead>
<tr>
<th>Test</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
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<tbody>
<tr>
<td>Neer impingement test</td>
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<td>Hawkins-Kennedy impingement test</td>
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<tr>
<td>Speed’s test (palm-up test)</td>
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35. **Can the supraspinatus manual muscle test predict the size of a rotator cuff tear?**

   Yes, especially as it relates to large and massive rotator cuff tears. Large and massive rotator cuff tears result in approximately a 50% reduction in strength compared with the noninvolved side (testing performed in 10 degrees of shoulder abduction with focus on the supraspinatus).
36. How accurate is a clinical examination of the shoulder in predicting rotator cuff pathology?
The sensitivity of a clinical examination of the shoulder (which includes the use of various shoulder-special tests) is approximately 91% with a specificity of 75%. Data that assist in the specific diagnosis of a rotator cuff tear are age of the patient (>40 years), previous trauma (minor), and degenerative changes on radiologic examination. Thus a good clinical examination is accurate and more cost-effective than a battery of radiologic studies in diagnosing rotator cuff pathology.

37. What clinical tests are most predictive for a rotator cuff tear?
According to Murrell and Walton, three simple tests are highly predictive of rotator cuff tear: supraspinatus weakness, weakness in external rotation, and impingement sign. When all three of these clinical tests are positive, or if two tests are positive and the patient is aged 60 or older, the individual has a 98% chance of having a rotator cuff tear. Furthermore, the investigators reported that any patient with a positive drop-arm sign also has a 98% chance of rotator cuff tear. If none of the aforementioned clinical features is present, the chance of rotator cuff tear diminishes to only 5%. The predictive influence of clustering these three clinical tests is comparable to the best results for MRI and ultrasonography and, as suggested by the investigators, easier to perform and more cost-effective.

38. Which imaging modality—plain radiographs, arthrography, or ultrasonography—is most accurate in diagnosing a rotator cuff tear?
- Standard radiographs reveal bony avulsions, calcific deposits, sclerotic areas, traction spurs, and other conditions associated with rotator cuff pathology, such as AC arthritis, calcific tendinitis, tuberosity displacement, and upward displacement of the head of the humerus in relation to the glenoid and the acromion.
- The single contrast arthrogram has been considered the gold standard technique for the diagnosis of rotator cuff tears. Recent literature suggests that the arthrogram has an accuracy of 82%. In addition, it has a sensitivity of 50% and specificity of 96% when used to evaluate full-thickness rotator cuff tears. Other research has reported the arthrogram to have a 0% to 8% probability of a false negative.
- Ultrasonography, when performed by experienced clinicians, can reveal noninvasively and nonradiographically not only rotator cuff integrity but also the thickness and location of the tear(s). The diagnostic sensitivity (98%) and specificity (91%) of ultrasonography have been compared with surgical findings. Matsen et al. suggest that expert ultrasonography provides the most efficient and cost-effective method for imaging of rotator cuff tendons.

39. Are there radiographic findings associated with symptomatic rotator cuff tears?
Pearsall et al. documented rotator cuff tears (n = 40) with asymptomatic age-matched controls (n = 84) using three views: acromioclavicular joint view, AP view with 30 degrees of external rotation, and supraspinatus outlet view. They reported that subjects with rotator cuff tear demonstrate radiographic findings (eg, greater tuberosity sclerosis, osteophytes, subchondral cysts, and osteolysis) that are not observed in asymptomatic individuals. They also noted that in patients with small to moderate rotator cuff tears, acromial morphology, and spurbing were not predictive of a full-thickness rotator cuff tear.

40. How accurate is MRI in determining a rotator cuff tear?
Although commonly used, MRI is controversial. Recent literature has reported 78% accuracy, 81% sensitivity, and 78% specificity in determining full-thickness rotator cuff tears. Other researchers have reported an accuracy of 80% and sensitivity and specificity values of 92% and 93%, respectively. MRI appears to be slightly less accurate than arthrography. However, arthrography is not as good at imaging partial-thickness rotator cuff tears, whereas an MRI in the hands of a trained individual can provide valuable information about extent, location, and classification of rotator cuff pathology.

41. What are the outcomes of rehabilitation for rotator cuff disease?
Early surgical intervention is recommended in patients with rotator cuff disease who have greater than 12 months’ duration of symptoms, severe functional impairment, or a confirmed rotator cuff tear greater than 1 cm. All other patients should undergo a minimum of 18 months of conservative management, including NSAIDs, physical therapy, and subacromial injection. Of these patients, 76% can anticipate a good or excellent result in 6 to 12 months. Of patients with impingement syndrome without rotator cuff tear, 85% will experience a good or excellent result with conservative management of at least 18 months.
42. What are the expected ROM, strength, pain, and function of a patient with rotator cuff repair at 1 and 5 years?
Problems with analyzing outcomes after rotator cuff repair are as a result of variable accuracy in describing preoperative functional levels, extent and location of the tears, tissue quality, follow-up schedule, and postoperative functional status. Cofield’s investigations describing the outcomes of rotator cuff repair reported improvements in pain (averaging 87%) and patient overall satisfaction rates of 77%. Hawkins et al. reported pain relief in 86% of patients; 78% were able to perform activities of daily living (ADLs) above the level of the shoulder after repair compared with only 16% before repair. Neer et al. reported excellent (77%) or satisfactory (14%) results in 91% of patients (n = 233) after rotator cuff repair at an average follow-up of 4.6 years. Gore et al. reported subjective improvement in 95% of patients (n = 63), including significant pain relief and minimal to no limitations in ADL function at an average follow-up of 5.5 years. In the same series, flexion ROM averaged 126 degrees actively and 147 degrees passively. Matsen et al. reported that patients with intact repairs at 5-year follow-up averaged flexion of 132 degrees, external rotation (at 90 degrees of abduction) of 71 degrees, and functional internal rotation to T7. At least 12 months is required to restore strength after rotator cuff repair; the most significant increases are noted 6 to 12 months after surgery. Walker et al. reported that isokinetic abductor strength returned to 80% of normal (uninvolved shoulder) and external rotation to 90% of normal after 1 year. Rokito et al., reporting isokinetic torques after rotator cuff repair at 1 year, demonstrated side-to-side comparisons (involved/uninvolved) for flexion, abduction, and external rotation of 84%, 90%, and 91%, respectively. Brems et al. reported that the strength of the external rotators of the repaired shoulder was 71% of the uninvolved shoulder.

43. What percentage of patients undergoing rotator cuff repair has a favorable outcome?
Favorable outcomes after rotator cuff repair include reduction in pain and increases in strength, ROM, and ADL function. Favorable outcomes are achieved in more than 75% of patients undergoing rotator cuff repair. However, some studies have reported satisfactory results in upward of 90% of patients.

44. What is the clinical outcome of a patient suffering structural failure of a rotator cuff repair?
Jost et al. reported that patients who ruptured a repaired rotator cuff reported a subjective shoulder outcome score of approximately 75% of the normal study. Fifty-five percent of the subjects reported that they were very satisfied, 30% were satisfied, and 15% were disappointed with the outcome.

45. Does open or arthroscopic acromioplasty provide a better result?
According to Van Holsbeeck et al., patients receiving open or arthroscopic acromioplasty demonstrated no significant difference at a 2-year follow-up. However, Hawkins has reported 87% satisfaction with open acromioplasty versus 40% satisfaction with arthroscopic technique. Although much of the literature seems to support both open and arthroscopic techniques, interpretation is difficult because not all patients begin with the same level of soft tissue involvement.

46. Should a patient with a confirmed rotator cuff tear undergo physical therapy? Can physical therapy make a rotator cuff tear worse?
Recent evidence supports the value of a supervised nonoperative strengthening program for chronic, full-thickness rotator cuff tears, although the range in improvement and overall satisfaction varies from 33% to 90%. However, an acute partial-thickness tear may progress if rehabilitation programs address rotator cuff strengthening too aggressively and in isolation.

47. What are the options for management of an irreparable rotator cuff tear secondary to arthropathy?
Because each patient has different levels of pain and functional disability, options for the management of arthropathy vary. Patients with mild degrees of pain usually are treated with analgesics and exercise programs to maintain levels of ADL function. Shoulder arthrodesis and total shoulder arthroplasty are options in severe cases.

48. When developing an outcome measure for shoulder function, is the evaluation of strength of the opposite shoulder important to measure?
Yes; the use of the contralateral limb as an internal control eliminates confounding variables such as age. Thus shoulder outcome tools should measure involved and uninvolved shoulder strength.
49. What are some of the common physical therapy interventions for shoulder (rotator cuff) pain, and are they effective?
According to a Cochrane Database Systematic Review, the following statements can be made regarding common physical therapy interventions for shoulder (rotator cuff) pain:
- Exercise (rotator cuff) is effective in the short-term recovery in rotator cuff disease.
- Exercise (rotator cuff) has long-term benefits with respect to function.
- Exercise and nonthrust mobilization (posterior/inferior glide of glenohumeral joint) are more effective than exercise alone.
- Laser is not any more effective than placebo for rotator cuff tendinitis.
- Ultrasound is of no additional benefit than exercise (rotator cuff) alone.

50. If a patient cannot attend formal physical therapy programs after surgical repair of the rotator cuff, is a standardized home program effective?
The literature has indicated that a standardized home program (to include written and video instructions) for patients following rotator cuff repair resulted in favorable outcomes in regard to range of motion, strength, and patient-reported outcomes.

BIBLIOGRAPHY


1. How do the size, shape, and orientation of the glenoid fossa affect glenohumeral joint stability?

The glenoid cavity can be described as an irregularly shaped oval, much like an inverted comma. On the basis of studies conducted by Saha, the average height is 35 mm and the average width is 25 mm. Saha also demonstrated that in 75% of the specimens examined, the glenoid fossa was retroverted approximately 7 degrees. In the remaining 25%, the glenoid was anteverted from 2 to 10 degrees. The glenoid is also tilted from superomedial to inferolateral by an average of 15 degrees. The depth of the fossa is enhanced by the glenoid labrum, which can contribute up to 50% of the fossa’s depth.

2. Describe the passive stabilizing mechanisms for the glenohumeral joint.

Passive stability is provided by the bony geometry, glenoid labrum, limited joint volume, negative intraarticular pressure, adhesion and cohesion, and capsuloligamentous structures. The glenohumeral joint has a slightly negative pressure of $-4.0 \text{ mm Hg}$, which creates a relative vacuum. As long as the relative vacuum effect is maintained, limited joint volume does not allow the joint surfaces to be easily distracted or subluxated.

The close match of the articular surfaces produces intermolecular forces of surface tension, cohesion, and adhesion, which provide continued coupling of the humerus to the glenoid. Adhesion refers to the attraction of unlike substances (joint fluid to bone), whereas cohesion refers to the attraction of like substances (joint fluid to joint fluid). In addition, the glenoid labrum deepens the fossa by 5 mm in an anteroposterior direction and by 9 mm in the superior and inferior direction.

3. What are the primary static stabilizers of the glenohumeral joint?

The superior, middle, and inferior glenohumeral ligaments provide anterior stability. With the arm in the adducted position, the superior glenohumeral and coracohumeral ligaments act in a suspensory role to resist inferior translation of the humeral head. As the arm is brought up into the mid-range of abduction, the middle glenohumeral ligament provides more of a stabilizing role. In addition, as the arm is abducted to 45 degrees and beyond, the anterior and posterior portions of the inferior glenohumeral ligament complex become the stabilizers to resist inferior translation. Above 90 degrees of abduction, the inferior glenohumeral ligament becomes the primary stabilizing function. The anterior band of the inferior glenohumeral ligament complex is the primary restraint to anterior translation at 90 degrees of abduction. Posterior stabilization of the glenohumeral joint with the arm at 90 degrees of abduction is provided primarily by the posterior band of the inferior glenohumeral ligament complex.

4. Describe the mechanisms for achieving dynamic stability at the glenohumeral joint.

Stability is achieved through three mechanisms: 1) joint compression of matching concave-convex surfaces as the muscles press the humeral head into the fossa; 2) synergistic, coordinated contraction of the rotator cuff muscles, acting to steer the humeral head into the glenoid in different positions of arm rotation; and 3) dynamization or tensioning of the glenohumeral ligaments through the direct attachment or blending of the rotator cuff tendons into the glenohumeral capsule and ligaments. In addition, the glenoid fossa has an upward, lateral, and forward orientation that serves as a shelf for the humeral head. This source of stability is provided by the normal muscle control of the scapular protractors. When these muscles (serratus anterior, upper trapezius) become weakened, dynamic stability may be lost and the humeral head may simply slide down and off the near vertical glenoid fossa.

5. What is the most common direction and mechanism of injury causing shoulder instability?

Subcoracoid anterior dislocation is the most common direction of dislocation. The most common mechanism of injury for anterior shoulder dislocation is an indirect force with the arm in an abducted, extended, and externally rotated position. The majority of dislocations results from trauma.
6. What is the most common nerve injury after anterior shoulder dislocation?
Injury to the axillary nerve has an overall incidence of approximately 30%. The risk of axillary nerve injury increases with age, duration of dislocation, and force of trauma. The most common type of axillary nerve injury is traction neurapraxia. Because the axillary nerve originates at the posterior cord of the brachial plexus and its anterior branch (humeral circumflex) wraps directly around the humeral wall in the area of the surgical neck, the nerve can be exposed to trauma. Anterior dislocation may cause traction to the portion of the axillary nerve lying in close relation to the capsular structures. Most patients respond to conservative treatment over 10 weeks.

7. Describe the most common mechanism of posterior shoulder dislocation.
A posterior dislocation results most commonly from axial loading of the arm in an adducted, flexed, and internally rotated position. The classic mechanism of injury is either a blow to the front of the shoulder or a fall onto the outstretched arm. Lesser tuberosity fractures are common and often cause the humeral head to become locked in the dislocated position. Posterior dislocations are less common than anterior and account for only 2% to 4% of all dislocations.

8. Why is posterior shoulder dislocation more likely than anterior dislocation after electric shock or convulsive seizures?
Electric shock and convulsive seizures can result in violent contracture of all muscle groups surrounding the shoulder girdle. The combined strength of the latissimus dorsi, pectoralis major, and subscapularis overwhelms the infraspinatus and teres minor muscles by virtue of greater muscle bulk. As a result, the stronger internal rotators simply overpower the relatively weaker external rotators, resulting in a posterior dislocation.

9. What is multidirectional instability with atraumatic onset?
Multidirectional instability is a symptomatic glenohumeral subluxation or dislocation in more than one direction. The basic pathologic changes of multidirectional instability include 1) a loose, redundant, or torn joint capsule; 2) a lax ligamentous mechanism; and 3) a weakened musculotendinous system.

10. In describing shoulder instability, what is meant by the acronym TUBS?
- **T**—Traumatic onset
- **U**—Unidirectional (anterior)
- **B**—Bankart lesion (usually present)
- **S**—Surgery (success rate with nonoperative treatment is >20%)

11. What is meant by the acronym AMBRI in describing shoulder instability?
- **A**—Atraumatic onset
- **M**—Multidirectional in nature
- **B**—Bilateral (usually)
- **R**—Rehabilitation (success with conservative treatment is usually >80%)
- **I**—Inferior capsular shift (procedure of choice if conservative treatment fails)

12. What type of lesion is characterized by the acronym ALPSA?
The ALPSA lesion as originally described by Neviaser stands for anterior labroligamentous periosteal sleeve avulsion. This will often accompany a traumatic anterior dislocation and is characterized by the labrum and periosteal sleeve of the anterior glenoid being avulsed and displaced medially.

13. What type of lesion is characterized by the acronym HAGL?
The HAGL lesion occurs with traumatic dislocation when the arm is forced into a hyperabducted position. The acronym stands for humeral avulsion of the glenohumeral ligament.

14. Describe the load-shift test.
The load-shift test allows evaluation of glenohumeral translation. A compressive axial load is applied to the humeral head to reduce it into the glenoid. This reduction is important because the humeral head may be resting in a subluxated position, which may give a false sense of the direction of the instability. Anterior and posterior forces then are placed on the proximal humerus, and the direction and degree of translation are determined.

15. Describe the anterior release test.
The patient is supine and the shoulder is placed in 90-degree abduction and 90-degree external rotation (ER) (apprehension position), during which a posterior-directed force is applied to the humeral head (anterior surface of shoulder). The posterior force is then released; if the patient experiences pain and apprehension, then the test is considered positive.
16. What are the sensitivity and specificity values of commonly performed shoulder instability tests?

<table>
<thead>
<tr>
<th>Test</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Load-shift (under anesthesia)</td>
<td>83*</td>
<td>100</td>
</tr>
<tr>
<td>Sulcus sign</td>
<td>Not reported</td>
<td>Not reported</td>
</tr>
<tr>
<td>Apprehension</td>
<td>57</td>
<td>100</td>
</tr>
<tr>
<td>Relocation</td>
<td>30</td>
<td>50</td>
</tr>
<tr>
<td>Anterior release</td>
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<td>89</td>
</tr>
<tr>
<td>M&amp;IGHL/PC tests</td>
<td>Not reported</td>
<td>Not reported</td>
</tr>
</tbody>
</table>

*Hawkins suggests it may be overly sensitive.

17. What type of grading scheme is used to assess increased glenohumeral translation?

Anterior translation of 25% or less of the humeral head diameter is considered normal. Hawkins suggested a grading system that may be more appropriate for reporting the test results than distance or percentages:

- **Grade I**—humeral head can be felt to ride up the face of the glenoid to the glenoid rim but cannot be felt to move over the rim edge. Grade I corresponds to approximately up to 50% of humeral head translation.
- **Grade II**—humeral head can be felt to move over the glenoid rim but reduces with release of pressure, corresponding to clinical subluxation. For grade II the humeral head has more than 50% translation.
- **Grade III**—head remains dislocated on release, corresponding to clinical dislocation.

18. Describe the clinical tests for posterior shoulder instability.

In addition to the load-shift test, posterior instability can be assessed with the jerk test. The arm is flexed to 90 degrees with internal rotation (IR), and an axial load is delivered to the shoulder in a posterior direction. The arm is brought into a horizontally adducted position, and posterior slippage is noted. The arm then is brought back into a horizontally abducted position. A jerk may be experienced when the humeral head relocates onto the glenoid fossa.

19. What radiologic studies and views are best suited for confirming or evaluating shoulder instability?

The recommended views in a trauma series include a true anteroposterior (AP) view, a true scapular lateral view, and an axillary view. The most commonly obtained views of the shoulder include the AP view of the shoulder with the humerus in both internal and ER, a true AP of the glenoid view, a scapulolateral (Y) view, an axillary view, a West Point projection, and a Stryker notch view.

20. Describe the Hill-Sachs and reverse Hill-Sachs lesions.

The Hill-Sachs lesion is a compression fracture of the posterolateral aspect of the humeral head. It results from impact to the anteroinferior rim of the glenoid during an anterior dislocation of the shoulder. A reverse Hill-Sachs lesion involves a compression fracture of the anteromedial humeral head as the result of a posterior dislocation.

21. What is the suggested radiologic view to visualize a Hill-Sachs lesion?

The Hill-Sachs lesion is demonstrated best by either the IR or the Stryker notch views; each has a sensitivity of 92%. The detection of a Hill-Sachs lesion is prognostically important because patients with a Hill-Sachs lesion may be prone to redislocation.

22. What is a Bankart lesion? What is its significance?

A Bankart lesion is an avulsion or detachment of the anterior portion of the inferior glenohumeral ligament complex and glenoid labrum off the anterior rim of the glenoid. Although a Bankart lesion can contribute to increased translation of the humeral head, complete dislocation requires associated capsular injury. Bankart lesions can contribute to recurrent instability.

23. Describe the clinical presentation of a posterior shoulder dislocation.

Observation is often difficult because most patients hold the shoulder in the traditional sling position of adduction and IR. ER usually is limited, and it is not uncommon to find the posteriorly dislocated shoulder locked into IR secondary to a fracture of the lesser tuberosity. Observation usually reveals a prominent coracoid process and a flattening of the anterior aspect of the shoulder.
24. What is the suggested initial medical treatment for anterior shoulder dislocation? Why is early relocation important?

Initial treatment includes application of ice and use of a sling. Acute glenohumeral dislocations should be reduced as quickly and gently as possible because early relocation quickly reduces stretch and compression of neurovascular structures, minimizes the degree of muscle spasm that must be overcome to reduce the joint, and prevents progressive enlargement of the humeral head defect in the locked dislocation.

25. Following reduction for an anterior dislocation, should the arm be immobilized in IR or ER?

Postreduction management after traumatic anterior shoulder dislocation is controversial. A 10-year prospective study by Hovelius comparing immobilization with no immobilization found no difference in recurrence rates. The position of immobilization is also controversial. Itoi investigated immobilizing the arm in IR or ER after initial traumatic anterior dislocation. The recurrence rate was approximately 30% in the IR group and 0% in the ER group at a mean follow-up time of 15.5 months, suggesting ER as the position of immobilization. However, it is still more customary to immobilize the arm in IR at this time.

26. What is the most common complication in managing a traumatic anterior dislocation?

Recurrence is the most common complication. Other complications include fractures of the humerus, vascular injuries, neural injuries, and rotator cuff tears (more common in patients >40 years).

27. What accounts for the high incidence of recurrent dislocation?

Several factors have been identified as contributing to recurrence and instability. Age at the time of onset correlates most closely to recurrence. Patients under the age of 20 years may have a recurrence rate up to 80%, whereas after the age of 40 the rate drops to under 10%. Males have a higher recurrence rate than females, and most recurrences are seen within 2 years of the initial traumatic dislocation. The recurrence rate varies inversely with the severity of the initial trauma. If dislocation occurs a second time in younger patients, the chance of frequent recurrence is almost 100%.

28. What is the incidence of associated rotator cuff tears in patients older than 40 years? Why is the rate increased?

The incidence of rotator cuff tears after acute dislocation in patients older than 40 years ranges from 35% to 86%. The reason for the variability in numbers is the unknown amount of rotator cuff pathology before the initial dislocation. With dislocation of the humeral head anteriorly, the anterior and/or posterior structures are disrupted. With dislocations in younger patients, the anterior capsuloligamentous complex tends to disrupt because it is less strong than other tissues of the shoulder. In older patients, the posterior structures (rotator cuff and greater tuberosity complex) are weaker by attrition and tend to disrupt, leaving the anterior capsuloligamentous complex intact.

29. What nonoperative management is appropriate after anterior shoulder dislocation?

After an initial period of immobilization, a regimen of shoulder rehabilitation should be implemented. Initially, range of motion exercises are instituted to help prevent stiffness. Positions of abduction and ER should be avoided to prevent excessive stress on the anterior capsule. Strengthening of the shoulder musculature is of paramount importance to improve dynamic stability. Because the capsular stabilizing structures are compromised, the shoulder has a greater dependence on dynamic stabilizing mechanisms. Early focus is placed on the stabilizers of the scapula. The scapula must provide a stable base on which the humerus can rotate and maintain the glenoid in a position that provides maximal congruence with the humeral head. The core scapular exercises are scaption, protraction, retraction, and seated press-up.

Once scapular stability is addressed, emphasis is placed on reestablishing the strength of the rotator cuff musculature, which is the main dynamic stabilizer of the glenohumeral joint. Exercises should be performed in the scapular plane, which provides the greatest congruence between the humeral head and glenoid and minimizes the stress placed on the anterior capsule. The supraspinatus can be isolated with prone horizontal abduction and ER. Activation of the teres minor and infraspinatus draws the humeral head posteriorly and thus unloads the stress on the damaged anterior structures. These two muscles are best isolated with prone ER with the arm positioned in 90-degree abduction. In addition to strengthening exercises, proprioception exercises should be used to enhance the patient’s sense of position.
30. What nonoperative management is appropriate after posterior shoulder dislocation?
Reduction is accomplished by longitudinal forward traction on the arm with the elbow bent, accompanied by anterior pressure on the humeral head. The arm then is brought into an adducted, externally rotated, and internally rotated position to reduce the humeral head back into the glenoid fossa. Principles of nonoperative treatment include pain management, activity modification, and a shoulder strengthening program involving the scapular and rotator cuff musculature. Nonoperative treatment produces superior results in posterior instability compared with anterior instability. The joint is immobilized for only 2 to 3 weeks in a handshake cast. Integral to the strengthening program is the periscapular and rotator cuff musculature. ER and posterior deltoid strengthening is emphasized during rehabilitation. Push-ups and bench press activities should be avoided. The patient must be instructed to avoid activities that place the shoulder at the limits of flexion, IR, or horizontal adduction. Otherwise the shoulder may redislocate.

31. What nonoperative management is appropriate for multidirectional instability?
Overall, patients tend to respond well to rehabilitation. Aggressive physical therapy with strengthening of the scapular stabilizers and rotator cuff musculature frequently provides sufficient dynamic stability. If the patient does not respond to conservative treatment, an inferior capsular shift should be included as part of the surgical procedure.

32. Describe the modern surgical management of patients for whom operative treatment is advisable.
Several different surgical procedures are used to control shoulder instability. The success and/or failure rate for each is quite variable and highly dependent on the skill of the surgeon. Currently, the gold standard is some variation of capsulorrhaphy, which directly affects the size and/or orientation of the glenohumeral capsule:
- Bankart repair—suturing of the anterior capsule and labrum to the anterior glenoid rim
- Capsular shift—tightening of the joint capsule, depending on the precise amount and location of laxity
- Staple capsulorrhaphy—securing the detached anterior capsule and labrum onto the glenoid
- Thermal capsulorrhaphy—thermal shrinkage of the capsular collagen tissue to restore normal stability
- Putti-Platt procedure—subscapularis and capsule shortening

33. How does the outcome of immediate surgical stabilization compare to the nonoperative management of shoulder instability in the young, healthy adult?
Kirkley conducted a prospective randomized clinical trial comparing the effectiveness of immediate arthroscopic stabilization versus immobilization and rehabilitation in first time, traumatic anterior shoulder dislocations. At an average of 32 months’ follow-up, a significant reduction in redislocation and an improvement in disease-specific quality of life were afforded by early arthroscopic stabilization in patients less than 30 years of age with a first-time, traumatic anterior dislocation of the shoulder.

The Bankart lesion was noted in a very high percentage of traumatic first-time dislocations—97% in one series of patients who underwent arthroscopic evaluation soon after their injury. The standard of care in the overhead athlete is early repair of the capsular structures. With the Bankart lesion, the capsulolabral complex avulses from the glenoid. If the anteroinferior labrum and capsule do not heal in their anatomic position, the depth of the concavity will be lost in that isolated area, thereby contributing to an increased recurrence rate, especially when the arm is placed in a position of abduction and ER. Early stabilization in athletic high-risk patients should diminish progressive soft tissue and bony damage.

34. What are superior labrum anterior and posterior (SLAP) lesions?
SLAP lesions most often result from a sudden downward force on a supinated outstretched upper extremity or from a fall on the lateral shoulder. Patients complain of popping and sliding of the shoulder, especially with overhead activities. The average time to diagnosis from onset of symptoms is about 2.5 years.

35. What are the types of SLAP lesions?
In 1990 Stephen Snyder coined the name SLAP lesion to describe a more extensive injury pattern involving the superior labrum. Snyder further classified superior labrum disorders into four types:
- Type I—degenerative fraying of the labrum
- Type II—avulsion of the superior labrum and biceps tendon
- Type III—bucket-handle tears of the superior labrum
- Type IV—same as grade II or III with extension into the biceps tendon
Maffet and co-workers described three additional types of SLAP lesions:

- Type V—an anterior-inferior Bankart lesion that propagates superiorly to the biceps tendon
- Type VI—an unstable flap tear of the labrum with separation of the biceps anchor
- Type VII—a superior biceps-labral detachment that extends anteriorly beneath the middle glenohumeral ligament

BIBLIOGRAPHY


CHAPTER 39 QUESTIONS

1. With the arm positioned in 45 degrees of abduction, which of the glenohumeral ligaments is the primary restraint to ER?
   a. Inferior glenohumeral ligament
   b. Middle glenohumeral ligament
   c. Posterior glenohumeral ligament
   d. Superior glenohumeral ligament
2. Which of the following surgical procedures is performed for recurrent anterior instability or dislocation of the glenohumeral joint and involves reattachment and repair of the capsulolabral complex to the anterior rim of the glenoid?
   a. Bankart repair
   b. Anterior capsular shift
   c. Hill-Sachs repair
   d. Repair of a SLAP lesion

3. This lesion is seen in recurrent anterior glenohumeral dislocation as an indentation or compression fracture of the articular surface of the humeral head as created by the sharp edge of the anterior glenoid as the humeral head dislocates up and over it:
   a. Hill-Sachs lesion
   b. Bankart lesion
   c. Reverse Hill-Sachs lesion
   d. Reverse hills sachs is a real condition.

4. Which of the following does NOT contribute to the passive stability of the glenohumeral joint?
   a. Negative intraarticular pressure
   b. Glenoid labrum
   c. Osseous geometry
   d. Supraspinatus

5. A bicyclist falls off of a bike onto his outstretched arm, with the arm adducted and internally rotated. He has symptoms of severe pain, inability to move his shoulder, and an apparent prominence in the infraspinatus region. The most likely diagnosis is:
   a. Anterior shoulder dislocation
   b. Brachial plexus injury
   c. Posterior shoulder dislocation
   d. Rotator cuff tear

6. In the glenohumeral joint, the presence of a sulcus sign is a reflection of laxity of what structure?
   a. Inferior capsular ligament
   b. Coracoacromial ligament
   c. Supraspinatus tendon
   d. Superior glenohumeral ligament
   e. Biceps tendon
1. Describe the epidemiology of adhesive capsulitis.
Adhesive capsulitis, or “frozen shoulder,” is more common in females than males and occurs most often in the age range of 40 to 60 years. Bilateral involvement is seen in about 12% of patients. The incidence is 2% in the general population and 10% to 35% in diabetic patients.

2. What are the predominant cell types in adhesive capsulitis? What growth factors are present?
Fibroblasts and myofibroblasts are the predominant cell types. The presence of type III collagen in those with adhesive capsulitis indicates new deposition of collagen within the capsule. The frequency of staining for transforming growth factor-β, platelet-derived growth factor, and hepatocyte growth factor is greater in adhesive capsulitis tissue than in tissue from patients with nonspecific synovitis. Intercellular adhesion molecule-1 is present in the capsule of patients with frozen shoulder.

3. Are other cell types present in the capsule of adhesive capsulitis?
Increased chondrogenesis is also seen along with fibrosis in the capsule of those with frozen shoulder.

4. Is there a role for the use of hyaluronan in the treatment of adhesive capsulitis?
Hyaluronan may modulate cell proliferation and expression of the mRNA of adhesion-related procollagens and cytokines in the synovial fluid and capsule, thus preventing the progression of adhesion formation in patients with frozen shoulder.

5. Define primary and secondary adhesive capsulitis.
Lundberg described stiff shoulder with insidious onset as primary adhesive capsulitis. Frozen shoulder after some type of trauma or inciting event is classified as secondary adhesive capsulitis.

6. What imaging techniques are useful for the diagnosis of adhesive capsulitis?
Plain films are useful in excluding other pathology, but no pathognomonic changes are associated with capsulitis. Arthrography is the gold standard for diagnosis. The normal capsular volume decreases from 25 ml to about 6 ml with obliteration of the biceps sheath, axillary fold, and subscapular bursa. Dynamic ultrasonography is 91% sensitive and 100% specific for the detection of capsulitis. Thickening of the inferior capsule can also be evaluated by ultrasonography.

7. What MRI findings are associated with adhesive capsulitis?
Thickening of the coracohumeral ligament (CHL) to >4 mm is 95% specific and 59% sensitive for the diagnosis of adhesive capsulitis. Thickening of the capsule in the rotator interval to >7 mm has a specificity of 86% and sensitivity of 64%. Obliteration of the fat triangle between the CHL and the coracoid process was 100% specific but 32% sensitive.

8. Describe the natural resolution of adhesive capsulitis.
Reeves described the three classic stages of adhesive capsulitis:
- Early painful stage (freezing)—lasts 2 to 9 months; patients have diffuse pain and difficulty sleeping on the affected side; patients begin to have restricted movement secondary to pain
- Stiffening stage (freezing)—lasts 4 to 12 months; progressive loss of ROM and decreased function are noted
- Recovery stage (thawing)—lasts 5 to 24 months, with gradual increases in ROM and decreased pain

9. What outcomes are associated with the natural resolution of adhesive capsulitis?
The time to resolution is quite variable, averaging 12 to 36 months. Approximately 20% to 60% of patients have some limitation in ROM and residual pain for up to 10 years.
10. What are the outcomes associated with a home stretching program for adhesive capsulitis?
Griggs et al. found that 90% of patients reported a satisfactory outcome. However, after 22 months, patients still had restricted range of motion compared with the contralateral side. Abduction was 145 degrees, flexion was 155 degrees, passive internal rotation at 90 degrees of abduction was 29 degrees, and external rotation was 60 degrees.

11. What factors have been proposed in the pathogenesis of adhesive capsulitis?
Cervical spine disorders, autoimmune disorders, tendinitis, hypothyroidism, diabetes, hormonal disorders, and poor posture have been postulated as predisposing factors for capsulitis. Hypercholesterolemia has been found to be an independent risk factor for adhesive capsulitis.

12. What is the role of physical therapy for the treatment of capsulitis?
Exercise has been found to be more effective than modalities, nonsteroidal antiinflammatory drugs, or steroid injections. Nicholson found that mobilization significantly improved ROM into abduction. However, mobilization offered no significant advantage over exercise alone in other motions. One study found mobilization to be more effective than manipulation for increasing ROM. Numerous case studies have found mobilization to be effective in treating adhesive capsulitis.

13. Do end range mobilization techniques improve range of motion in patients with adhesive capsulitis?
Vermeulen et al. found that passive abduction increased from 96 to 159 degrees, flexion increased from 122 to 154 degrees, external rotation increased from 21 to 41 degrees, and the mean glenohumeral capsular volume increased from 10 to 15 ml.

14. Does translational mobilization increase glenohumeral abduction?
In a cadaver model Hsu et al. found increases in glenohumeral abduction after both ventral and dorsal translational mobilization. Small improvements in external rotation were found after ventral translational mobilization.

15. Does the direction of the mobilization matter?
Posterior mobilization has been found to improve external rotation greater than anterior mobilization, likely as a result of stretching the rotator interval.

16. What outcomes are associated with steroid injections for capsulitis?
Although steroid injections may provide transient relief of pain, no studies show conclusive evidence that they increase ROM or function. This may be due to the fact that few studies differentiate between injections given at different stages of the disease.

17. How does translational manipulation differ from traditional long lever manipulation?
Translational manipulation uses linear forces applied at the humeral head to restore normal kinematic gliding associated with glenohumeral movements. By avoiding long lever forces, translational manipulation minimizes the stress applied to the brachial plexus and the glenohumeral, acromioclavicular, scapuloclavicular, and scapulothoracic joints.

18. What outcomes are associated with traditional long lever manipulation under anesthesia for capsulitis?
Despite reported complications of dislocation, fracture, brachial plexus injury, rotator cuff tearing, and failure to regain ROM secondary to pain, manipulation under anesthesia remains a proven treatment technique with a low incidence of the previously mentioned complications. Hill and Bogumill reported significant increases in ROM immediately and in the long term (flexion = 139 degrees, abduction = 143 degrees, external rotation = 54 degrees, and internal rotation = 63 degrees) after manipulation.

19. Does manipulation tear the rotator cuff?
Although the inferior capsule is torn, it is unusual for any tear of the rotator cuff to occur.

20. What outcomes are associated with translational manipulation under anesthesia for capsulitis?
Placzek et al. reported significant increases in ROM immediately and in the long term (flexion = 163 degrees, abduction = 163 degrees, external rotation = 84 degrees, and internal rotation = 69 degrees) after manipulation. Furthermore, pain was significantly reduced (7.6/10 down to 1.5/10), and function was significantly increased (Wolfgang score of 5.5/16 increased to 14.1/16).
21. What outcomes are associated with the brisement technique (arthrographic distention)?
Distention arthrography in general provides minimal immediate increases in ROM. However, it speeds improvement in ROM over the next several weeks to months. The steroids and local anesthetics used usually provide some pain relief.

22. What outcomes are associated with arthroscopic release for capsulitis?
In general, ROM gains have been somewhat less than with manipulation under anesthesia. The best results have been published by Jerosch et al., where abduction improved from 75 to 165 degrees, external rotation improved from 3 to 75 degrees, external rotation and abduction improved from 4 to 81 degrees, and internal rotation improved from 17 to 59 degrees. Arthroscopic capsular release may be particularly helpful in recalcitrant cases in which therapy and manipulation have failed.

23. Is traditional long lever manipulation under anesthesia associated with intraarticular lesions?
Loew et al. found that in a group of 30 patients 22 had localized synovitis in the rotator interval and 8 had disseminated synovitis. After manipulation, the capsule was ruptured superiorly in 11 patients, anteriorly in 24, and posteriorly in 16. In 4 patients an iatrogenic SLAP lesion was found, 3 had partial tearing of the subscapularis, and 4 had anterior labral detachments. Two patients had tears of the middle glenohumeral ligament. Although manipulation is effective for increasing range of motion, certain iatrogenic intraarticular damage can occur.

BIBLIOGRAPHY
CHAPTER 40 QUESTIONS

1. Adhesive capsulitis
   a. Is self-limiting within 6 months of onset
   b. Resolves with short-term NSAID use
   c. Is usually precipitated by a rotator cuff tear
   d. Lasts 15 to 18 months, often with some ongoing limitations

2. Which of the following cannot be used to diagnose adhesive capsulitis?
   a. Ultrasound
   b. MRI
   c. X-ray
   d. Arthrogram

3. Which of the following does not play a role in the pathogenesis of adhesive capsulitis?
   a. Hypercholesterolemia
   b. Thyroid disorders
   c. Diabetes mellitus
   d. Hypogonadism
1. Who is the typical patient who might undergo total shoulder arthroplasty (TSA)?

Traditionally the age of the patient who undergoes TSA is 55 to 70 years. However, in cases of arthritis due to previous dislocation and avascular necrosis, the age may be in the range of 40 to 50 years. Approximately equal numbers of males and females undergo TSA.

2. How many TSAs, reverse TSAs, and hemiarthroplasties are performed each year?

A recent review of Medicare Part A claims by Day revealed that 31,002 shoulder arthroplasties were performed in 2010 and were categorized as follows: 42% were TSAs, 37% were reverse TSAs, and 21% were hemiarthroplasties. In another study reported, there were approximately 20,178 hemiarthroplasties and 26,773 TSAs performed in 2008 in the United States. This is up from 15,000 TSAs reported in 1998.

3. What are the typical indications for TSA?

Medical indications for TSA include osteoarthritis, osteoarthritis secondary to previous trauma, such as shoulder instability or surgery, rheumatoid and other inflammatory arthritis, avascular necrosis of the humeral head, and rotator cuff tear arthropathy. Patients often present with shoulder pain, functional limitations in motion, and radiographic deterioration of the glenohumeral joint. Primary glenohumeral degenerative joint disease presents with central wearing of the humeral head, known as the “Friar Tuck” pattern of central baldness. The glenoid surface wears out primarily on the posterior margin, predisposing the joint to posterior subluxation.

4. What are the typical contraindications for TSA?

- Active infection
- Neurologic compromise of either deltoid or rotator cuff musculature
- Neurotrophic shoulder
- Unrealistic expectation of shoulder function after surgery
- Lack of appropriate motivation to perform rehabilitation program after surgery

5. What is the difference between unconstrained, constrained, and a reverse TSA?

(A) Unconstrained total shoulder arthroplasty with standard polyethylene glenoid component. (B) Semiconstrained total shoulder arthroplasty with superior hooded glenoid component.
Unconstrained TSA more closely resembles normal anatomic configuration of the glenohumeral joint and allows more humeral motion (see figure). Constrained TSA utilizes a ball-and-socket design that makes the glenoid function more like a true ball and socket but reduces humeral motion.

A third type of prosthesis, called a reverse prosthesis, has been developed to place the ball component on the glenoid side and the socket on the humeral side (see figure). The advantage of this design is for patients with a deficient cuff to place the deltoid in a better biomechanical position by medializing the center of rotation so more muscle fibers can assist in elevation.

6. What is the difference between hemiarthroplasty and TSA?

Hemiarthroplasty is the replacement of only the humeral component and is also known as humeral head replacement (HHR). A hemiarthroplasty is indicated when the humeral head is deteriorated or fractured but the glenoid surface is intact. Hemiarthroplasty is the surgery of choice if the patient has insufficient glenoid bone to support a glenoid component. When the physical demands are heavy after surgery, a hemiarthroplasty is indicated. Hemiarthroplasty is indicated when arthritis and rotator cuff deficiencies
coexist. A badly eroded glenoid cannot stabilize a glenoid component securely, and a nonfunctional rotator cuff produces unbalanced muscular forces on the glenoid, leading to loosening.

**TSA** is the replacement of both humeral head and glenoid. This procedure is undertaken when both joint surfaces are damaged and both are reconstructible. TSA is recommended in patients with osteoarthritis and rheumatoid arthritis.

7. **Is there a benefit for choosing HHR versus TSA?**

   It appears that the TSA is the best option for the treatment of patients with glenohumeral arthritis. Consistent relief of pain and improved function in multiple studies support this statement. There are times when the bone stock of the glenoid cannot support the prosthesis or the deficiency of the rotator cuff requires the use of HHR.

8. **What factors and conditions should be present for a person to consider undergoing a TSA or HHR?**

   Patients who have failed conservative management should have a functioning deltoid and rotator cuff musculature, demonstrate appropriate motivation toward rehabilitation, and be in sufficient health to undergo major surgical intervention. Patients without erosion of the glenoid have been found to have improved function after a HHR. Patients suffering from osteoarthritis or osteonecrosis tend to have higher levels of function after surgery than patients with rheumatoid arthritis and cuff tear arthropathy.

9. **Can an HHR be converted to a TSA if the HHR fails?**

   Yes, but it should be considered as a salvage procedure because a recent report found that nearly 50% of the 16 patients who underwent the procedure were unsatisfied with their outcome at 2 years. The advent of the modular components could lead one to believe that converting to a total shoulder replacement would not be difficult; however, this recent evidence suggests that is not the case.

10. **What postoperative complications are associated with TSA and reverse TSA?**

    The incidence of complications after a TSA is approximately 10%. According to Sperling, common complications are due to fractures, infection, anterior and posterior instability with an anatomic prosthesis, rotator cuff tear, and glenoid component loosening. A study published in 2006 found 53 surgical complications on 431 total shoulder arthroplasties (12%). In this study tearing of the rotator cuff accounted for approximately 32% of postoperative complications, fractures for approximately 24%, brachial plexopathy for approximately 15%, subluxation for approximately 9%, and dislocation for approximately 7%. Humeral loosening, humeral and glenoid loosening, infection, hematoma, and long head of biceps rupture individually accounted for approximately 1% of postoperative complications.

    The incidence of complications after a reverse TSA ranges from 15% to 50%. According to Zumstein, common complications are due to scapular notching, instability, infection, glenoid loosening, and scapular fractures. A systematic review in 2011 reviewed 782 cases and identified these most common complications: Scapular notching accounted for 35% of the complications; instability accounted for 5% of the complications; infection and glenoid loosening each accounted for 4% of the complications; and acromion and scapular spine fractures both accounted for 2% of the reverse TSA complications.

11. **What causes components to loosen?**

    Symptomatic loosening of glenoid and humerus components occurs in 3.5% of patients with TSA. The many contributing factors include glenoid preparation, soft tissue balancing, wear debris, bone reabsorption, prosthetic design, component geometry, and biomaterials. One major concern is the eccentric load placed on the glenoid component by the humeral component, particularly if the humerus has migrated superiorly. The humerus can migrate superiorly because of rotator cuff tear, poor humeral fixation, and soft tissue imbalance. During arm elevation the eccentric load of a proximal migrated humeral component can produce a “rocking horse” effect on the glenoid component that loosens the glenoid component.

12. **What are the postoperative goals after TSA?**

    The primary goal is to relieve pain. Approximately 90% of patients report no or slight pain after hemiarthroplasty or TSA. The secondary goal is to restore normal function, specifically shoulder range of motion, stability of the components, upper extremity strength, smooth motion between prosthetic components, prosthesis and bone interface, and smooth motion between proximal humerus, rotator cuff, and rotator cuff outlet.
13. How long does a TSA last?
Failure of prosthesis was defined as need for reoperation or patient dissatisfaction in a multicenter study of 470 cases of TSA. This study reported that, at 5-year follow-up, 3% of the procedures had failed. A smaller study of 53 operations, using similar criteria, reported that, at 11-year follow-up, 27% had failed. A study of 29 patients who had surgery at the age of 50 or younger revealed that 84% of the TSA prostheses were still intact 20 years postoperatively, with approximately 50% of these patients reporting satisfactory or better results.

14. How much pain, function, and motion improvement is expected after hemiarthroplasty, TSA, or reverse TSA?
Recent publications have demonstrated that all of these procedures can produce improvements in function, decrease pain, and improve range of motion. For total shoulder arthroplasties at an average of 31.4 months’ follow-up, 73 patients were found to have an average pain reduction on the numerical pain rating scale (NPRS) of 6 points out of a 10-point scale. Several different functional scales are used to assess patients undergoing a TSA, but, overall, the typical patient reports approximately 40% of normal function before surgery and improves to approximately 80% to 90% of normal function after surgery and rehabilitation. Active elevation improved approximately 42 degrees and external rotation 36 degrees.

Hemiarthroplasties at an average of 24 months’ follow-up were found to have an average pain reduction on the McGill pain visual analog scale of 51 points out of 100 points. Several different functional scales are used to assess patients undergoing a hemiarthroplasty, but, overall, the typical patient reports approximately 30% to 40% of normal function before surgery and improves to approximately 70% to 80% of normal function after surgery and rehabilitation. Forward elevation has been seen to improve up to 50 degrees and external rotation 20 degrees after hemiarthroplasty.

In a recent study by Jobin, patients after primary reverse TSA at an average of 16 months’ follow-up, were found to have an average improvement in their American Shoulder and Elbow Surgeons pain and function score, which improved by 44% from 24 to 69 on a 100-point scale. Active forward elevation improved from 38 degrees to 144 degrees while active external rotation at side improved from 11 degrees to 23 degrees. Active external rotation with arm abducted improved on average from 18 degrees to 44 degrees.

15. Can a patient participate in sports after TSA?
Yes, the patient should be counseled by the physician and therapist that activities that expose the patient to high-impact events are not recommended because of the potential trauma to the prosthesis. However, sports such as swimming, bowling, dancing, and bicycling should be resumed when appropriate healing has occurred.

16. What is meant by limited-goal rehabilitation? To what type of patient is it applied?
Limited-goal rehabilitation is meant for patients who have deficient rotator cuff and deltoid musculature and significant bone deficiency that does not tolerate the typical rehabilitation program. Patients having long-standing rheumatoid arthritis; rotator cuff arthropathy and some revision arthroplasties may fall into this category. The focuses of limited-goal rehabilitation are pain relief and stability. The shoulder functions primarily at the side with elevation restricted at or below 100 degrees and external rotation of 20 degrees.

17. When should postoperative rehabilitation begin for TSA and hemiarthroplasty?
Ideally rehabilitation begins preoperatively. Education about postoperative exercise regimens and typical postoperative symptoms alleviates the patient’s apprehension. Early passive motion should be initiated on day 1 or 2 after surgery to prevent intraarticular adhesions and soft tissue contractures. However, the surgeon may modify this procedure, depending on bony or soft tissue quality and fixation during surgery. The most common reasons for TSA and hemiarthroplasty revision surgery are contracture between the deltoid and rotator cuff due to prolonged immobilization.

18. Describe the technique of early passive motion (EPM).
EPM, as described by Neer, begins on the second day postoperatively. The patient takes an appropriate pain medication 45 minutes before EPM and applies dry or moist heat to relax the muscles. The patient performs pendulum exercises forward, backward, and in circles with the muscle relaxed like a rag doll. The patient sits or lies in a recumbent position while the surgeon or therapist slowly elevates the relaxed arm in the scapular plane, applying slight traction. Observation of patient’s face and constant communication with the patient are mandatory to assess pain during the exercise.
Patients are reminded frequently to relax as the arm is elevated to maximal levels. This maneuver is repeated 3 to 5 times twice daily. The point of maximal elevation, based on the surgical procedure, should be determined by the surgeon and communicated to the therapist. Typically, passive external rotation is also started with the arm at the side. Because of the recent changes in health care, exercises often are started on day 1 postoperatively and must be taught to a family member because of early discharge.

19. Is all passive elevation the same?
No. Passive elevation in the supine position produces less electromyographic (EMG) activity in shoulder musculature than passive elevation in the upright position. Minimal EMG activity has been recorded in the supraspinatus, infraspinatus, and anterior deltoid during supine self-assisted and helper-assisted elevation. However, more EMG activity is noted in the supraspinatus, infraspinatus, and anterior deltoid during passive elevation in an upright position using a pulley or a stick.

20. What is the Neer-phased rehabilitation program?
Charles Neer popularized three phases of shoulder rehabilitation for TSA, hemiarthroplasty, and rotator cuff repairs. **Phase I** consists primarily of passive motion exercises, including passive movement of the involved arm by a therapist or family member. Phase I also incorporates the use of assist devices such as rope and pulley, stick, or tabletop to aid the patient in performing passive and active assisted exercises independently.

**Phase II** consists primarily of active motion exercises. The patient progresses from active assisted to active exercises without assistive devices. The treating clinician must respect healing time frames and incorporate creative techniques to regain coordinated active range of motion.

**Phase III** consists of resistive exercises. Use of resistive devices, such as light weights and rubber tubing, is incorporated to regain shoulder strength.

21. Why do some patients need abduction pillows and others do not?
The surgical repair and status of the rotator cuff musculature dictate the necessity of an abduction pillow or splint postoperatively. The surgeon examines the quality of the soft tissues during the operation and at closure decides whether excessive tension is placed on the rotator cuff tendons with the arm at the side. Patients with undue tension with the arm at the side or poor tissue may be placed in an abduction splint to reduce stress on the compromised structures and allow for healing.

22. What are the standard precautions after TSA and reverse TSA?
Each surgery is different, and communication with the surgeon is critical. Events during surgery must be communicated to the therapist to ensure postoperative rehabilitation that enhances rather than damages the repair. However, some standard precautions are recommended. Self-transfers and ambulation with crutches should be avoided until adequate strength is regained (often about 6 months). If the patient is suffering from osteoarthritis, therapists are urged to avoid cardinal plane flexion activities because posterior glenoid wear is common and may predispose the patient to posterior subluxation. Patients undergoing TSA because of arthritis from previous dislocations may have weak deltoid and/or unstable joints, which may delay the resistive exercise phase. Patients with rheumatoid arthritis often have weak or torn rotator cuff tissues and proceed slowly through rehabilitation; they need frequent verbal reinforcement. Patients undergoing TSA because of rotator cuff tear arthropathy, congenital defects, neoplasm, and Erb’s palsy deformity most commonly fall into the limited-goal rehabilitation program.

Reverse TSA carries specific precautions. Most important, the reverse TSA is at great risk for dislocation; therefore, the patient must be made aware of positions of potential dislocation to avoid overstretching the anterior tissue of the subscapularis and anterior capsule. Instruct the patient to avoid internal rotation or hyperextension of the involved shoulder. The patient should not use the involved arm to assist in transfers from bed or chair.

23. What are typical outcomes for reverse total shoulder prosthesis?
Pain improves although it may not be completely eliminated because several studies have shown pain to decrease by 3 to 8 points. Active forward arm elevation usually improves to between 100 and 150 degrees. Function improves by a range between 30% to 50% on the American Shoulder and Elbow Society Functional Score (ASES) and Simple Shoulder Test (SST) scores. Complication rates are quite variable from 7% to 75% because of surgeon inexperience, prosthetic design, surgical technique, and case complexity.
BIBLIOGRAPHY


CHAPTER 41 QUESTIONS

1. Tearing of the rotator cuff accounts for approximately _______ % of total shoulder complications.
   a. 32
   b. 42
   c. 66
   d. 50

2. Complications following reverse total shoulder arthroplasty range from 7% to 75%. Which of the following helps explain this large variation?
   a. Surgeon inexperience
   b. Prosthetic design
   c. Case complexity
   d. All of the above

3. Phase II of the Neer-phased rehabilitation program consists of which of the following?
   a. Primarily active range of motion exercises
   b. Primarily passive range of motion exercises
   c. Primarily resistive exercises
   d. Primarily active assistive range of motion exercises
ACROMIOCLAVICULAR AND STERNOCLAVICULAR INJURIES
T.R. Malone, PT, EdD, ATC, FAPTA, and A.L. Pfeifle, EdD, PT, FNAP

ACROMIOCLAVICULAR INJURIES

1. What are the typical mechanisms of acromioclavicular (AC) injury?
The most common mechanism of AC injury is direct force to the tip of the shoulder with the arm adducted against the body. As a result, the acromion is driven downward or inferiorly, with resultant ligament disruption. The location and number of ligaments affected are directly related to the level of force; both AC and coracoclavicular (CC) ligament complexes are at risk for injury.

A secondary mechanism of AC injury is indirect force, as when a person falls on an outstretched hand, generating an impact load at the acromion through the humeral head. This injury typically involves only the AC capsule and ligaments. It should be noted that 18% of the time, following an AC joint injury, that other shoulder injuries occur (SLAP, 14%; RCT, 4%).

2. Who is at risk for AC injury?
AC injury most commonly occurs in men rather than women and in relatively young, as opposed to older, people. On average 56% of all AC joint injuries occur during the first 3 decades of life. AC injuries are approximately four or five times more prevalent than sternoclavicular injuries.

3. What is the common name for AC joint injury?
The layman’s term for AC joint injury is shoulder separation.

4. Describe the structure and function of the AC joint.
The AC joint is a diarthrodial plane joint, connecting the outer end of the clavicle with the anterior medial portion of the acromial process. It has fibrocartilage surfaces. The facet (surface) joint shapes include a convex clavicle and a concave acromion. An interesting component of the joint is the intraarticular fibrocartilaginous meniscus-like disc that is interposed between the joint surfaces. This disc typically begins to degenerate during the third and fourth decades of life. The AC joint, in concert with the sternoclavicular joint, allows the clavicle to serve as a crankshaft, keeping the arm in a functional position in relation to the body. The clavicle rotates in early and late phases during abduction and elevation of the humerus.

5. What are the ligaments of the AC joint?
The three major supporting ligaments of the AC joint are the conoid, trapezoid, and acromioclavicular ligaments. The conoid and trapezoid ligaments are collectively referred to as the coracoclavicular ligament.

The (superior and inferior) AC ligaments reinforce the joint capsule. Their primary role is to control horizontal movements of the clavicle. The superior portion is reinforced by the insertional fibers of the deltoid and trapezius muscles. Vertical stability of the clavicle (AC joint) is controlled by the coracoclavicular ligaments (conoid and trapezoid). The conoid lies medial to the joint, runs posteriorly, and is triangular in shape, whereas the trapezoid is positioned laterally, in the sagittal plane, and is quadrilateral in shape. The orientation of the coracoclavicular ligaments is critical to controlling the rotation of the clavicle, enabling full elevation of the arm. This dual pattern of muscle fiber orientation is particularly significant in that it is also the feature that makes a “simple surgical procedure” unlikely and unsuccessful.

6. Describe the acute presentation of a patient with an AC injury.
The patient with an AC injury often cradles the involved arm by grasping and supporting the elbow with the uninvolved hand and complains of pain over the AC joint. This position reduces the gravitational pull of the weight of the arm inferiorly and also provides some stabilization of the arm next to the trunk. It should be noted that grade IV AC joint injuries with posterior displacement of the distal clavicle and concomitant anterior SC dislocation can cause extreme pain over both the AC and SC joints. Patients can also complain of neck pain with type V and VI injuries because of soft tissue injury of the trapezius.
7. What radiographs are taken to diagnose/classify AC injuries?
Traditionally some patients received x-ray exams in both loaded (weighted) and unloaded patterns to determine the level of clavicle displacement. The key to this technique is that the weight must be freely suspended from the arm, using no muscular action to hold it in place. A second key to obtaining appropriate diagnostic information via radiograph views of the AC joint is to decrease the intensity of exposure because overexposure of the joint typically occurs with normal intensities. The use of weighted films continues to decline, with MRI becoming the modality of choice to document level of tissue disruption, as part of the classification process. Some special radiographic angles have been used to delineate the AC joint space more accurately. The usual (normal) anteroposterior view superimposes the joint space onto the spine of the scapula. To correct for this, Zanca recommends a 10- to 15-degree superior angulation view. The visualization of the coracoid is best provided by a supine notch view. Other possible modifications include a scapulolateral view.

8. How are AC injuries classified?
Because AC injury may include two ligament complexes, the classification scheme is somewhat complex. Rather than the simpler first-, second-, and third-degree pattern typically used with specific ligamentous implications, the AC scheme incorporates modifications reflecting horizontal and vertical motions. It also adds descriptors of the rare extreme vertical displacement injuries, classified as types IV to VI. Additional minor variations of these categories have been introduced and are under review for applicability.

<table>
<thead>
<tr>
<th>Type</th>
<th>Clinical Findings</th>
<th>Instability</th>
<th>Radiographs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I: sprain of AC ligaments; AC and CC ligaments are intact</td>
<td>Mild to moderate pain at AC joint; General movement is pain-free; Tender to palpation</td>
<td>None; minimal ligament damage</td>
<td>Normal</td>
</tr>
<tr>
<td>Type II: complete disruption of AC ligaments; sprain of CC ligaments</td>
<td>Moderate to severe pain at both AC joint and CC interspace; Limited function</td>
<td>Definite horizontal instability; possible slight change in vertical stability</td>
<td>Slight elevation of clavicle</td>
</tr>
<tr>
<td>Type III: complete disruption of AC and CC ligaments</td>
<td>High-riding clavicle; Exquisite pain; Inability to use unaffected extremity; Affected arm often cradled with unaffected extremity</td>
<td>AC (horizontal) and CC (vertical) instability</td>
<td>25% to 100% increase in CC interspace</td>
</tr>
<tr>
<td>Types IV, V, and VI</td>
<td>Severe pain and limited function; Extreme drooping of involved upper extremity</td>
<td>Horizontal and vertical (surgical intervention directed at restoration of ligamentous complexes and muscular insertions)</td>
<td>Severe displacement of CC follows Type IV: superior and posterior displacement of clavicle Type V: superior and posterior displacement of clavicle Type VI: 100% to 300% increase of CC interspace compared with normal</td>
</tr>
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Type VI: clavicle displaced inferior to coracoid (subcoracoid dislocation)

9. How are type I AC injuries treated?
Treatment of type I AC injuries does not require immobilization, but it can be utilized if needed to control pain. Ice is recommended for pain modulation, and the patient may return to activity as comfortably tolerated. If activity exposes the patient to contact or impact forces, a donut pad placed over the shoulder helps protect the joint. This pad is designed to allow distribution of impact around the AC joint rather than onto it; it is an oblong, dense foam base with the center removed (area of the AC) and is covered by thermoplastic material (taped in place). If used during an athletic event, the thermoplastic surface is covered with temper foam to protect others. When used by athletes participating in events where they wear shoulder pads, the clinician should place a corresponding pad on the uninvolved shoulder to ensure proper direct loading to the thorax.

10. Describe the treatment for type II AC injuries.
Patients who have sustained a type II AC injury typically use a sling as needed and apply ice as a pain modulator. Range of motion (ROM) exercises are initiated on an as-tolerated basis, often beginning in a passive form to minimize muscle activation of the trapezius and deltoid groups. An exercise program designed to fit a patient’s needs generally includes functional progression. If the shoulder is exposed to impact forces, the donut pad should be used as the patient returns to function, as outlined in answers 9 & 13. Specific strengthening exercises may be required, depending on patient activities. The anatomic presence and positions of the deltoid and trapezius fibers act as reinforcement to the AC joint capsule and thus are often part of the considerations for the long-term rehabilitation program. Usually the athlete can return to full function within 2 to 3 weeks after injury. Pain in the AC joint may persist for up to 6 months after grade I and II injuries. Thirty-three percent of patients sustaining grade I and II AC joint injuries may have continued reports of pain and instability for up to 6 years post injury.

11. Describe the operative and nonoperative approaches to type III AC injuries.
The most appropriate treatment for type III AC injuries is somewhat controversial.

**Operative Management**
Surgical techniques have been used to address the instability created by the disrupted ligaments. Surgeons generally attempt to pull or stabilize the clavicle downward, often to the coracoid, via a metal screw, Dacron tape, wire, pins, or tissue grafts. Complications of such procedures can include infection, pin breakage, pin/wire migration, and resection of the clavicle or coracoid if the wire cuts through the bone. Even after surgery, residual AC joint deformity or discomfort may occur, complicating rehabilitation and ultimate function. Early postoperative management often includes 4 to 6 weeks of immobilization after surgical intervention and a rehabilitation program thereafter. Functional outcomes following this procedure appear to be quite similar to those obtained through nonsurgical management. Hence current treatment is more often directed toward conservative nonsurgical management, with surgical management used in recalcitrant cases or when initial management does not allow for a return to desired levels of participation.

**Nonoperative Management**
Conservative management is much like that for a second-degree acromioclavicular injury but with a greater reliance on an immobilization and/or support device because of the vertical instability typically associated with a type III AC injury. Also because of associated vertical instability, a residual step deformity remains at the distal clavicle, even after healing is complete. Fortunately, this deformity rarely becomes a disability, and functional outcomes are relatively equal in patients managed with or without surgery. Because disability is most likely a problem in patients who regularly expose the arm to high-intensity demands, surgeons may consider surgical treatment under such conditions. Depending on the presenting circumstances (age, throwing status, expected ongoing demands, etc.), the MD’s decision for recommended treatment still typically favors nonoperative treatment. However, if symptoms do not abate adequately, the surgical choice is more commonly today achieved via soft tissue grafts attempting to better replicate the anatomy of the conoid and trapezoid ligaments.

“In summary, the recommendation for type III uncomplicated AC joint injury is to start with a trial of nonsurgical treatment for 3 to 4 months. If AC joint pain and dysfunction persist following nonsurgical treatment, surgical treatment should be considered.

12. Describe the initial treatment for significant (type III or greater) AC injuries.
Reduction and maintenance for comfort are the rules. Because type IV, V, and VI injuries may be corrected surgically, physician follow-up is important. Although the stated treatment is reduction, in reality the arm is immobilized or supported in a sling, but true reduction is not maintained. Devices have been designed to pull the humerus superiorly and the clavicle inferiorly, but their success is minimal because they are
frequently associated with a lack of patient compliance. The most commonly used device is the Kenny-Howard harness, which incorporates this combination. In reality, the outcomes of treatment with a harness or benign neglect are quite similar.

13. What can be done to minimize or prevent AC injuries?
In sports where tackling is the rule, shoulder pads are frequently worn. If you place a donut pad under one side, it is important also to pad the uninjured side to avoid alteration of shoulder pad alignment. Shoulder pads work via a cantilever design that enables forces to be placed onto the anterior and posterior thorax rather than the underlying area. Pads must be fitted properly and stabilized to the thorax. A good rule is that the proper fit of the shoulder pad is more important than its size or “model.”

14. What are the long-term consequences of AC injury?
Patients often develop a step deformity at the AC joint post injury, where the clavicle appears to sit higher on the affected side than on the normal side. In addition, the patient may experience some pain with high-demand activity. Of interest, significant disability is relatively rare, even with an obvious deformity. Patients may also experience long-term arthritis of the joint but with limited symptoms. In fact, postsurgical patients have similar long-term outcomes to those treated nonoperatively.

15. What can be done for the patient whose pain is associated with weight lifting?
Pain with weight lifting is a common complaint in athletes with a previous AC injury. The wide-grip bench press is the primary culprit for such pain. The anterior fly type maneuver, which replicates the cross-arm adduction test for AC pain and provocation, should also be avoided. Although it might be helpful to do so, athletes usually hesitate to use a narrower grip during weight lifting because it decreases the maximal load that can be performed during bench press. Antiinflammatory medications, local ice application before and after exercise, and exercise modification can be used successfully in select patients. Other patients will not have a successful outcome because of established osteolysis of the distal clavicle and its resultant osteoarthritis.

16. What other athletes are prone to AC problems?
Racquet-using athletes or throwing athletes may develop AC symptoms related to sport activities. They may exhibit symptoms on follow-through (cross-arm motions) as well as during weight training with wide-grip bench press, dips, or cross-arm fly maneuvers. Partial ROM (restricted ranges) during weight training and decreased maximal effort and repetitions of throwing can be helpful in alleviating pain or minimizing AC problems.

17. What is the surgical procedure of choice for arthritic AC disability?
Physicians often excise the distal clavicle of patients with recalcitrant pain and disability of the AC joint. The Mumford procedure is designed to remove approximately 0.5 to 2 cm of the distal clavicle, which prevents impingement with crossed-arm movements. Rehabilitation after the procedure is directed toward pain modulation and support for the first 10 to 14 days, followed by functional progression related to the specific needs of the patient. Today, this is typically an arthroscopic procedure.

18. Discuss briefly the role of AC joint mobilization.
AC mobilization can be successfully used in patients presenting with decreased elevation and limited cross-arm motion (horizontal adduction). Mobilization exercises are usually performed from behind, using the horizontally placed thumb to move the clavicle forward. The therapist should maintain as much contact with the distal clavicle during mobilization as possible to minimize the point of pressure. The arm is supported on a plinth or tabletop as mobilization is performed. Improvements in ROM (especially elevation) may follow this procedure.

**STERNOCLAVICULAR INJURIES**

19. What is the typical mechanism of sternoclavicular (SC) injury?
SC injury is relatively rare but may result from direct trauma, as in an athlete who sustains direct force to the clavicle via impact collision with another player or a hard surface, such as a goalpost or equipment. The more common method of SC injury is indirect, as when someone lying on his or her side receives an external load that causes a rolling of the body over the shoulder, thus combining compression and twisting. Anterior injuries are more common than posterior injuries; posterior dislocation is quite rare but may have serious implications.
20. Who is at risk for SC injuries?
SC injuries are far more common in men than in women and are more prevalent in relatively young, rather than older, people, but they are much less common than AC injuries. Acromioclavicular injuries occur four or five times more frequently than sternoclavicular injuries.

21. Describe the structure and function of the SC joint.
The SC joint, like the AC joint, contains a meniscus-like disc. Because the articulating surfaces of the sternum and clavicle are typically incongruent, the disc becomes the contact surface of the joint. The actual joint surfaces are saddle-shaped, using the disc independently to enable the unique actions of the clavicle in relation to the sternum (ie, the disc works or stays with either the sternum or the clavicle during specific actions). The movements allowed by the joint are elevation and depression, protraction and retraction, and rotation.

22. What ligaments support and control the SC joint?
The SC ligament complex includes the capsule itself, which is directly reinforced by the anterior and posterior SC ligaments. The costoclavicular ligament is quite strong and assists with the pivoting action of the clavicle in relation to the anchored, underlying first rib. The interclavicular ligament supports the superior aspect, reinforcing the position of the clavicle to minimize inferior displacement, which would endanger the underlying brachial plexus and subclavian artery.

23. Which radiographic views are used to assess SC injuries?
Special radiographic views can be used to assess the SC joint. These views minimize superimposed structures. Hobbs recommends that patients be imaged in a sitting position, leaning forward with elbows supported on the x-ray table. In this position a vertical (superior) radiograph is taken. Rockwood uses a “serendipity” view in which the patient is positioned supine with the x-ray tube angled approximately 40 degrees from the vertical and directed toward the clavicle.

24. How are SC injuries classified?

<table>
<thead>
<tr>
<th>Sternoclavicular Injury Classification</th>
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<tr>
<td><strong>TYPE</strong></td>
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<tr>
<td>Mild sprain</td>
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<tr>
<td>Moderate sprain (subluxation)</td>
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<tr>
<td>Severe sprain (dislocation)</td>
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Two subtypes:
1. anterior dislocation
2. posterior dislocation

25. Describe the treatment for a mild sprain of the SC joint.
No instability is present with a mild sprain. Ice can be used for pain modulation; in addition, a sling can be worn for protection from additional trauma for 2 to 4 days or until the patient is pain-free. A gradual return to activities should follow, as tolerated, through a functional progression.

26. Describe the treatment for a moderate sprain (subluxation) of the SC joint.
This type of injury requires immobilization and protection. Most patients wear a clavicle strap to maintain proper clavicular orientation and a sling to support the weight of the arm. Both devices are used for 2 to 4 weeks, followed by rehabilitation progression dictated by need and symptoms.

27. What is the initial treatment for an anterior severe sprain (dislocation) of the SC joint?
The first approach to treating an SC injury is to ensure that reduction is achieved and maintained. The majority of sternoclavicular dislocations occurs anteriorly and can be reduced with firm digital pressure. The decision to immobilize after SC reduction, however, is somewhat controversial. Many SC dislocations are stable after reduction, and patients often do well with brief immobilization and progression of activities, as tolerated. To reduce the dislocation, the patient is positioned supine and a pad is placed posteriorly, allowing shoulder extension. A posterior force applied to the proximal (displaced) clavicle completes the reduction. If used, a sling is typically worn for 3 to 6 weeks after reduction.
28. What is the initial treatment for a posterior severe sprain (dislocation) of the SC joint?  
The rare posterior sternoclavicular dislocation occurs with abrupt and extreme shoulder extension while
the trunk position is maintained, thus permitting a fulcrum/lever sequence. In such cases, reduction may
occur via an open procedure in the operating room, particularly because a closed technique may not be
successful. An open procedure uses forceps to pull the clavicle into the correct position. The patient with a
posterior SC dislocation may present as a medical emergency because of the potential for significant
injury/compression to underlying organs and structures. Typically, a figure-of-eight harness is used for
posterior dislocations after reduction is achieved for a minimum of 4 weeks. Some physicians combine
the clavicle harness with the arm sling. For both anterior and posterior injuries, use of ice is followed by
gentle, controlled movements after immobilization, leading to progressive functional rehabilitation.

29. What are the long-term consequences of SC injuries?  
After reduction of a SC injury, most patients have no significant long-term disability. If chronic joint instability
develops, corrective surgery can be performed, but the results are not uniformly positive. Potential adverse
outcomes after such a procedure include arthritis and pain, particularly in high-demand patients.

30. What types of surgical procedures are performed on patients with SC instability
and disability?  
Although relatively rare, some patients experience recurrent dislocations and demonstrate instability,
leading to chronic disability and pain. Surgical intervention results are inconsistent, and the surgery is
difficult to perform. Most procedures use some type of graft material (subclavius tendon, palmaris longus
muscle, or toe extensor muscle) to redevelop proximal stability of the SC pivot joint. Unfortunately, mixed,
rather than consistent, postsurgical results are typical.

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CHAPTER 42 QUESTIONS
1. Following a grade II AC joint injury, most athletes can return to full function in:
   a. 1 week
   b. 2 to 3 weeks
   c. 4 to 5 weeks
   d. 6 to 8 weeks
2. Which of the following dislocations can be a medical emergency?
   a. Anterior SC joint dislocation
   b. Posterior SC joint dislocation
   c. Grade III AC joint injury
   d. Grade V AC joint injury
3. What is the standard of care for a noncomplicated grade III AC joint injury?
   a. Sling for 6 months
   b. Surgical stabilization
   c. Mumford procedure
   d. Nonoperative management
1. **What is the role of the scapula in glenohumeral movement?**
   The scapula provides a mobile base for humeral motions in all directions; assists in providing an appropriate muscle length-to-tension ratio for rotator cuff and deltoid musculature throughout arm elevation; and serves as a bony attachment for most of the upper quarter proximal musculature. The scapula and surrounding musculature are critical in force transmission from the lower extremities and trunk to the arm in throwing activities.

2. **What is the 3-D kinematics of the scapula with respect to the humerus and trunk in arm elevation?**
   Scapular motion occurs in three cardinal planes during arm elevation: upward rotation, external rotation, and posterior tilt. In a healthy person, as the arm is elevated in the scapular plane, the scapula rotates upwardly $\approx 50^\circ \pm 4.8^\circ$, externally rotates about a vertical axis $\approx 24^\circ \pm 12.8^\circ$, and tilts posteriorly about a horizontal axis $\approx 30^\circ \pm 13.0^\circ$.

3. **What muscular force couples act on the scapula during arm elevation?**
   A force couple is two or more lines of force acting on different points of the same structure to produce rotation. The upper trapezius, lower trapezius, and serratus anterior are involved in scapular upward rotation. The posterior tilting and external rotation of the scapula are thought to result from action of the lower serratus anterior musculature and lower trapezius.

4. **Does the scapular musculature activation pattern change when the glenohumeral joint is injured?**
   Yes. Several different studies have demonstrated that motor activity level or onset of motor activity is altered in patients with impingement or glenohumeral instability. Diminished serratus anterior activity has been documented in throwers with unstable shoulders and swimmers with impingement. Delayed onset of serratus anterior activity in overhead reaching has been demonstrated in swimmers with impingement.

5. **Can abnormal scapular movement be associated with rotator cuff impingement?**
   Yes, previous research suggests that patients with subacromial impingement syndrome will present with impaired scapular posterior tilt, upward rotation, and external rotation.

6. **Define scapular dyskinesia.**
   Scapular dyskinesia describes abnormal or atypical movement of the scapular during normal active motion tasks, such as reaching overhead. Similar terms used in the literature include abnormal scapulohumeral rhythm, scapular winging, and scapular dysrhythmia.

7. **How common is scapular dyskinesia?**
   It is very common. Based on the current literature, it ranges from 75% of the healthy asymptomatic population. In healthy overhead athletes it is typical to see asymmetry, primarily with more anterior tilt and internal rotation on the dominant side compared with the nondominant side. Warner found 64% of patients diagnosed with an unstable glenohumeral joint present with some form of scapula dyskinesia, while all patients with impingement demonstrated some degree of scapular dyskinesia.

8. **What populations need to be watched for scapula pathology?**
   Scapular pathology should be suspected of any age overhead athlete or patient who presents with pain in the shoulder region. Current research suggests little league baseball players as young as 10 years old present with increased upward scapular rotation compared with nonathletic children of the same age.
9. What causes scapular dyskinesis?
It is not clear whether the scapula dyskinesis is primary or secondary to shoulder pathology. The general consensus is that deficiency of the scapular musculature, particularly the serratus anterior and trapezius, is often involved. The deficiency may be simple weakness, tightness, or a compensatory motor pattern developed in response to pain. Congenital deformities, such as scoliosis or Sprengel’s deformity, may cause scapular dyskinesis.

10. What is Sprengel’s deformity?
Also called Eulenburg’s deformity, Sprengel’s deformity is failure of the scapula to descend during normal development. Typically, it is seen in infancy or early childhood as a prominent lump in the web of the neck. The scapula often is hypoplastic, abnormally shaped, and malrotated so that the superomedial angle is curved anteriorly into the supraclavicular region and the inferior angle abuts the thoracic spine. Arm abduction may be limited. Associated musculoskeletal deformities, such as scoliosis, rib abnormalities, Klippel-Feil syndrome, and spina bifida are common.

11. What is SICK scapula syndrome?
SICK scapula stands for scapular malposition, inferior medial border prominence, coracoid pain, and dyskinesis. It is a severe form of scapular dyskinesis associated with overuse syndrome and fatigue. The SICK scapula is commonly found in overhead athletes and can be noted by a unilateral drop in the affected shoulder.

12. How do you assess abnormal scapular movement?
Abnormal scapular movement or scapular dyskinesis can be observed statistically, but recent research suggests the use of dynamic activities to determine whether abnormal scapular movement exists. Several observational clinical methods exist, with the most recent incorporating the two-type scapular observational method of “yes,” scapular dyskinesis is observed, or “no,” scapular dyskinesis is not observed. Interestingly, a second group of investigators came up with a similar approach but used the terms “obvious abnormality” and “no abnormality” to represent the presence or absence of scapular dyskinesis. These approaches were found to have moderate to good (kappa = 0.4 to 0.6) reliability and to be less restrictive than a previous four-category approach. The overall concept in both techniques is to watch the patient elevate the arm through a full range of motion in either flexion, scapular plane abduction, or pure abduction multiple times. McClure used hand weights during shoulder flexion and frontal-plane abduction to help load the scapular muscles to enhance the observational analysis. Either one of these methods may be used to assess dyskinesis. The use of multiple repetitions or weight suggests that, to observe scapular dyskinesis, the musculature needs to be challenged.

13. How do you treat scapular dyskinesis?
The first step is to do a complete neuromuscular examination of the shoulder girdle and cervical region. Based on your findings, tight structures need to be stretched and weak structures need to be strengthened. Strengthen the scapular protractors with resistance exercises that emphasize scapular protraction, and activate the serratus anterior without overactivating the upper trapezius. One of the most important treatments is education about proper posture and typical movement of the scapula. Biofeedback techniques, such as mirrors, verbal cueing, tactile cueing, and video monitoring during exercises, help the patient visualize the trunk and scapula. The patient benefits by observing the trunk and scapula during exercises to learn how to voluntarily control scapular musculature.

14. Which scapular muscles should be targeted for rehabilitation?
There are approximately 20 muscles attached to the scapula; however, those most involved in stabilization of the scapula against the thoracic wall are the rhomboids, major and minor; upper, middle, and lower trapezius muscles; the rotator cuff musculature; and the serratus anterior. According to Cools, side-lying external rotation, side-lying forward flexion, prone horizontal abduction with external rotation, and prone extension stimulate lower and middle trapezius muscle activity while diminishing upper trapezius activity. Although a scapular stabilizer, excessive activation of the upper trapezius is a contributing factor to abnormal scapular motion. Current literature suggests that during prone horizontal abduction at 90 degrees with full external rotation, the middle and lower trapezius are working between 55% to 75% of maximal muscle activity while lifting 3% of the individuals’ body weight. Side-lying external rotation tends to bias the lower trapezius to be more active than the middle trapezius because the middle trapezius is only working at approximately 20% while the lower trapezius is working at 55% while lifting the same resistance as the prone horizontal abduction exercise.
The serratus anterior is best activated during the plus phase of exercises compared with those exercises without a plus phase. The lower fibers of the serratus anterior are best activated with exercises that require the arm to be elevated to at least 120 degrees. Strengthening and stabilization of these muscles will help reestablish neuromuscular pathways and aid in prevention of instability and secondary impingement, labral pathology, and certain overuse pathologies by maintaining glenohumeral joint congruency.

15. Which exercises target the scapula muscles?
Current electromyographic (EMG) research on scapular rehabilitation suggests the following exercises most effectively target the scapular stabilizers. The push-up plus elicits 80% maximum voluntary isometric contraction (MVIC) of serratus anterior activity, prone flexion over head for lower trap elicits 95%, and rows elicit 112% MVIC for upper trap and middle trap muscles. In addition, forces generated from the lower extremity during throwing are transferred through the scapula to achieve increased power, and thus the scapula is considered an integral part of the kinetic chain. All scapular rehabilitation should include a strong lower extremity and core strengthening program.

16. How does dyskinesis differ from scapular winging?
Scapular winging typically is associated with thoracic nerve palsy. Scapular winging is noted when the patient leans into a wall, supporting his or her weight with the arms, or when resistance is applied to outstretched arms as the patient attempts to forward flex. The entire medial and inferior border of the scapula lifts off the thoracic wall because of serratus anterior deficiency.

17. What are the peripheral nerves and their corresponding root levels that innervate the muscles that attach to the scapula?
Biceps: long and short head (musculocutaneous); C5, C6
Triceps: radial; C6–C8, T1
Supraspinatus: suprascapular; C4–C6
Deltoid, all components: axillary; C5, C6
Teres major: lower subscapular; C5–C7
Pectoralis minor: medial pectoral with communicating branch of lateral pectoral; C6–C8, T1
Latissimus dorsi: thoracodorsal; C6–C8
Subscapularis: upper and lower subscapular; C5–C7
Infraspinatus: suprascapular; C4–C6
Teres minor: axillary; C5, C6
Rhomboids, major and minor: dorsal scapular; C4, C5
Lever scapulae: dorsal scapular; C4, C5, and C3 and 4
Trapezius, all portions: spinal accessory (cranial nerve XI) and ventral ramus; C2–C4
Serratus anterior: long thoracic; C5–C8

18. What causes long thoracic nerve palsy?
Long thoracic nerve palsy typically presents idiopathically without a history of a macrotrauma. Several mechanisms have been described, such as surgical complications, viral illnesses, immunizations, and trauma (often a traction mechanism).

19. What is the standard treatment for long thoracic nerve palsy?
The palsy usually resolves gradually over time. An electromyographic (EMG) study confirms the diagnosis and can be used to track progress. Strengthening exercises for the weak serratus anterior should be delayed until EMG indicates regeneration. The patient should restrict heavy pushing and overhead lifting activities. Some patients have benefited from a shoulder orthotic that keeps the scapula pressed against the thoracic wall to relieve pain. In 1996 a study conducted on long-term outcomes (6 years; range 2–11 years) of iatrogenic long thoracic palsy reported residual symptoms in 25 of 26 patients. Eighty-one percent could not lift or pull heavy objects, 54% could not work with hands above shoulder level, and 58% could not participate in sports such as tennis and golf. Recently several case studies have been written on nerve transfers as treatment options for long thoracic nerve palsy.

20. What are alternative treatments for long thoracic nerve palsy?
Muscle transfer of the pectoralis major is a procedure that has shown (at a 2-year follow-up) improvement in function (58%) and pain reduction (50%) with a 40-degree increase in active arm elevation. Surgical release of the scalenes to relieve compression on the long thoracic nerve has been reported with good
success. Case reports of nerve transfer of the thoracodorsal for the long thoracic have been reported. Outcomes demonstrated full shoulder flexion with no scapular winging and reported no functional limitations within his daily activities of living (at a 16-year follow-up). A case report describing a two-level motor nerve transfer of the pectoral fascicle of the middle trunk and part of the thoracodorsal nerve was conducted to reinnervate the serratus anterior muscle. Post surgery, the patient’s pain diminished from a 4 out of 10 to a 1 out of 10, and after 13 months of physical therapy, the patient was able to return to all normal activities, with mild difficulty with overhead activities.

21. What is scapulothoracic dissociation?
Scapulothoracic dissociation results from severe trauma involving lateral displacement of the scapula. It has been described as closed, traumatic forequarter amputation. This injury typically is associated with motorcycle, motor vehicle, or farm implement accidents. The lateral displacement of the scapula ruptures surrounding soft tissue. Typical associated injuries are clavicle fracture, significant neurovascular damage, and major trauma.

22. A patient presents with severe shoulder and neck pain and a drooped shoulder after cervical lymph node resection. What do you suspect is the cause?
One complication of a lymph node or benign tumor removal is iatrogenic injury to spinal accessory nerve. The injury typically involves the trapezius but often spares the sternocleidomastoid muscle. Trapezius weakness is often noted with the inability to lift the arm above horizontal, and the involved side presents with drooped posture. Patients describe significant shoulder pain, with a sensation of heaviness or the feeling that the shoulder is being pulled out of the socket on the involved side.

23. Define snapping scapula.
Snapping scapula is attributed to friction between the mobile scapula and its attached soft tissues and the relatively stable thoracic wall. The noise or grating sound may be audible or sensed by the patient. The incidence of grating in the general population has been reported to be as high as 70%. A general friction sound is typically nonpathologic. Grating, loud snap, or pop sounds associated with pain are thought to be pathologic. Anatomic explanations for snapping scapula include thickened bursa, bone spurs on scapula or a rib, Luschka’s tubercle (an exostosis at the superomedial angle of the scapula), and osteochondroma (a common scapular tumor). A tangential scapulolateral view or computed tomographic scan is more helpful in identifying anatomic anomalies associated with snapping scapula than standard anteroposterior scapular radiographs.

24. What is the differential diagnosis of snapping scapula?
Pain may be referred from the glenohumeral joint, cervical nerve root compression, or cervical joint disease. Thoracic disc disease should be ruled out, along with thoracic outlet syndrome. Tumors also must be considered and evaluated with appropriate imaging studies.

25. How is snapping scapula treated?
Conservative management with antiinflammatory medication, physical therapy modalities, and exercise to strengthen the lower trapezius and serratus anterior musculature often are prescribed. Supportive strapping or bracing may be beneficial. Injection of marcaine may be helpful. Surgical treatment is rare and should be considered only if diagnostic imaging demonstrates the presence of an exostosis or a space-occupying lesion in the scapulothoracic space.

BIBLIOGRAPHY
CHAPTER 43 QUESTIONS

1. The push-up plus elicits __________ % maximum voluntary contraction (MVC) of serratus anterior activity, while the prone flexion overhead elicits __________ % MVC of lower trapezius activity.
   a. 90, 80
   b. 90, 115
c. 80, 95
d. 70, 95
2. Which of the following is an exercise that best elicits muscle activity within the lower trapezius?
   a. Push-up plus
   b. Prone horizontal abduction at 90 degrees with full external rotation
   c. Prone shoulder extension
   d. Prone 120-degree abduction with palms facing floor

3. Which of the following are causes of long thoracic nerve palsy?
   a. Viral illnesses
   b. Immunizations
   c. Surgical complications
   d. All of the above
1. How are clavicle fractures classified?
Clavicle fractures are classified according to their location. Middle-third fractures are the most prevalent, occurring in about 80% of clavicle fractures. Proximal-third and distal-third fractures occur in 5% and 15%, respectively, of clavicle fractures.

2. Describe the subclass of distal-third clavicle fractures.
Distal-third fractures of the clavicle are subclassified into three groups: type I, type II, and type III. Type I fractures are nondisplaced because the coracoclavicular ligaments remain attached to the medial fragment. Type II fractures result in detachment of the coracoclavicular ligaments from the medial fragment and displacement of the fracture. Type III fractures involve the articular surface of the acromioclavicular joint without detachment of the coracoclavicular ligaments. Type III fractures lead to joint degeneration rather than displacement.

3. What nerve is most frequently injured with a fracture of the clavicle?
The most frequently injured nerve is the ulnar nerve because it passes between the first rib and the fractured clavicle.

4. How are middle-third clavicle fractures usually treated?
Closed treatment is used for middle-third clavicle fractures without displacement and shortening. The most common methods of closed immobilization include casting, sling and swathe, or figure-of-eight dressings. No closed method can maintain a reduction; therefore, a sling and swathe is most commonly used to maintain patient comfort while the fracture heals.

5. What are the indications for operative treatment of clavicle fractures?
- Open fractures
- 100% displacement with shortening of >15 mm (midshaft fractures)
- Nerve or vascular injury requiring repair
- Displaced type II distal clavicle fractures
- Multitrauma victim where upper extremity assist for weight-bearing is needed.

6. What are the risks of fracture nonunion for displaced midshaft clavicle fractures treated conservatively?
Although the healing rate of nondisplaced fractures of the clavicle is excellent, the rate of union for displaced and shortened midshaft clavicle fractures is between 10% to 15%. Therefore, most of these fractures are treated surgically.

7. What are the results of operative treatment of displaced midshaft clavicle fractures?
Randomized trials of surgical and conservative treatment of displaced midshaft clavicle fractures show a lower risk of nonunion and better functional outcomes with surgical treatment. Healing can be successfully obtained with plating and intramedullary pinning.

8. When should shoulder motion be initiated in closed treatment of clavicle fractures?
Most middle-third clavicle fractures require 6 weeks for union to occur. Gentle shoulder motion is initiated once early stability is noted, around 2 to 3 weeks. The elbow, wrist, and forearm should be used immediately after the fracture to prevent atrophy and stiffness.
9. What is the incidence of proximal humerus fractures?
Proximal humerus fractures are common injuries, representing 4% to 5% of all extremity fractures. The majority of proximal humerus fractures occurs in patients over 65 to 70 years of age. Several studies have shown that the incidence of proximal humerus fractures is increasing. The explanation for the rising incidence is believed to be related to the high prevalence of osteoporosis in the aging population.

10. Describe the Neer classification of proximal humerus fractures.
Fractures of the proximal humerus can occur in several patterns. Fractures typically propagate through the greater tuberosity, lesser tuberosity, surgical neck, and/or anatomic neck. Fractures are classified according to the number of displaced fracture fragments and dislocation of the humeral head. The number of fragments can vary from one to four. Fracture fragments are considered to be present only when displaced 1 cm or more or angulated a minimum of 45 degrees.

11. What are the deforming muscular forces responsible for the pattern of fracture displacement encountered with proximal humerus fractures?
The greater tuberosity is displaced in a posterior and superior direction from the pull of the supraspinatus and infraspinatus muscles. Displaced greater tuberosity fractures also create a tear in the rotator cuff in the region of the rotator interval. The subscapularis will pull displaced lesser tuberosity fractures medially. The humerus displaces medially and internally rotates from the pull of the pectoralis major with displaced surgical neck fractures. In addition, varus angulation and shortening are common because of the pull of the deltoid muscle on the humeral shaft. Often the humeral head will abduct because of the unopposed force of the rotator cuff, further contributing to the varus alignment of the humeral head. The biceps tendon frequently will become trapped within the fracture fragments with displaced surgical neck fractures.

12. How often do nerve injuries accompany proximal humerus fractures?
Nerve injury after proximal humerus fractures is common, especially with displaced fractures. Clinically detectable nerve injuries after proximal humerus fractures have been reported in up to 45% of cases. The axillary and suprascapular nerves are most commonly involved. Often these injuries are incomplete and may manifest as temporary weakness that recovers along the same course of time as fracture healing. One author reported a 6.1% incidence of brachial plexus injuries after displaced proximal humerus fractures. Electromyography (EMG) studies have revealed a very high incidence of occult nerve injury after both nondisplaced (52%) and displaced (82%) fractures. The risk of neurovascular injury after fractures of the proximal humerus is greater in high-energy injuries, fracture dislocations, and penetrating trauma.

13. What percentage of proximal humerus fractures can be treated nonoperatively?
The majority of proximal humerus fractures are minimally displaced (considered one-part fractures) and can be treated successfully with conservative measures. Approximately 80% of proximal humerus fractures meet the criteria for conservative treatment. Fractures of the surgical neck are often accompanied by a moderate degree of displacement and angulation. The high degree of mobility available at the glenohumeral joint, angulation of 30 to 45 degrees and translation of up to 75% of the width of the humeral shaft can be tolerated as long as there is good apposition of the fracture fragments.

14. When is the treatment of conservatively managed proximal humerus fractures initiated?
Numerous authors have noted that early range of motion is critical for successful outcomes after proximal humerus fractures. The shoulder is immobilized in a sling or cuff and collar for comfort and to facilitate fracture reduction afforded by the weight of the arm. Elbow, wrist, and hand range of motion exercises are initiated immediately. Pendulum exercises are initiated as soon as tolerable for most stable one-part fractures. The shoulder is evaluated clinically at 1-week intervals to assess for clinical signs of early union. Once the humerus and the proximal fragments move as one unit with gentle shoulder range of motion exercises, formal range of motion exercises are initiated. Early fracture stability is usually noted at 2 to 3 weeks from the time of injury.

15. What are the outcomes of conservatively treated proximal humerus fractures?
The outcomes of conservatively treated proximal humerus fractures are generally good. Fracture union rates are greater than 90% in those patients with minimally displaced fractures. Early range of motion of the shoulder has been shown to improve the functional outcome after proximal humerus fractures. Most patients will regain normal function of the shoulder after these injuries and experience minimal residual pain. A patient can expect 130- to 150-degree elevation, near symmetric external rotation range of motion, and only mild weakness and functional limitations compared with the opposite shoulder.
16. What are the indications for surgical management of proximal humerus fractures?
The decision to operatively stabilize proximal humerus fractures is related to the severity of the fracture
displacement, the age of the patient, bone quality, and coexisting humeral head dislocations. For two-part
fractures, there is some debate about the degree of deformity that is tolerable at the surgical neck.
Fracture angulation at the surgical neck greater than 45 degrees or translation of the humeral shaft leading to
minimal contact of the bony fragments is best treated surgically. Displacement of the greater tuberosity
is not tolerated well because of the risk of fragment impingement within the subacromial space. Therefore,
greater tuberosity displacement greater than 5 mm is another indication for surgery. Most three- and
four-part fractures require surgery except in low-demand elderly patients. Anatomic neck and head
split fractures generally require operative treatment in the form of hemiarthroplasty. Fractures associated
with humeral head dislocations require open reduction, followed by fracture stabilization or hemiarthroplasty.

17. When is proximal humeral replacement (hemiarthroplasty) preferred over fracture
fixation for the management of proximal humerus fractures?
The decision to replace the proximal humerus to provide fracture stabilization is based on several factors
including the risk of avascular necrosis of the humeral head, the quality of bone, and the age and
functional demands of the patient. Displaced four-part fractures, head split fractures, anatomic neck
fractures, and three- and four-part fracture dislocations are best treated with hemiarthroplasty. Displaced
three-part fractures in older patients or those with poor bone quality is another indication for
hemiarthroplasty.

18. What are the final outcomes of surgical fixation of proximal humerus fractures?
The outcomes of surgically managed proximal humerus fractures are good with proper indications and
appropriate rehabilitation. Factors related to outcome include the type of fracture, the preoperative shoulder
function, bone quality, and the development of a complication related to the injury or surgery itself. Both
percutaneous pinning and open reduction and internal fixation provide reliable results as long as bony
union occurs and avascular necrosis of the humeral head does not develop. Most patients will regain
functional range of motion of the shoulder. Most will attain active shoulder elevation to 120 to 150 degrees,
external rotation to 30 to 45 degrees, and internal rotation to the lumbar spine. Studies using validated
outcome scales note mild residual functional problems such as occasional pain, slight weakness, and
limited function after two- and three-part fractures of the proximal humerus. These studies also note
poor function or a significant complication, such as avascular necrosis, in 10% to 40% of patients with
displaced three- and four-part fractures. Full recovery can take 6 months post surgery.

19. What are the potential complications of surgical fixation of proximal humerus
fractures?
Complications are relatively common after fixation of proximal humerus fractures, ranging from 15% to
30% in most series. The rates of complications are more common with three- and four-part fractures and
in fractures with poor bone quality. The use of fixed angle locking plates has decreased the risk of loss of
fracture reduction, particularly when poor bone quality is present. The most frequent complications
remain: loss of fracture reduction, avascular necrosis of the humeral head, glenohumeral joint screw
penetration, poor return of function, and need for hardware removal.

20. What are the outcomes of hemiarthroplasty for the treatment of proximal humerus
fractures?
Most studies of displaced proximal humerus fractures report inferior results from hemiarthroplasty
compared with fracture fixation. However, hemiarthroplasty remains the treatment of choice in
many complex fracture patterns. Complications such as malunion and nonunion of the tuberosity
fragments and humeral component malposition are thought to be related to poor outcomes. Most
patients experience reliable pain relief after hemiarthroplasty for proximal humerus fractures; however,
functional outcomes can be quite variable. The majority of patients can use the involved extremity
well for activities of daily living below shoulder height, but overhead function is variable. A good outcome
after hemiarthroplasty for humerus fracture is elevation to 110 to 130 degrees, external rotation to
30 degrees, minimal to no pain, and only a modest functional limit. These goals are generally achieved in
between 60% and 70% of patients. Full recovery may take up to 12 months post surgery.

21. Is there a role for reverse shoulder arthroplasty in patients with displaced proximal
humerus fractures?
Given the inconsistent clinical results with hemiarthroplasty for proximal humerus fracture, reverse total
shoulder arthroplasty is more popular for fractures not amenable to repair. The indications for reverse total
shoulder arthroplasty are the same as hemiarthroplasty as long as the patient is 65 years of age or older. Reverse shoulder arthroplasty provides a better and more consistent return of shoulder function compared with hemiarthroplasty. Reverse shoulder arthroplasty requires an intact deltoid for function; therefore, careful assessment of the axillary nerve should be performed before surgery.

22. How often do nerve injuries accompany humeral shaft fractures?
Radial nerve injuries complicate between 6% and 18% of humeral shaft fractures. Other nerve injuries are rare, although brachial plexus injuries have been reported in higher energy trauma. Risk factors for radial nerve injuries include high-energy fractures, open fractures, and distal-third humeral shaft fractures. Over 90% of radial nerve injuries will recover spontaneously over a 4-month period.

23. What is the usual treatment for fracture of the humeral shaft?
The majority of humeral fractures can be treated conservatively. Most fractures are treated for a short period of time in a plaster or fiberglass coaptation splint. Within 1 to 2 weeks, the arm is placed in a prefabricated functional brace. Functional braces facilitate reduction of the fracture through soft tissue compression.

24. What are the outcomes of conservative management of humeral shaft fractures?
Most patients with humeral shaft fractures do well with conservative measures. Conservative treatment generally shows fracture union rates greater than 90% to 95% for those injuries that meet the criteria for surgical management. Fracture union is usually obtained between 8 and 12 weeks. Between 80% and 98% of patients will obtain full range of motion and function of the shoulder and elbow joints.

25. What are the indications for surgical management of humeral shaft fractures?
There are several well-recognized indications for surgical management of humeral shaft fractures. These include pathologic fractures, associated brachial plexus injuries, associated forearm fractures (floating elbow), open fractures with high-grade soft tissue injuries, vascular repair, bilateral humerus fractures, multiple trauma, and inability to maintain adequate alignment (varus angulation of 20 degrees, sagittal plane angulation of 30 degrees, or shortening of 3 cm).

26. What is the recommended treatment for radial nerve palsies associated with humeral shaft fractures?
Radial nerve palsy occurs in 6% to 18% of humeral shaft fractures. The majority of radial nerve palsies represents neurapraxic injuries and will improve with observation alone (>90%). Splinting and range of motion exercises of the hand are encouraged to prevent contracture formation. Electromyography and nerve conduction tests are performed after 3 months if failure of improvement of the palsy is noted clinically. Exploration and neurolysis or repair of the nerve is performed if no signs of recovery are seen after 3 to 4 months. Indications for acute nerve exploration include penetrating open fractures, high-grade soft tissue injuries, or secondary nerve palsies (in some cases).

27. What are the outcomes of surgical management of humeral shaft fractures?
Both open reduction and internal fixation (ORIF) and intramedullary fixation (IMF) produce reliable clinical results. The rate of fracture union after ORIF generally ranges from 94% to 98%, whereas union rates after IMF range from 87% to 94%. Both types of surgeries are associated with low but significant rates of complications. Residual elbow pain is more common after ORIF. Shoulder pain and the need for repeat surgery are more common after IMF. Direct comparisons between the two surgical techniques show trends toward slightly better outcomes after ORIF.

BIBLIOGRAPHY


**CHAPTER 44 QUESTIONS**

1. Operative treatment of displaced midshaft clavicle fractures results in:
   a. More pain and disability
   b. Poorer return of function
   c. A greater chance of bony healing
   d. A higher risk of shoulder stiffness
   e. All of the above

2. A four-part displaced proximal humerus fracture in a 70-year-old, active, healthy female with osteoporosis should be treated with:
   a. Sling immobilization for 3 weeks, then therapy
   b. Open reduction and internal fixation with a plate
   c. Hemiarthroplasty
   d. Reverse shoulder arthroplasty
   e. Sling and bone stimulator

3. Which of the following is true regarding minimally displaced proximal humerus fractures?
   a. They are more common in the elderly.
   b. They have a relatively high rate of occult nerve injury.
   c. They can be treated nonsurgically in most cases.
   d. They typically result in only mild residual loss of shoulder function.
   e. All of the above
1. How is the spinal accessory nerve usually injured?
   The spinal accessory nerve (cranial nerve XI) is a purely motor nerve and supplies motor fibers to the upper, middle, and lower trapezius muscles as well as the sternocleidomastoid. Mechanisms of injury include tumor, surgical procedures to the posterior triangle, and stretch and whiplash injuries. The most common mechanism of injury to the accessory nerve is iatrogenic during lymph node biopsy in the posterior triangle of the neck.

2. Describe the typical presentation of a patient with a spinal accessory nerve injury.
   The patient's symptoms may include a drooping shoulder girdle and/or flat upper trapezius muscle on the involved side. Shoulder pain is a major disabling factor, often attributed to traction at the brachial plexus. Winging of the scapula caused by trapezius weakness increases with abduction, whereas winging caused by serratus anterior weakness increases with forward elevation. If the level of injury is above the innervation of the sternocleidomastoid, the patient also may demonstrate weakness in rotating the face toward the opposite shoulder. Symptoms may seem to mimic shoulder dysfunction, with pseudo-weakness of the rotator cuff secondary to decreased stability of the scapula, which, in turn, can contribute to rotator cuff pathology.

3. How is the diagnosis of spinal accessory nerve injury made?
   Delayed diagnosis of a spinal accessory nerve injury as the cause of shoulder pain remains a common problem, particularly in iatrogenic injuries. An electrophysiologic evaluation is required to precisely identify the level of the lesion and assess for the potential for regeneration. Electrophysiologic testing will also reveal the degree of damage and, in particular, whether or not the nerve is still intact or completely severed. Imaging procedures, such as MRI and neurosonography, can also deliver important information for planning interventions.

4. What are the expected outcomes after a spinal accessory nerve injury?
   If the injury was from a stretch or whiplash, recovery is typically spontaneous over the course of several months, with an excellent outcome. If the nerve suffered surgical trauma, the most important prognostic factor that can influence the outcome is the timing of the corrective operation. It should be performed within 3 to 4 months after the injury and, at the latest, before 6 months. Outcomes after spinal accessory nerve surgery are typically good.

5. What are the common sites of entrapment of the suprascapular nerve?
   The suprascapular nerve courses from nerve roots C5 and C6 and runs posterolaterally to the suprascapular notch beneath the transverse scapular ligament. The nerve is commonly injured at the suprascapular notch by ganglia or tumor. Injury at the suprascapular notch affects both the supraspinatus and the infraspinatus muscles and mimics rotator cuff pathology. The presenting symptoms include shoulder joint pain, weakness in external rotation, and, to a lesser degree, weakness in abduction.

   The suprascapular nerve is also susceptible to traction and compression injuries because it travels around the spine of the scapula through the fibro-osseous tunnel formed by the spinoglenoid ligament and the spine of the scapula. Injury at this level results in strength changes in the infraspinatus muscle and shoulder pain, with sparing of the supraspinatus muscle. There may be wasting in the infraspinatus fossa.

6. What are common causes of injury to the suprascapular nerve?
   A common cause of injury is traction on the nerve produced by a retracted superior or posterior rotator cuff tear. Electrodiagnostic findings of suprascapular neuropathy have been reported in patients with massive rotator cuff tears. There are also published reports that suggest that neuropathy associated with retracted rotator cuff tear may partially or completely resolve with repair of the rotator cuff. The
suprascapular nerve may also be injured during surgery for rotator cuff repair because the cuff is pulled laterally to reattach, and tension is placed on the neurovascular bundle. Certain procedures for massive rotator cuff repair (double interval slide technique) place the suprascapular nerve close to the operative field, and iatrogenic injuries have been reported. Repetitive overhead athletes have been reported to experience neuropathy secondary to traction and microtrauma. Compression of the nerve can also occur because of a bone tumor or cyst secondary to a labral or capsular injury. Other causes include brachial neuritis, shoulder dislocation, fracture in the shoulder girdle, and penetrating injury.

Prevalence ranges from 12% to 33% in athletic populations and 8% to 100% in patients with massive rotator cuff tears.

7. What diagnostic tests are available to help confirm suprascapular nerve injury?
Electromyography and nerve conduction studies are usually considered the criterion standard for diagnostic testing for suprascapular nerve injury, although when each test is used alone, it is not directly diagnostic. In patients with weakness, electrodiagnostic testing has shown a diagnostic accuracy of 91%, leading to a single, correct diagnosis. Diagnostic nerve blocks of the suprascapular nerve at the suprascapular notch have been used with a positive result—defined as temporary relief of the pain being experienced by the patient. Sensitivity and specificity studies are lacking with regard to the value of diagnostic nerve blocks for this problem. Magnetic resonance imaging has been shown to have a sensitivity and specificity of 94.5% and 100%, respectively, in detecting muscular edema associated with nerve injury, using electromyography and nerve conduction studies as the reference standard.

8. What is the “Unhappy or Terrible Triad” in regard to the shoulder?
The “Terrible Triad,” or sometimes called the “Unhappy Triad,” describes an anterior shoulder dislocation along with rotator cuff tear and peripheral nerve injury. Dislocations should be considered as a clinical spectrum that includes 1) isolated dislocations, 2) injuries producing either detachment of the rotator cuff or neurologic deficit alone, and 3) combined injuries.

9. What nerve is most commonly injured after anterior shoulder dislocation?
The most common neurologic deficit after anterior shoulder dislocation is isolated axillary nerve palsy, although any component of the brachial plexus may be injured. The next most common nerve injury after dislocation is injury to the ulnar or median nerves. Neurologic injury occurs mostly in the young, predominantly male population during sports activities. The axillary nerve is most vulnerable to injury in anterior shoulder dislocations because it travels from the quadrilateral space, passing anteriorly and lying against the surgical neck of the humerus. The incidence of axillary nerve injury has been reported to be between 19% and 55% after anterior shoulder dislocations and up to 58% of proximal humeral fractures. Full recovery of axillary nerve injury, resulting from dislocation or fracture, occurs 85% to 100% of the time with nonoperative management within 6 to 12 months from the time of injury.

10. Describe the motor and sensory distributions of the musculocutaneous nerve.
The musculocutaneous nerve, which arises from the roots of C5, C6, and sometimes C7, is the terminal branch of the lateral cord of the brachial plexus. It innervates and penetrates the coracobrachialis muscle and travels between and innervates the biceps brachii and brachialis muscles. The musculocutaneous nerve emerges laterally to the biceps tendon as the lateral antebrachial cutaneous nerve, providing sensory innervation to the lateral forearm. Damage to this nerve causes weakness in elbow flexion and supination and numbness or paresthesias in the lateral forearm.

11. What are the common mechanisms of injury to the musculocutaneous nerve?
Isolated musculocutaneous nerve injury is very rare and is usually described in association with other nerve injuries, such as brachial plexus injury. Many of the reported cases have been sports-related and include weight lifting, resistive exercise, rowing, football and baseball throwing, swimming, tennis, racquetball, and windsurfing. Traumatic causes include fractures or dislocations of the humerus, fracture of the clavicle, gunshot or stab wounds, entrapment by the coracobrachialis muscle, heavy exercise and complications from anterior shoulder surgery.

12. What are common causes for long thoracic nerve injuries?
Isolated injury to the long thoracic nerve is rare. Traumatic causes of injury result from overuse and strenuous exercise of the shoulder, blunt trauma, or sudden depression of the shoulder. Iatrogenic nerve injury may occur after axilla or chest surgery or after incorrect positioning of the arm during general anesthesia. Long thoracic mononeuropathy is sometimes associated with infectious diseases, natural delivery, cervical manipulation, electric burn, C7 radiculopathy, or use of a single axillary crutch.
13. What are expected outcomes for someone with a long thoracic nerve palsy?
Overall, outcomes are generally favorable, even with no surgical intervention. If the cause of injury was inflammatory or idiopathic, the probability of a full recovery is increased. Electromyography (EMG) is an invaluable tool to confirm the clinical diagnosis of long thoracic mononeuropathy, but EMG findings do not seem to be a good predictor of the final outcome.

14. What are the common causes of brachial plexus injuries?
Motorcycle/snowmobile accidents, gunshot wounds, traction to arm or neck, fractures of the humerus, dislocations of the shoulder, primary nerve tumors, metastatic breast cancer, and radiation therapy can cause brachial plexus injuries. Closed injuries account for the majority of brachial plexus injuries, and 75% of injuries occur at the root level. Idiopathic brachial neuritis (Parsonage-Turner syndrome or neuralgic amyotrophy) is a postinfectious inflammatory condition that initially presents with acute onset of painful upper limb weakness. It is thought to be immune mediated. This initial phase is followed by a painless paresis that typically recovers over a 6- to 18-month time span.

15. What are the clinical signs and symptoms of typical brachial plexus injuries?
- Upper trunk lesions affect the suprascapular, musculocutaneous, and axillary nerves as well as parts of the median and radial nerves. Patients’ symptoms include weakness in shoulder flexion, abduction, and extension as well as marked weakness in elbow flexion, supination, and pronation and in wrist flexion. Areas of numbness and paresthesia may include the lateral forearm and hands as well as the thumb and index fingers.
- The middle trunk is rarely injured in isolation. Lesions produce weakness in the general distribution of the radial nerve, partially involving the triceps and sparing the brachioradialis.
- Lower trunk lesions cause motor weakness in muscles innervated by the ulnar nerve, the C8 components of the radial nerve, and muscles innervated by the distal median nerve, including the thenar muscles and the lumbricals. Patients have profound weakness of hand intrinsic muscles and sensory changes in the medial forearm (medial antebrachial cutaneous nerve), the medial hand, and the entire ring and little fingers.
- Lesions of either the posterior or anterior division are rare in isolation, although a posterior division lesion has been reported.
- Lateral cord lesions are similar to upper trunk lesions with sparing of the suprascapular nerve and upper trunk contributions to the axillary and radial nerves. Normal shoulder strength in flexion, extension, abduction, and external rotation; weakness in elbow flexion, supination, and pronation and wrist flexion; and numbness in the lateral forearm implicate the lateral cord.
- Medial cord lesions are similar to lower trunk lesions with sparing of C8 contributions to the radial nerve. Finger extension has normal strength.
- Posterior cord lesions are rare in isolation.

16. What key muscle tests help differentiate a C5–C6 root injury from a lateral cord lesion?
A C5–C6 root lesion affects all C5–C6 muscles, whereas an upper trunk lesion typically spares the dorsal scapular nerve to the rhomboids and the long thoracic nerve to the serratus anterior. A lateral cord lesion spares the suprascapular nerve (shoulder external rotation and abduction) as well as contributions to the posterior cord.

17. What is thoracic outlet syndrome (TOS)?
Thoracic outlet syndrome (TOS) refers to the compression of the neurovascular structures (roots or trunks of the brachial plexus and axillary or subclavian arteries) between the neck and axilla. TOS can be subdivided into vascular or neural compression symptoms, or both, depending on which specific structures within the cervicoaxillary canal are compromised. Klaassen reports the majority of cases (nearly 95%) are neurogenic in nature. True neurologic TOS manifests as a chronic lower trunk brachial plexopathy, caused by anatomic anomalies. The anomalies include a taut band extending from near the tubercle of the first thoracic rib to the tip of either the C7 transverse process or a rudimentary cervical rib. The C8 and T1 anterior primary rami can be stretched around this band either before or after, forming the lower trunk. Electromyography may show evidence of denervation in the intrinsic hand muscles, but this is not common. Neural compression symptoms occur more commonly than vascular symptoms. The cause of TOS also can be traumatic. A midshaft fracture of the clavicle occasionally results in injury to the blood vessels or brachial plexus, which are situated between the clavicle and first thoracic rib. With this type of injury, the terminal portion of the subclavian artery, the initial portion of the subclavian vein, and the proximal aspects of the cords of the brachial plexus...
may be damaged. Urschel and Razzuk performed a 50-year analysis of 2210 patients treated surgically for TOS, describing 250 with upper plexus compression, 1508 with lower plexus compression, and 452 symptomatic for both.

18. Describe the various tests used to evaluate a patient suspected of having TOS.

- The Adson maneuver is performed in the sitting or standing position with the examiner palpating the radial pulse in the patient’s abducted and extended arm. The examiner extends and externally rotates the arm as the patient rotates his or her head toward the examiner and takes a deep breath. A diminished or absent radial pulse suggests compression of the subclavian artery by the scalene muscles. Specificity has been reported from 32% to 87%.

- The Allen test is similar to Adson’s test, except the arm is abducted 90 degrees and the elbow is flexed 90 degrees. The patient turns his or her head away from the examiner and holds the breath. A diminished or absent radial pulse is a positive finding. Specificity has been reported from 18% to 43%.

- In the Roos test the patient holds both arms in the 90/90 position of the Allen test and then rapidly opens and closes the fingers for 3 minutes. Inability to maintain the test position, diminished motor function of the hands, or decreased sensation or paresthesia is suggestive of TOS secondary to neurovascular compromise.

- In the Wright test the arm is hyperabducted so that the hand is brought over the head with the elbow and arm in the coronal plane. Wright advocated performing the test in the sitting and then supine positions. Taking a breath or rotating or extending the head and neck may have an additional effect. The pulse is palpated for differences. This test is used to detect compression in the costoclavicular space.

- The costoclavicular syndrome test or military brace is accomplished by palpating the radial pulse and drawing the patient’s shoulder down and back. A positive test is indicated by the absence of the pulse. Sensitivity has been reported at 95%, with specificity from 53% to 85%.

- In the provocation elevation test the patient elevates both arms above the horizontal and rapidly opens and closes the hands 15 times. If fatigue, cramping, or tingling occurs, the test is positive for vascular insufficiency and TOS.

- In the shoulder girdle passive elevation test, the patient crosses one arm on the chest. The examiner stands behind the patient and passively elevates the shoulder girdle upward and forward (passive shoulder shrug). The position is held for 30 seconds. A positive test is reported if the pulse becomes stronger, skin color improves, or hand temperature increases. The patient also may report a “relief phenomenon,” which can range from numbness, pins and needles, or pain as the ischemia to the nerve is released.

- In the Halstead maneuver the radial pulse is palpated and the examiner applies a downward traction on the arm while the patient’s neck is hyperextended and the head is rotated to the opposite side. Absence or decreased pulse indicates a positive test for TOS.

19. What diagnostic tests are helpful in diagnosing TOS?

Klaassen describes one of the issues with TOS as a lack of a gold standard for definitive diagnosis. Radiographs, CT scans, and MRIs provide for detection of cervical ribs and fibrous bands for identification of potential factors causing TOS. Confirmation of a vascular abnormality is aided by the use of duplex ultrasound, which has been found to be 92% sensitive and 95% specific. In addition, electrophysiologic testing is valuable for differential diagnosis and determining the presence of additional abnormalities such as cervical nerve root or distal peripheral nerve pathology.

20. How many TOS tests should be performed in a clinical examination?

The false-positive rate for all of the TOS tests is relatively high. Many of them test only the vascular component of TOS. One way to decrease the chance of a false-positive test is to perform at least three different tests. The literature reports a false-positive rate of 12% when two TOS tests are performed. If three or more are performed, the false-positive rate can be reduced to 2% or less. Gillard reports a mean sensitivity and specificity of 72% and 53%, respectively, when the Adson, hyperabduction, and Wright tests are used in a cluster.

21. What outcomes are associated with physical therapy treatment of TOS?

Physical therapy treatment of TOS shows the following typical outcomes: 60% will have symptomatic improvement, 24% will not change, and 16% continue to worsen.
22. What outcomes are associated with surgical treatment of TOS?
The frequency of good to excellent results varies widely, ranging from 24% to 90%. In the Urschel and Razzuk study of 2210 patients that were treated with a transaxillary first rib resection, 95% of the patients reportedly became asymptomatic postoperatively. However, 1221 patients had recurrent symptoms, requiring a second surgery with a concomitant dorsal sympathectomy in patients who had not received one during their initial surgery. Abnormal somatosensory evoked potentials (SSEPs) and arterial photoplethysmography correlate with improved results.

23. What causes “dead arm syndrome”?
Dead arm syndrome historically has been attributed to various causes, including recurrent transient anterior shoulder subluxation, rotator cuff tear, labral tears, and psychological disorders. Often radiographs and electromyograms are normal, and the young athlete is frustrated. The mechanism of injury in overhead-throwing athletes appears to be related to acceleration in the late-cocking phase of throwing. The injury also can be caused by direct trauma to the arm. Some believe a type 2 superior labral anterior-to-posterior (SLAP) lesion, with or without rotator cuff involvement, is the underlying cause. Although many symptoms described by patients suggest possible neural compromise, true neurologic changes are rarely, if ever, present.

24. What is a “burner”?
A “burner” or “stinger” is a nerve injury that often occurs during sporting activities, most frequently in football. It is generally thought to be a traction or compression injury of the upper trunk of the brachial plexus or the fifth or sixth cervical roots. The disorder usually produces transient pain, numbness, and paresthesia. Chronic burner syndrome may result from nerve root compression in the intervertebral foramina secondary to disc disease in older collegiate and professional athletes.

25. Describe the clinical findings of a patient with Pancoast’s tumor.
A Pancoast tumor (also known as a superior sulcus tumor) can compromise the C8–T1 roots of the brachial plexus via compression from the apex of the lung. The peripheral location of these tumors minimizes typical lung cancer symptoms, such as cough or hemoptysis, and patients with Pancoast tumors are often not recognized until a later stage of diagnosis. The presenting symptoms of the patient are sensory changes in the medial forearm and hand, including the fourth and fifth digits. Other signs may include intrinsic muscle wasting, Horner syndrome, and a history of night pain. Clinicians should be especially suspicious of a Pancoast tumor in smokers who have these symptoms and no history of trauma or neurologic disease.

26. What is a “burner” or “stinger”?
“Burner” or “stinger” are commonly used terms to describe a transient upper trunk brachial plexus neuropraxia that usually occurs during a contact sport. The athlete often describes a “burning” or “stinging” sensation down the arm when injured. Several mechanisms of injury have been proposed, including traction of the plexus due to rapid lateral flexion of the neck as well as compression of the fixed brachial plexus between the shoulder pad and the superior medial scapula when the pad is pushed into the area of Erb’s point, where the brachial plexus is most superficial.

Researchers have identified high rates of structural abnormalities in the cervical spine in athletes with recurrent stingers, particularly spinal canal or neural foraminal stenosis.

27. What are effective management/prevention strategies for burners and stingers?
Strength training to the cervical spine and shoulder girdle musculature is an effective way to reduce the occurrence of burners and stingers in football players. In a biomechanical analysis comparing the Cowboy Collar, Bullock Collar, and Kerr Collar, it was found that the Kerr Collar and Bullock Collar reduced head accelerations and force transmission through the neck. According to Rowson et al., these reductions in loads correlated with the degree to which each collar restricted the motion of the and neck. They concluded that restricting the range of motion of the neck and redistributing load to the shoulders, neck loads can be effectively lowered.

BIBLIOGRAPHY
CHAPTER 45 QUESTIONS

1. John is a 36-year-old male who complains of left shoulder pain and weakness in his shoulder. On examination he has full ROM of his shoulder but has 3/5 weakness in external rotation and 4+/5 shoulder abduction. His sensory exam was normal, and he does not have increased pain with resisted shoulder motions. John most likely has a lesion involving the:
   a. Axillary nerve
   b. Spinal accessory nerve
   c. Suprascapular nerve
   d. Upper trunk of the brachial plexus

2. The “Unhappy or Terrible Triad” related to the shoulder refers to a/an:
   a. Acromioclavicular arthritis, rotator cuff tear, and peripheral nerve injury
   b. Anterior shoulder dislocation, rotator cuff tear, and peripheral nerve injury
   c. Glenohumeral arthritis, rotator cuff tear, and peripheral nerve injury
   d. Rotator cuff tear, labral lesion, and peripheral nerve injury

3. Mary is a 23-year-old, right-hand-dominant volleyball player who has developed right shoulder pain and winging of her right scapula. She has full cervical and upper extremity ROM. She appears to have near normal strength in her shoulder, and resisted motions of the shoulder are painless. However, she has marked right scapular winging with wall push-ups. This is most likely a lesion of the:
   a. Dorsal scapular nerve
   b. Long thoracic nerve
   c. Spinal accessory nerve
   d. Suprascapular nerve
1. Describe the joints of the elbow.
The elbow consists of three joints: the ulnohumeral, radiocapitellar, and proximal radioulnar joints. The olecranon forms the greater sigmoid notch of the ulna, which articulates with the trochlea to form a uniaxial ginglymoid joint. The radiocapitellar and proximal radioulnar joints form a trochoid or pivoted joint. The thin elbow capsule and synovial membrane define the confines of the joint, beginning proximal to the coronoid and olecranon fossae and ending beyond the tips of the coronoid and olecranon processes. Because the maximal volume of the capsule is 15 to 30 mL at 80-degree flexion, the elbow often is held in this position to minimize pain from capsular distention secondary to acute hemiarthrosis.

2. What is the normal carrying angle of the elbow?
The carrying angle of the elbow varies with flexion and extension, ranging from 6 degrees of varus with full flexion to 11 degrees of valgus in full extension. In men the mean value is between 11 and 14 degrees (full extension). Some studies show that women tend to have larger carrying angles than men, with an average value between 13 and 16 degrees.

3. Describe the articular geometry of the distal humerus.
The articular surface has a 30-degree anterior angulation, 5 to 7 degrees of internal rotation, and 6 to 8 degrees of valgus tilt.

4. Describe the interosseous membrane (IOM) of the forearm.
The interosseous membrane is composed of the central band, the proximal band, several accessory bands, and the membranous portion. The most important structure is the central band, which originates from the radius and is angled distally to attach to the ulna at a 21-degree angle. The central band is 1.5 to 2 cm wide and is responsible for 71% of the IOM stiffness after excision of the radial head.

5. What portion of the longitudinal growth of the upper arm does the elbow contribute?
The elbow accounts for only 20% of the total longitudinal growth of the humerus. The proximal humerus accounts for the remaining 80%.

6. What structures contribute to elbow stability?
Elbow stability is maintained by a combination of bony and soft tissue components. Primary stabilizers include the coronoid (ulnohumeral joint), lateral ulnar collateral ligament, and anterior band of the medial collateral ligament. Secondary stabilizers include the radial head, extensor and flexor muscle masses, and joint capsule.

7. Describe the medial ligamentous complex.
The main constraint to elbow valgus instability is the medial collateral ligament (MCL). The MCL originates on the central two thirds of the anteroinferior medial condyle and inserts onto the anteromedial coronoid. It has three distinct bundles. The anterior bundle, which is the strongest, inserts on the anterior coronoid and greater sigmoid notch. The thin posterior bundle attaches to the posterior greater sigmoid notch. The oblique bundle is variable in its attachments. The anterior bundle is divided into anterior and posterior bands. The anterior band is the primary restraint to valgus stress from 30 to 90 degrees, while the posterior band tensions from 90 to 120 degrees.

8. Describe the lateral ligamentous complex.
The lateral collateral ligament (LCL) complex consists of the radial collateral ligament (lateral epicondyle to annular ligament), the annular ligament (anterior to posterior edge of sigmoid notch), the accessory collateral ligament (variable, posterior annular ligament to supinator crest), and the lateral ulnar collateral
ligaments (LUCLs, lateral epicondyle to supinator crest), which blend intimately with the underlying joint capsule and more superficial extensor tendons. The LUCL insertion may be broad-based or bilobed and is the primary constraint to posterolateral rotatory instability.

9. Describe the most important varus and valgus stabilizers of the elbow at 0 and 90 degrees of flexion.

Varus stress at the elbow is resisted by the LCL, anconeus muscle, and joint capsule. With full extension, the LCL contributes 14% of the restraint to varus stress; 54% is provided by the joint surface and 32% by the capsule. With 90 degrees of flexion, restraint to varus stress provided by the LCL, joint articulation, and capsule changes to 9%, 78%, and 13%, respectively.

Valgus stress is resisted mainly by the fan-shaped MCL complex, which consists of anterior, intermediate, and posterior fibers. The anterior oblique fibers are taut throughout flexion-extension and are the most important valgus stabilizers. The posterior oblique ligaments are taut only during flexion. At full extension, contributions from the MCL, joint surface, and anterior capsule to resisting valgus stress are equal. At 90 degrees of flexion, the MCL contributes 54% of the resistance, the radial head contributes 30%, and the remainder is supplied by articular congruity and the anterior capsule.

10. What provides the most dynamic stabilization of the medial elbow?
The flexor carpi ulnaris (FCU) provides the greatest stability followed by the flexor digitorum profundus (FDS) and pronator teres (PT).

11. Describe posterolateral rotatory instability.
Posterolateral rotatory instability (PLRI) is a common pattern of acute elbow instability, caused by a fall onto an outstretched arm. The humerus rotates internally on the elbow, which undergoes external rotation and valgus loading as the elbow flexes. Specifically, the ulnar rotates externally while the radiohumeral joint subluxates posterolaterally, allowing the coronoid to pass under the trochlea as the ulna swings into a valgus position.

12. What is the Morrey elbow instability scale?
Morrey described five elbow instability types based on damage to particular structures about the elbow:
- Type 0—elbow reduced and stable when stressed
- Type I—PLRI with a positive shift test; torn LUCL
- Type II—perched condyles, unstable elbow with varus stress; torn LUCL and anterior and posterior capsules
- Type IIIa—posterior dislocation of the elbow with valgus instability; torn LUCL, posterior MCL, and anterior and posterior capsules
- Type IIIb—posterior dislocation of the elbow with gross instability; torn LUCL, anterior MCL, posterior MCL, and anterior and posterior capsules

13. During closed-chain upper extremity exercise, how much weight is transmitted through the radiocapitellar and ulnohumeral joints?
Approximately 60% of the force is transferred through the radiocapitellar joint and 40% through the ulnohumeral joint. The greatest amount of force is transmitted between 0 and 30 degrees of flexion.

14. Describe normal arthrokinematics at the elbow.
Motion at the elbow is primarily gliding for both flexion and extension. Rolling occurs in the final 5 to 10 degrees of range of motion (ROM) for both flexion and extension. Minimal adduction may occur with flexion and minimal abduction with extension, although the magnitude of these movements is debated.

15. Differentiate “normal” from “functional” elbow ROM.
The normal average ROM of the elbow is from 0 degrees (full extension) to 150 degrees (full flexion) with 85 degrees of supination and 80 degrees of pronation. However, activities of daily living usually can be accomplished with a ROM of 30 to 130 degrees of flexion, 50 degrees of supination, and 50 degrees of pronation. If pronation and supination are normal with good motion of the wrist and shoulder, functional mobility may occur with as little as 75 to 120 degrees of motion.

16. Where is the axis of flexion and extension in the elbow? Where is the axis during pronation and supination?
The axis of flexion of the elbow is a line through the center of the capitellum and the center of curvature of the trochlear groove, colinear with the distal anterior humeral cortex. Motion resembles a “loose hinge,” with 3 to 5 degrees of rotation and varus/valgus motion during the flexion arc. During pronation and supination, the radius rotates along an axis passing through the center of the radial head and the distal ulnar fovea. The radial head translates 1 to 2 mm proximally during pronation.
17. Which muscle is considered the “workhorse” of elbow flexion?
The brachialis muscle is the primary flexor of the elbow, inserting approximately 1 cm distal to the
coroid onto both the ulna and the capsule. The brachioradialis has the longest lever arm.

18. What is the primary function of the brachioradialis?
The brachioradialis is active during all aspects of elbow flexion regardless of forearm rotation, indicating
its role as elbow stabilizer. It is also more active in pronation than supination, indicating it acts as a
secondary pronator.

19. Describe the effect of speed on muscle recruitment during supination.
During slow, unresisted supination activity, the supinator may act independently. However, all rapid and
resisted movements are assisted by the biceps. This holds true regardless of elbow position.

20. Describe the effects of speed and joint angle on pronation activity.
The pronator quadratus is the primary pronator of the forearm, regardless of elbow position. With
increasing speeds or resistance, activity of the pronator teres increases.

21. What is the effect of changing forearm position on muscle testing of elbow flexion
strength?
Resisting elbow flexion with the forearm in neutral position places maximal stress on the
brachioradialis muscle. Testing the elbow with the forearm pronated minimizes bicep activity.
Forearm position does not affect the activity of the brachialis.

22. At what position are elbow flexion strength and supination strength maximal?
Elbow flexion strength is maximal at 90 to 110 degrees of flexion. The biceps act most strongly as a
supinator at 90 degrees of flexion. Pronation strength is 15% to 20% less than supination strength in the
normal elbow.

23. Describe the innervation of the various muscles controlling movement at the elbow.

<table>
<thead>
<tr>
<th>Action</th>
<th>Muscles</th>
<th>Nerve Root</th>
<th>Nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexion</td>
<td>Brachialis</td>
<td>C5, C6</td>
<td>Musculocutaneous</td>
</tr>
<tr>
<td></td>
<td>Biceps brachii</td>
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<tr>
<td></td>
<td>Brachioradialis</td>
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<td>Radial</td>
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<tr>
<td>Extension</td>
<td>Triceps</td>
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<td>Median</td>
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<td></td>
<td>Pronator quadratus</td>
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<tr>
<td>Supination</td>
<td>Biceps</td>
<td>C5, C6</td>
<td>Musculocutaneous</td>
</tr>
<tr>
<td></td>
<td>Supinator</td>
<td>C5, C6, C7</td>
<td>Deep branch of radial</td>
</tr>
</tbody>
</table>

24. Which arteries supply blood to the elbow?
Three arcades surround the elbow joint. The medial arcade is formed by the superior and inferior ulnar
collateral arteries and the posterior ulnar recurrent artery. The posterior arcade is formed by the medial
and lateral arcades and the middle collateral artery. The lateral arcade is formed from the radial and
middle collateral, radial recurrent, and interosseous recurrent arteries.

25. What is the order (and approximate age) of ossification of structures around
the elbow?
Ossification follows the acronym CRMTOL: capitellum (6 months to 2 years), which includes the
lateral crista of the trochlea; radial head (4 years); medial epicondyle (6–7 years); trochlea (8 years);
olecranon (8–10 years); and lateral epicondyle (12 years).

26. Describe the anatomy of the ulnar nerve at the elbow.
The ligament of Osborne is present in all elbows, and two thirds of elbows will also display a
discrete arcade of ligament of Struthers. An average of one capsular branch diverges from the ulnar
nerve 7 mm proximally to the medial epicondyle. An average of three motor branches to the flexor
carpus ulnaris (FCU) is typical. Approximately 45% of elbows will have an aponeurosis distal to
Osborne’s ligament, which runs between the FCU and medial epicondylar muscles.
27. Does the ulnar nerve really innervate the medial triceps?
Gross connection of the ulnar nerve to the medial triceps may be apparent; however, these fibers typically can be traced back as branches originally carried by the radial nerve that crossed over in the axillary region.

28. The medial antebrachial cutaneous nerve is subject to painful neuromas if disrupted during surgery. Where do branches of this nerve typically cross the medial elbow?
In approximately 61% of the time, a branch is noted an average of 1.8 cm above the medial epicondyle; 100% of the time branches cross distally to the medial epicondyle at an average distance of 3.1 cm.

29. What is the blood supply to the extensor carpi radialis brevis (ECRB) tendon?
The radial recurrent artery supplies the tendon through branches on its medial and lateral borders. Important contributions are given from the posterior branch of the radial collateral artery and more minor contributions from the interosseous recurrent artery. These arteries form a superficial network with the deep portion of the tendon being nearly avascular.

30. Describe the anatomy of the lateral joint capsule.
The ECRB originates as a tendon with no muscular fibers, which are seen in other extensors. The anterior portion is thin under the ECRB, whereas the posterior portion was thicker and mingled with fibers from the supinator and annular ligament. This thin attachment can explain why patients with lateral epicondylitis often have pain with stressing of the lateral joint capsule, which may play a role in the development of lateral epicondylitis.

31. What is the relationship of the posterior interosseous nerve (PIN) near the lateral elbow?
Pronation of the forearm increases the distance from the capitellum to the PIN to an average of 52 mm. Supination draws the nerve proximal with an average distance of 33 mm from the capitellum.

32. What is the innervation pattern of the radial nerve in the forearm?
The most common innervation pattern in the forearm was brachioradialis, extensor carpi radialis longus, superficial sensory, extensor carpi radialis brevis, supinator, extensor digitorum/extensor carpi ulnaris, extensor digiti minimi, abductor pollicis longus, extensor pollicis brevis, extensor pollicis longus, and extensor indicis.

33. What distal bicep tendon repair technique is at greatest risk for radioulnar impingement?
Repair with suture anchors carries greater risk than a bony trough or suture button technique.

34. What are the differences in function regarding the long and short head of the biceps at the elbow?
The short and long heads of the biceps have separate insertions. The short head’s insertion allows greater efficiency with the elbow flexed at 90 degrees. In the neutral and pronated forearm, the short head is a more efficient supinator. In the supinated forearm, the long head becomes more efficient at supination.

35. What advantage does a two-incision technique have over a one-incision technique for distal biceps repairs?
A two-incision technique allows for a more anatomic repair to the tuberosity, likely allowing for increased strength, particularly into supination.

36. How is elbow flexion strength affected after release of the brachioradialis tendon during repair of distal radius fractures?
Brachioradialis torque does not drop down to less than 80% of normal, and therefore, overall elbow flexion torque changes less than 5% because of the primary flexion effects of the biceps and brachialis.

BIBLIOGRAPHY


**CHAPTER 46 QUESTIONS**

1. What is last muscle in the forearm that is innervated by the radial nerve?
   - a. Extensor indicis
   - b. Extensor digitorum
   - c. Extensor digiti minimi
   - d. Abductor pollicis longus

2. The anterior lateral joint capsule of the elbow:
   - a. Is thick and stout
   - b. Blends with the annular ligament
   - c. Blends with the supinator
   - d. Is thin under ECRB

3. The last growth plate to ossify in the elbow is the:
   - a. Capitellum
   - b. Lateral epicondyle
   - c. Medial epicondyle
   - d. Olecranon
1. What are patient-reported outcome questionnaires, and which is best when working with patients with elbow pathologies?

There are two basic types of outcome measures used in orthopedic practices, clinician-based outcome measures and patient-reported outcome questionnaires. Clinician-based measures can be affected by observer bias. Patient-reported measures provide their perspective and have been associated with prediction of return to work. Three of the most commonly used patient-reported outcome questionnaires are the Patient-Related Elbow Evaluation (PREE), the patient-reported form of the American Shoulder and Elbow Surgeons elbow Questionnaire (pASES-e), and the Disabilities of the Arm, Shoulder, and Hand Questionnaire (DASH). Vincent et al. (2014) compared the internal consistency, concurrent construct validity, longitudinal validity, and sensitivity to change features of these three commonly used questionnaires prospectively on 128 patients. Their conclusions were that all three questionnaires have acceptable validity and sensitivity to change. However, the pASES-e function subscale was the least sensitive to change and is less correlated to the other measures.

2. Describe an elbow with joint effusion.

All three joints of the elbow complex are affected because they have a common joint capsule. The joint swelling is most evident in the triangular space between the radial head, tip of the olecranon, and lateral epicondyle. The elbow is held in the loosely packed position of about 70 degrees of flexion because, in this position, the joints have maximal volume.

3. What is “little league elbow”?

Little league elbow is a generic term referring to several overuse injuries in young throwers. Examples include osteochondritis dissecans of the capitellum with or without loose bodies, injury and premature closure of the proximal radial epiphysis, overgrowth of the radial head, and medially stressed valgus overuse. The repetitive valgus stress of throwing results in microtrauma of the medial anterior oblique ligament and compression of the radiocapitellar joint. Repeated traction on the olecranon at the site of the triceps brachii insertion may produce olecranon apophysitis or an olecranon stress fracture. Excessive repeated traction through the medial elbow may result in enlargement of the medial humeral epicondyle as well as inflammation of the medial humeral apophysis. Osteochondrosis dissecans of the radial head and/or capitellum or osteochondrosis of the capitellum (Panner’s disease) may result from compressive forces through the lateral elbow during the throwing motion. These same forces can result in injury to the proximal radial epiphysis and early closure of its growth center.

4. How is little league elbow treated?

In general, little league elbow is treated with relative rest and absolutely no throwing for up to 1 year. If significant fragmentation or separation of the medial humeral apophysis is seen on plain radiographs, surgery may be indicated.

5. Describe the recommended sequence of pitches for adolescent athletes.

One of the main causes of elbow injury in adolescent athletes is throwing pitches that they are not physically prepared to perform. Baseball’s Medical and Safety Advisory Committee has recommended when various pitches should be introduced. The first pitch introduced is the fastball at 8 years, followed by the change-up at 10 years, the curveball at 14 years, the knuckleball at 15 years, and the slider and forkball at 16 years.
6. What functional tests help confirm the diagnosis of little league elbow?
Flexing and extending the elbow with maintenance of valgus stress should elicit elbow pain. Valgus stress testing may reveal pain and increased range. Loss of passive elbow extension may result from early flexion contracture, which is common in professional pitchers and may represent serious damage in children or adolescents.

7. What is lateral epicondylitis?
The term “tendinitis” has been used to describe a hypothetical chronic inflammatory process in the overused tendon. However, histologic examinations of excised pathologic tendons have consistently failed to display the presence of inflammatory cells. Instead, the tissue is characterized by the presence of dense populations of fibroblasts, vascular hyperplasia, and disorganized collagen, termed by Nirschl as angiofibroblastic hyperplasia. Angiofibroblastic hyperplasia appears to be the result of a failed healing response to microtears, combined with vascular deprivation in the tendon’s origin, preventing healing to occur.

8. Which structure is most commonly involved in lateral epicondylitis (tennis elbow)?
The most commonly involved structure is the extensor carpi radialis brevis (ECRB) tendon, followed by the extensor digitorum communis (EDC) tendon. These tendons may be histologically indistinguishable at the common origin. The ECRB tendon has the greatest electromyography (EMG) activity of the forearm muscles, especially in the acceleration and early follow-through phases of the tennis swing. Tendon fibers attaching to the periosteum are relatively avascular and tend to heal very slowly. Immature granulation tissue is present at the injury repair site.

9. What are the differential diagnoses for lateral epicondylitis?
- Entrapment of the radial nerve
- Degenerative changes of the radiocapitellar joint
- Posterolateral rotatory instability
- Occult fractures of the radial head or lateral humeral epicondyle
- Posterior epicondylitis at the triceps attachment to the olecranon
- Panner’s disease
- Tumor of the capitellum or in the supinator muscle
- Rheumatoid arthritis
- Tendinitis of the long head of the biceps (caused by insertion on the radius)
- Cervical spinal problems

10. Are forearm support bands (counterforce braces) an effective orthosis for lateral epicondylitis?
Counterforce braces consist of a flexible band that fits around the proximal forearm and applies pressure to the underlying tissues during activity. These braces may reduce acceleration forces by 46%. Although one study showed that they increase the rate of fatigue in unimpaired people, other studies have shown decreased pain threshold with no changes in isokinetic strength.

11. Describe the incidence and demographics of lateral epicondylitis.
Lateral epicondylitis most commonly occurs in patients between 35 and 50 years of age. The incidence varies in different populations. In studies performed at industrial health clinics, epicondylitis was most commonly associated with work-related activities (35% to 64% of all reported cases). Tennis players also are at high risk: 10% to 50% will have symptoms at some time in their career. Risk increases with poor stroke mechanics, striking the ball off center, improper grip size, and harder court surfaces. Amateurs tend to have lateral epicondylitis secondary to the backhand, whereas professionals usually have medial epicondylitis because of forceful serving.

12. What is the best treatment for lateral epicondylitis?
Corticosteroid injections have shown short-term improvement (6 weeks) with this condition; however, the few long-term follow-up studies for this intervention show success rates at 60% or lower. There has been one study that has compared both corticosteroid injection and physical therapy. Hart found that at 6 weeks, 92% of patients receiving corticosteroid injection reported improvement in symptoms compared with 47% of patients receiving physical therapy. However, at 52 weeks only 69% of patients receiving corticosteroid injections had positive outcome measurements (self-reported and blinded assessments) compared with 91% for the physical therapy interventions. The physical therapy intervention used was nine treatments of pulsed ultrasound, deep friction massage, and an exercise program. Studies
comparing the effects of phonophoresis versus ultrasound have shown that both treatment options result
in decreased pain and increased pressure tolerance in selected soft tissue injuries, but the addition of
phonophoresis does not augment the benefits of ultrasound alone.

13. What is the Mills maneuver? Is it an effective treatment for lateral epicondylitis?
Numerous studies have reported that about 10% of patients with lateral epicondylitis are unresponsive to
conservative treatment. A final option before surgery is the Mills maneuver, which is intended to pull apart
the two surfaces joined by a painful scar. Once separation is attained, permanent lengthening of the
common extensor tendon results. In one study of over 100 resistant cases, repeat manipulation was
needed in only six patients and surgical intervention was needed in only one patient over a 20-year period.
The maneuver is performed with the patient in a supine position, the wrist in full flexion, and the
forearm fully pronated. The elbow is moved suddenly from a flexed position to full extension. This
maneuver is painful because it places maximal stretch on the scar. Wadsworth recommends performing
the maneuver only after the site is injected with 0.5 mL of methylprednisolone and 0.5 mL of 2%
lidocaine.

14. What are the common surgical treatments of lateral epicondylitis?
Nirschl and Pettrone recommend a procedure in which the degenerated extensor carpi radialis brevis
origin is resected and the lateral epicondyle is decorticated. In a 10-year follow-up study, pain relief was
noted in over 90% of patients. Spencer and Herndon recommend simple fasciotomy of the extensor origin.
They reported “excellent” or “good” results in 96% of 23 patients. Other studies have reported “excellent”
or “good” results in 31 of 35 patients at long-term follow-up. This technique is recommended because of
its simplicity, minimal complications, and recovery time of 3 to 4 weeks. Arthroscopic release of the ECRB
tendon has excellent results in 80% to 95% of the cases with early return to work (approximately
2 weeks).

15. What other treatments are available for lateral epicondylitis?
Autologous blood injections demonstrate ≈79% pain relief in those who have failed steroid injection and
exercise. Platelet-derived growth factor is thought to stimulate a healing response. Lithotripsy studies
have shown mixed reviews, with little benefit shown over placebo in the blinded studies.

16. What is radial tunnel syndrome? Why is it confused with lateral epicondylitis?
The radial tunnel is about 2 inches in length, extending proximally from the capitellum of the humerus,
between the brachioradialis and brachialis, and distally through the supinator muscle. The radial
nerve may become entrapped in this tunnel, resulting in persistent pain around the lateral epicondyle and
an aching sensation in the extensor and/or supinator muscle mass distal to the lateral epicondyle.
The nerve is typically impinged at the arcade of Froshe. Tennis elbow straps may increase symptoms
because of increased pressure compression over the radial tunnel.

17. What is “nursemaid’s elbow”?
Nursemaid’s elbow (“pulled elbow”) is subluxation of the radial head, usually in children younger than
5 years. It usually occurs when a child is forcefully pulled or jerked by the arm with the arm in extension.
Radiographs are of little benefit, even with comparison views of the uninjured elbow. The combination of
patient history and limitation of motion, especially absence of supination of the elbow, usually makes
the diagnosis. A sudden pull on the extended elbow while the forearm is pronated may produce a tear in
the distal attachment of the annular ligament to the radial neck. The radial head penetrates partially
through the tear as it is distracted from the capitellum. Then the proximal part of the annular ligament
slips into the radiohumeral joint, where it becomes trapped between the joint surfaces once the pull
is released. The source of pain is the trapped annular ligament. The entrapped ligament can be freed by
suddenly supinating the forearm while the elbow is flexed.

18. Describe medial epicondylitis.
Medial epicondylitis has been called golfer’s elbow, medial tennis elbow, and even swimmer’s elbow. It is
an overuse injury that results from repetitive valgus stress on the medial elbow, combined with wrist
flexion and pronation. The patient with medial epicondylitis usually presents with pain, inflammation, and
point tenderness at the medial epicondyle where the flexor/pronator group originates.

19. What are the differential diagnoses for medial epicondylitis? How are they ruled out?
The differential diagnoses for medial epicondylitis are medial collateral ligament (MCL) injuries, ulnar
nerve injuries, and degenerative changes of the medial elbow joint. Both medial epicondylitis and MCL
injury can create pain on valgus stress testing. It is possible to differentiate the two injuries by applying
valgus stress to a slightly flexed elbow while the wrist is flexed and the forearm is pronated. This arm position eliminates the symptoms attributed to medial epicondylitis and results in a painless valgus stress test, provided that the ulnar collateral ligament (UCL) is uninjured. Passive wrist extension and active resisted wrist flexion and pronation can further distinguish medial epicondylitis from UCL injury. A positive Tinel’s sign, tenderness of the ulnar nerve to palpation, and paresthesia and numbness in the fourth and fifth fingers confirm ulnar nerve injuries. According to Nirschl, 60% of patients with medial epicondylitis have ulnar nerve symptoms. Radiographic changes include bone spurs and degenerative disease.

20. **What is olecranon bursitis?**

   The olecranon bursa is located between the skin and tip of the olecranon process. Bursitis is caused by trauma because of chronic overuse (eg, leaning on the elbow or “student’s elbow”) or by direct impact that results in inflammation or infection. The differential diagnoses include fracture of the olecranon process of the ulna, gout, rheumatoid arthritis, and synovial cyst of the elbow joint. Usually the elbow joint is not involved because the bursa and joint do not communicate unless rheumatoid arthritis is present. If the joint is infected, all motion is resisted.

21. **Describe the management of olecranon bursitis.**

   Traumatic bursitis is managed symptomatically with immobilization in a splint, compressive dressings, and contrast baths. Aspiration of the bursa usually does not prevent recurrence of swelling because of continued flexion and extension activities. If the bursa is painful and prevents activity, aspiration is indicated and may be both diagnostic and therapeutic.

22. **How common is a rupture of the distal biceps tendon, and what is the etiology of injury?**

   Rupture of the distal biceps tendon is a relatively rare injury that can have significant functional impact on the upper extremity. In a retrospective study performed by Safran and Graham, the incidence rate was found to be 1.2 per 100,000 patients with an average age of 47 years at the time of injury. The dominant arm was involved 86% of the time, and smokers were 7.5 times more likely to sustain the injury compared with nonsmokers.

   The theories for rupture of the distal biceps tendon include hypovascular and mechanical mechanisms. Seiler et al. performed an anatomic study that included vascular injections of 27 cadavers. A consistent, hypovascular zone, measuring 2.14 cm in diameter, was found at the musculotendinous junction with light microscopy. Other studies used sequential computed tomography scans of patients with their forearms in positions of maximal pronation, neutral, and maximal supination. With the forearm in maximum pronation, the distance between the lateral border of the ulna and the radial tubercle was 48% less than the distance in full supination. Additionally, with the forearm in pronation, the biceps tendon consumed 85% of the radioulnar space at the level of the radial tubercle. Thus, mechanical impingement of the tendon is the other proposed theory for distal biceps tendon rupture.

23. **What is the clinical presentation for a patient with a distal biceps tendon rupture?**

   Patients with a distal biceps tendon rupture frequently report sustaining an unexpected forced extension of the elbow, resulting in an eccentric contraction of the biceps and a tearing sensation in the antecubital fossa. The patient will also report weakness with elbow flexion and significant weakness with forearm supination.

24. **What clinical tests can be used to assess for a distal biceps tendon rupture?**

   Despite the clinical presentation, a distal biceps tendon ruptures can still be missed clinically, especially if the lacertus fibrosus remains intact. The two most common clinical tests are the biceps squeeze test and the hook test.

   Ruland et al. developed the biceps squeeze test, which is similar to the Thompson test used to identify Achilles tendon ruptures. The biceps brachii is squeezed, which will result in forearm supination if the tendon is intact. In Ruland’s study, 23 of 24 patients with a positive test had a complete distal biceps tendon rupture, which was confirmed surgically or by MRI.

   O’Driscoll et al. developed the hook test to identify distal biceps tendon ruptures. The test is performed by placing the patient’s elbow at 90 degrees of flexion and inserting a finger under the lateral edge of the biceps tendon and hooking the finger under the cord-like structure, crossing the antecubital fossa. The authors of this study reported 100% sensitivity and specificity for this test. One key with the hook test is to hook the lateral edge of the biceps tendon, not the medial edge, so that the lacertus fibrosus would not be mistaken for an intact biceps tendon.
25. What are the outcomes for untreated, complete distal biceps tendon tears?
Flexion strength is decreased 20% to 30%, and supination strength is decreased 40% to 50%, compared with the uninvolved limb.

26. What are the outcomes for surgically repaired acute distal biceps ruptures?
Typically, strength is 90% to 95% of the contralateral limb, and motion is normal or near normal. Slight decreases in extension, pronation, or supination may be present.

27. What are potential complications of distal bicep tendon repairs?
Transient or permanent paresthesias, PIN palsy, rerupture, tuberosity fracture, vascular injury, heterotopic bone formation, radial ulnar synostosis, infection, and hematoma.

28. What is the clinical presentation of osteoarthritis of the elbow?
Primary osteoarthritis of the elbow is uncommon but usually presents on the dominant side in middle-aged males with a history of heavy use through sports or work. In the early stages, the typical presentation is pain at end range extension and flexion. This has been associated with osteophytes at the tips of the coronoid process and the olecranon. As the disease progresses, there is pain throughout the range of motion. The osteophytes can enlarge and actually become space-occupying lesions, which will lead to capsular contracture.

BIBLIOGRAPHY


CHAPTER 47 QUESTIONS

1. How much supination strength is lost after an untreated distal biceps rupture?
   a. 10%
   b. 20%
   c. 40%
   d. 70%
2. Lateral epicondylitis primarily affects the:
   a. Brachioradialis
   b. ECRL
   c. ECRB
   d. Anconeus

3. Little league elbow:
   a. Is caused by excessive batting practice
   b. Affects the lateral epicondylar epiphysis
   c. Usually occurs in 17 and 18 year olds
   d. Affects the medial epicondylar apophysis
1. How are fractures of the distal humerus classified?
Distal humeral fractures historically have been divided into extraarticular and intraarticular, with the following subdivisions: supracondylar, epicondylar, transcondylar, condylar, intercondylar, capitellar, and trochlear. In an attempt to develop a universal system, the AO/ASIF classification encompasses all periarticular distal humeral fractures.

<table>
<thead>
<tr>
<th>AO/ASIF Class</th>
<th>Description</th>
<th>Treatment</th>
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<tbody>
<tr>
<td>Type A: extraarticular fractures</td>
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<tr>
<td>A1</td>
<td>Avulsion fractures with no loss of column support to articular surface</td>
<td>Brief immobilization with early ROM</td>
</tr>
<tr>
<td>A2</td>
<td>Metaphyseal fractures with limited comminution</td>
<td>Nondisplaced: cast/brace &lt;3 weeks</td>
</tr>
<tr>
<td>A3</td>
<td>Significant metaphyseal comminution</td>
<td>ORIF with 4.5 DC plates</td>
</tr>
<tr>
<td>Type B: partial articular fractures</td>
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<td></td>
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<tr>
<td>B1</td>
<td>Lateral column disruption</td>
<td>ORIF with plates and/or screws</td>
</tr>
<tr>
<td>B2</td>
<td>Medial column disruption</td>
<td>ORIF with plates and/or screws</td>
</tr>
<tr>
<td>B3</td>
<td>Disruption of capitellum or trochlea</td>
<td>ORIF with or without primary fragment excision</td>
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<td>Type C: entire articular fractures</td>
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<tr>
<td>C1</td>
<td>Intercondylar split without comminution</td>
<td>ORIF</td>
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<td>C2</td>
<td>C1 with metaphyseal comminution</td>
<td>ORIF with or without bone graft</td>
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<td>C3</td>
<td>C2 with articular surface comminution</td>
<td>ORIF with or without excision and with or without bone graft</td>
</tr>
</tbody>
</table>

AO/ASIF, Arbeitsgemeinschaft fur Osteosynthesefragen/Association for the Study of Internal Fixation; ORIF, open reduction and internal fixation.

2. Define Malgaigne (supracondylar) fractures.
Most commonly seen in children, Malgaigne fractures occur above the olecranon fossa and are characterized by dissociation of the humeral diaphysis from the condyles of the distal humerus. Fracture
lines may extend distally to involve the articular surface. In adults intercondylar fractures are much more common and must be suspected.

3. **Describe two classification systems for Malgaigne fractures.**

The simpler system, based on the mechanism of injury, includes either extension-type or flexion-type supracondylar fractures. Falls onto an outstretched hand can produce the more common extension-type supracondylar fracture (80%), in which the fracture line passes from anterodistal to posteroproximal on lateral radiographs. Flexion-type supracondylar fractures result from force directed against the posterior aspect of a flexed elbow. The fracture line passes obliquely from anteroproximal to posterodistal on lateral radiographs. When displaced, the sharp proximal bone fragment often pierces the triceps and overlying skin, creating an open fracture.

A more comprehensive classification system, based on the presence of intercondylar extension and fracture comminution, is used more commonly in adults. Four types of supracondylar fractures are recognized:
- **Type I**—fractures without intercondylar extension
- **Type II**—fractures with intercondylar extension but without comminution
- **Type III**—fractures with intercondylar extension and supracondylar comminution
- **Type IV**—fractures with intercondylar extension and intercondylar comminution

4. **How are supracondylar fractures managed in adults?**

Anatomic reduction with stable fixation in adults is best achieved with plate-and-screw fixation (see table). External fixators are used when rapid stabilization of the elbow is required (eg, vascular disruption), when an open wound is associated with significant soft tissue injury or loss, or when plate-and-screw fixation is precluded by extensive bone loss or comminution. External fixator pins are placed laterally into the distal humerus and dorsally into the ulna. Skin incisions that are followed by blunt dissection to bone under direct visualization help prevent injury to the radial nerve. Ulnar pins are inserted with the forearm in 30 degrees of supination to permit forearm rotation.

<table>
<thead>
<tr>
<th>Treatment of Supracondylar Fractures in Adults</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FRACTURE TYPE</strong></td>
</tr>
<tr>
<td>Type I</td>
</tr>
<tr>
<td>OPERATIVE TREATMENT</td>
</tr>
<tr>
<td>Medial, lateral, or triceps, splitting with application of medial- and lateral-column, 3.5-mm reconstruction plates</td>
</tr>
<tr>
<td>Orthogonal configuration preferred over parallel placement</td>
</tr>
<tr>
<td>Medial-column plate applied to medial ridge; lateral-column plate placed on posterior column surface</td>
</tr>
<tr>
<td>Types II and III</td>
</tr>
<tr>
<td>Transolecranon exposure, followed by reduction and lag-screw fixation of intercondylar fracture</td>
</tr>
<tr>
<td>Reduce and stabilize supracondylar component with medial and lateral plates</td>
</tr>
<tr>
<td>Use bone graft in regions of supracondylar comminution (autogenous graft)</td>
</tr>
<tr>
<td>Type IV</td>
</tr>
<tr>
<td>Same as type III, but do not use lag-screw construct to fix intercondylar component because mediolateral condylar distance will decrease, creating joint incongruity</td>
</tr>
</tbody>
</table>

5. **Describe the classification and management of supracondylar fractures in children.**

The Garlant classification of supracondylar humerus fractures in children is based on the degree of displacement. Treatment ranges from percutaneous pin placement to formal open reduction and internal fixation (ORIF). Short-term immobilization in a bivalved cast is common to all treatments.
6. How are Granger (epicondylar) fractures classified and managed?
Lateral epicondylar fractures are extremely rare and usually are managed symptomatically with brief splinting, followed by early ROM exercises. The medial epicondyle—a traction apophysis for the wrist flexors and medial collateral ligament—is the last ossification center to fuse with the humeral metaphysis (age 15–20 years). Fractures are classified as undisplaced, minimally displaced, displaced >5 mm but proximal to the elbow joint, and entrapped (usually between the olecranon and trochlea). Acute fractures are differentiated from chronic tension stress injuries (little league elbow). Treatment of nonincarcerated fragments involves closed manipulation with short-term immobilization (10–14 days) with the forearm pronated and the elbow and wrist flexed. ORIF is indicated for incarcerated fractures that cause ulnar neuropathy. Chronic stress fractures are treated conservatively with brief immobilization and activity modification.

7. Which age group is most susceptible to transcondylar humerus fractures?
Transcondylar fractures usually are seen in older adult patients as a consequence of osteoporotic bone. The fracture line passes between the articular surface and the old epiphyseal line, traversing the coronoid and olecranon fossae. Treatment ranges from closed reduction and splinting to percutaneous pinning or ORIF. Excessive callus formation in the coronoid or olecranon fossa may result in loss of motion.

8. How are condylar fractures classified in adults?
Condylar fractures are rare in adults, representing <5% of all distal humerus fractures. Lateral condylar fractures, which include the capitellum and lateral epicondyly, are more common than medial condylar fractures. In Rockwood's *Handbook of Fractures*, Milch describes two types of fractures based on the presence of the lateral trochlear ridge: type I fractures leave the lateral trochlear ridge intact, whereas type II fractures, which are less stable, include the lateral trochlear ridge as part of the fracture fragment. Jupiter describes Milch fractures as high or low, based on extension of the fracture line into the supracondylar region. Low Jupiter fractures are equivalent to Milch type I fractures and high Jupiter fractures to Milch type II fractures. Preferred treatment in adults is ORIF with early ROM exercises.

9. How are condylar fractures classified in children?
In children, both Milch and Jacob systems are used. The Jacob system accounts for fracture displacement:
- Stage I fractures are undisplaced with an intact articular surface.
- Stage II fractures have moderate displacement.
- Stage III fractures are unstable elbow injuries with fragment displacement and rotation.
  Closed treatment of initially nondisplaced fractures in a long-arm cast is associated with a loss of reduction. Frequent serial radiographs are recommended to detect fracture displacement. ORIF is recommended for stage II fractures and for failed closed treatment.

10. Define intercondylar fractures.
Intercondylar fractures are the most common distal humerus fractures in adults. Usually they result from forces directed against the posterior aspect of a flexed elbow that cause the ulna to impact the trochlea. The resultant force splits the condyles, which are pulled apart by the flexor (medial) and extensor (lateral) muscle masses.

### Treatment of Supracondylar Fractures in Children

<table>
<thead>
<tr>
<th>CLASS</th>
<th>DESCRIPTION</th>
<th>EXTENSION TYPE</th>
<th>FLEXION TYPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>Undisplaced fracture</td>
<td>Immobilization at 90 degrees of flexion</td>
<td>Immobilization in near-extension</td>
</tr>
<tr>
<td>Type II</td>
<td>Displaced with one intact cortex</td>
<td>Closed reduction and percutaneous pin placement (two lateral)</td>
<td>Closed reduction and percutaneous pin placement (two lateral)</td>
</tr>
<tr>
<td>Type III</td>
<td>Complete displacement</td>
<td>Closed reduction and crossed-pin placement (two lateral, one medial); ORIF if unstable</td>
<td>Closed reduction and crossed-pin placement (two lateral, one medial); ORIF if unstable</td>
</tr>
</tbody>
</table>
11. Describe three classification systems for intercondylar fractures in adults.
The universal AO/ASIF classification of intercondylar fractures includes subtypes C1, C2, and C3. The Jupiter classification describes the shape and direction of the fracture as high T, low T, Y, H, medial lambda, or lateral lambda. Riseborough and Radin describe four types: type I (undisplaced), type II (slight displacement with no condylar fragment rotation in the frontal plane), type III (displacement of the condylar fragments with rotation), and type IV (type III fracture with severe comminution of the articular surface).

12. How are intercondylar fractures managed?
Treatment of intercondylar fractures must be individualized according to the patient’s age, medical status, bone quality, and fracture pattern. Older adult patients with osteoporotic bone and comminuted articular fractures may be managed with either closed treatment (cast/traction) or total elbow arthroplasty using a semiconstrained device. In general, ORIF with plates and screws is the preferred treatment for intercondylar fractures.

13. What are typical functional outcomes after an intraarticular distal humerus fracture?
Approximately 70% of patients have a good or excellent outcome; 25% have a fair outcome; and 5% have a poor outcome. Other typical outcomes are the following: mean flexion arc, \( \approx 112 \) degrees; pronation and supination, \( \approx 75 \) degrees each; grip strength, \( \approx 80\% \) of the contralateral side. About 75\% of patients return to their previous occupation.

14. Describe the three types of capitellar fractures.
Capitellar fractures are rare, representing <1% of all elbow fractures. Shear stress in the coronal plane may produce three types of fracture patterns:
- Type I fractures involve both osseous and cartilaginous portions of the capitellum, producing a Hahn-Steinthal fragment.
- Type II fractures of the capitellum shear off the articular cartilage with little underlying subchondral bone. This “uncapped condyle” is called a Kocher-Lorenz fragment.
- Type III fractures are markedly comminuted compression fractures of the capitellum.

15. How are capitellar fractures managed?
Treatment of nondisplaced fractures involves placing the elbow in maximal flexion and forearm pronation to allow for the radial head to act as an internal splint. However, extreme flexion in the face of soft tissue edema can cause vascular compromise and subsequent compartment syndrome. Immobilization at 90 degrees of flexion in a long-arm cast decreases the risk of compartment syndrome but is associated with loss of fracture reduction. Displaced fractures are treated with ORIF or fragment excision. Type I fractures are exposed through the anconeus surgical approach. Provisional fixation with Kirschner wires simplifies placement of small-fragment cancellous bone screws (directed posteriorly to anteriorly) or Herbert screws (placed anteriorly to posteriorly and buried below the articular surface). Excision of fracture fragments is indicated for most displaced type II fractures and for severely comminuted type III fractures.

Trochlear fractures are rare injuries produced by coronal shear forces directed against the trochlea by the coronoid process. Often associated with capitellar fractures, trochlear fractures are distinguished by a double-arc sign on lateral distal humerus radiographs. One arc represents the lateral ridge of the trochlea, and the other arc represents capitellar subchondral bone.

17. How are trochlear fractures managed?
Trochlear fractures are managed much like capitellar fractures. Nondisplaced fractures are managed by splinting and casting with early ROM exercises. Displaced fractures with significant osseous fragments are exposed through an extended lateral Kocher approach and stabilized via cancellous or Herbert screws. Severely comminuted or extensive articular injuries are managed via excision followed by early ROM exercises.

18. Describe the Colton classification of olecranon fractures.
Colton modified the original Schatzker classification system of olecranon fractures to include the following classes: undisplaced, displaced, oblique, and transverse fractures; comminuted fractures; and fracture-dislocations.
19. How are undisplaced olecranon fractures treated?
   Treatment of undisplaced fractures involves immobilization in a long-arm cast with the elbow in 45
to 90 degrees of flexion for approximately 3 weeks. Radiographic evaluation 5 to 7 days after cast
application is needed to rule out fracture displacement. Protected ROM in a hinged brace with 90 degrees
of maximal flexion is initiated at 3 weeks. Fracture union is not expected until 6 to 8 weeks after
injury. Joint stiffness and loss of motion are common, particularly in older adult patients who undergo
prolonged immobilization.

20. How are displaced olecranon fractures treated?
   Displaced fractures or fractures associated with a loss of active elbow extension are commonly treated
with tension band wiring, 3.5-mm DC/reconstruction plates, or excision of up to 50% of the olecranon
fragment and reattachment of the triceps. The coronoid must be intact.

21. What outcomes are associated with olecranon fractures?
   Decreased ROM is noted in 50% of patients. Deficits usually are minimal, and patients maintain a
functional ROM. Paresthesias, usually transient, are noted in 10% of patients. Nonunion occurs in
about 5% of olecranon fractures. Approximately 85% of patients have no complaints at long-term
follow-up; 50% will show arthritic changes compared with 11% in the uninjured extremity. Approximately
22% of plates used for fixation require removal, and up to 50% of tension band wires will need
to be removed.

22. Describe the Regan and Morrey classification of coronoid fractures.
   Three types of coronoid fractures, based on fragment size, were described by Regan and Morrey.
Type I is a tip avulsion fracture, type II involves <50% of the coronoid, and type III involves >50% of the
coronoid and is associated with recurrent elbow dislocations. Management of type I and type II
fractures is short-term immobilization in flexion, followed by early ROM exercises. Type III fractures
with associated elbow instability are best managed with ORIF.

23. Do type I fractures represent true avulsions of the coronoid?
   No. The brachialis inserts an average of 11 mm distal to the tip of the coronoid. Therefore, most type I
fractures represent shear fractures of the tip of the coronoid.

24. Summarize the mechanisms of injury and general management of radial head
   fractures.
   Radial head fractures result from indirect trauma (eg, fall onto an outstretched hand) when longitudinal
forces drive the radial head into the capitellum. Because of the mechanism of injury, concomitant
Essex-Lopresti injury to the distal radioulnar joint (DRUJ), capitellum, and medial collateral ligament
must be ruled out. A mechanical block to motion or elbow instability is an indication for operative
intervention. Aspiration of an elbow hemarthrosis with injection of lidocaine through a direct lateral

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<table>
<thead>
<tr>
<th>Treatment of Displaced Olecranon Fractures</th>
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<tbody>
<tr>
<td><strong>MODIFIED COLTON TYPE</strong></td>
</tr>
<tr>
<td>Avulsion fracture</td>
</tr>
<tr>
<td>Oblique fracture</td>
</tr>
<tr>
<td>Transverse fracture</td>
</tr>
<tr>
<td>Comminuted fracture</td>
</tr>
<tr>
<td>Coronoid intact</td>
</tr>
<tr>
<td>Coronoid fracture</td>
</tr>
<tr>
<td>Fracture dislocation</td>
</tr>
</tbody>
</table>
approach can decrease pain and allow for evaluation of passive ROM. ORIF is accomplished by placing cortical screws, Herbert screws, or miniplates in the anterolateral quadrant of the radial head (nonarticulating surface).

25. How are radial head fractures classified in adults?
Mason’s classification of radial head fractures in adults was modified by Johnston. Recommended treatment is listed in the following table.

<table>
<thead>
<tr>
<th>Fracture Type</th>
<th>Description</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>Undisplaced fracture involving &lt;25% of head</td>
<td>Splint and ROM as pain subsides</td>
</tr>
<tr>
<td>Type II</td>
<td>Marginal fracture with displacement of head</td>
<td>Excision or ORIF if angulation &gt;30°, more than one third of head is fractured, or displacement &gt;3 mm. Otherwise treat conservatively with splinting and early ROM.</td>
</tr>
<tr>
<td>Type III</td>
<td>Entire head comminuted</td>
<td>Early vs late radial head excision; repair DRUJ; repair/reconstruct MCL</td>
</tr>
<tr>
<td>Type IV</td>
<td>Associated elbow dislocation or Monteggia fracture</td>
<td>Reduce elbow; assess Monteggia or Essex-Lopresti injury; repair DRUJ; reconstruct MCL</td>
</tr>
</tbody>
</table>

ROM, range of motion; ORIF, open reduction and internal fixation; DRUJ, distal radioulnar joint; MCL, medial collateral ligament.

26. How are radial head fractures classified in children?
In children 90% of proximal radial fractures involve either the physis or the radial neck and are associated with fractures of the olecranon, coronoid, and medial epicondyle. The O’Brien classification is based on the degree of angulation of the radial neck. ORIF is indicated with angulation >60 degrees, failed closed reduction, complete displacement of the radial head, or >4 mm of radial head translocation. Radial head excision in children is associated with a high incidence of overgrowth and poor outcome.

<table>
<thead>
<tr>
<th>O’Brien Type</th>
<th>Angulation</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>&lt;30°</td>
<td>Simple immobilization</td>
</tr>
<tr>
<td>Type II</td>
<td>30–60°</td>
<td>Closed reduction and immobilization</td>
</tr>
<tr>
<td>Type III</td>
<td>&gt;60°</td>
<td>ORIF with Kirschner wires</td>
</tr>
</tbody>
</table>

27. How are elbow dislocations classified?
Elbow dislocations are classified based on the position of the ulna and radius relative to the distal humerus. Several types of elbow dislocations are recognized: posterior, posterolateral, postero medial, medial, lateral, anterior, and divergent.

28. What are the most and least common types of elbow dislocations?
Posterolateral elbow dislocations account for 11% to 28% of injuries to the elbow and are more common than other types of elbow dislocations. The incidence of posterolateral dislocations is highest in the 10- to 20-year-old age group and frequently is associated with sports-related injuries.

Divergent elbow dislocations are rare and consist of two types: anteroposterior and mediolateral (divergent).

29. Which fractures are commonly associated with elbow dislocations?
Medial or lateral epicondyle fractures (12%–34%) can become entrapped in the joint, causing a mechanical block to motion. They are seen more commonly in children. ORIF is occasionally necessary.

Coronoid process fractures (5%–10%) are seen most commonly with posterior dislocations. Fragments are graded as type I, II, or III as size increases. Type III fractures are associated with recurrent dislocations, and ORIF is recommended.

Radial head fractures involving the proximal intraarticular portion of the radius are managed nonoperatively in the absence of a bony mechanical block to motion. ORIF is indicated with concomitant radial head dislocation.
30. What complications are associated with elbow dislocations?
- Loss of motion (average of 10- to 15-degree loss of extension with simple dislocations)
- Loss of strength (15% average)
- Chronic instability
- Redislocation
- Posttraumatic arthritis
- Neurologic or vascular injury
- Compartment syndrome (Volkmann’s ischemic contracture)
- Ectopic calcification of the capsule or collateral ligaments (75% of cases)
- Heterotopic ossification of the capsule (5%), collateral ligaments, or brachialis

31. What are typical outcomes for triad injuries of the elbow (radial head fracture, coronoid fracture, and ligament instability)?
Typical outcomes include the following: flexion arc, $\approx 112$ degrees; pronation/supination arc, $\approx 136$ degrees; and complications resulting in reoperation, $\approx 20\%$. Approximately 78% of patients have a good to excellent result.

32. What are the types of coronoid fractures?
- Type I—tip avulsion fractures
- Type II—fractures of less than 50% of the coronoid height
- Type III—basal coronoid fractures
- The O’Driscoll classification recognizes subclassifications and anteromedial facet fractures because these often result in posterior medial instability.

33. What is the treatment of coronoid fractures?
Stable type I and type II fractures can be treated by early protected motion. If unstable, or associated with radial head fractures, these should undergo ORIF. Type III and anteromedial facet fractures usually undergo ORIF. Associated injuries should be treated as well, and, if ongoing instability is present, the use of an external fixator should be considered.

34. What surgical approach gives the best view of anteromedial fractures?
The flexor carpi ulnaris (FCU) splitting approach gives the most extensile exposure.

35. What other structure is often injured with anteromedial coronoid fractures?
Commonly tears of the lateral collateral ligament can allow for varus laxity and increased instability.

BIBLIOGRAPHY
CHAPTER 48 QUESTIONS

1. Anteromedial coronoid fractures:
   a. May result in posterolateral instability
   b. **May result in posteromedial instability**
   c. Are usually stable fractures
   d. Require surgery less often than type I fractures

2. Radial head fractures that require ORIF include:
   a. Type I fractures
   b. All type II fractures
   c. **Fractures blocking full ROM**
   d. Severely comminuted 5-part fractures

3. What is the most common elbow dislocation?
   a. Medial
   b. Lateral
   c. Posterior
   d. **Posterior lateral**
1. What anatomic structures could compress the ulnar nerve at the elbow, and why would elbow flexion increase pressure on the nerve?

The nerve can be compromised within the ulnar groove (proximal cubital tunnel) by a fibrous band often referred to as the cubital tunnel retinaculum (CTR). The CTR attaches to the medial epicondyle and olecranon and passes over the ulnar nerve.

As the elbow is flexed, the CTR becomes taut and forces the nerve against the bone, and with the elbow extended, the CTR becomes lax. Distal to the CTR, the ulnar nerves pass beneath an aponeurosis between the two heads of the flexor carpi ulnaris, another potential site for compression.

2. Dislocation or subluxation of the ulnar nerve at the elbow is due to the absence of what anatomic structure?

The ulnar nerve can slip out of the ulnar groove (dislocate or sublux) when the elbow moves from extension to flexion in individuals lacking a cubital tunnel retinaculum (CTR). Repeated dislocation of the ulnar nerve at the elbow could subsequently result in neuritis.

3. What are the sensitivity or provocation tests for cubital tunnel syndrome?

Test results for cubital tunnel syndrome include the following: Tinel’s sign = 0.70, elbow flexion = 0.32, pressure provocation = 0.55, pressure-flexion test = 0.91.

4. Focal demyelination of motor and sensory axons of the ulnar nerve at the elbow could result in what finding during an ulnar motor nerve study?

Demyelination could result in reduced motor nerve conduction velocity (NCV) from the above- to below-elbow segment, while the NCV from the below-elbow to the wrist and axilla to the above-elbow segments would be normal.

5. What is a Martin-Gruber anastomosis? Explain its clinical significance.

A Martin-Gruber anastomosis is an anastomosis from the median nerve to the ulnar nerve in the forearm before the median nerve crosses the wrist. The Martin-Gruber anastomosis may confuse understanding of symptoms in patients with compressions of either the ulnar or the median nerve. It is found in approximately 8% to 54% of the population, according to various studies. Leibovic and Hastings described four types of this anastomosis:

- Type I (60%)—motor branches sent from the median to the ulnar nerve to innervate “median” muscles;
- Type II (35%)—motor branches sent from the median to the ulnar nerve to innervate “ulnar” muscles;
- Type III (3%)—motor fibers sent from the ulnar to the median nerve to innervate “median” muscles;
- Type IV (1%)—motor fibers sent from the ulnar to the median nerve to innervate “ulnar” muscles.

6. What are the clinical differences (sensory changes and muscle weakness) that would help distinguish a motor and sensory axonal loss lesion of the ulnar nerve at the elbow from a lower trunk of the brachial plexus?

Ulnar nerve lesion at the elbow:
- Decreased sensation of the medial hand and digits 4 and 5
- Weakness in the flexor carpi ulnaris, ulnar portion of the flexor digitorum profundus, abductor digiti minimi, and first dorsal interosseus

Lower trunk lesion:
- Decreased sensation of the medial arm (medial brachial cutaneous), medial forearm (medial antebrachial cutaneous), medial hand (ulnar nerve), and digits 4 and 5 (ulnar nerve)

*Deceased.
• Weakness in ulnar innervated muscles: flexor carpi ulnaris, ulnar portion of the flexor digitorum profundus, abductor digiti minimi, and first dorsal interosseous
• Weakness in radial innervated muscles supplied by the C8 and T1 anterior rami, such as extensor indicis, extensor pollicis longus, and extensor digitorum longus
• Weakness in median innervated muscles supplied by the C8 and T1 anterior rami, such as flexor pollicis longus, pronator quadratus, abductor pollicis brevis, and opponens pollicis

7. At what site above the elbow may the median nerve be compressed?
The ligament of Struthers runs from a bony projection toward the medial epicondyle. The median nerve, along with the ulnar nerve in some instances, passes below this bony projection. The ligament may be a site of compression of both nerves, but more typically only the median nerve is involved.

8. Define radial tunnel syndrome.
In radial tunnel syndrome, the deep branch of the radial nerve is compressed in the forearm, causing pain and occasionally weakness in the most distally supplied muscles of the radial nerve. There is no sensory loss because the compression occurs below the level of the superficial radial nerve. Signs and symptoms include deep, aching pain in the upper dorsal forearm without tenderness over the lateral epicondyle or radial head. The tenderness is located just below the radial head in the groove formed between the brachioradialis muscle and the extensor carpi radialis. Patients also may have a positive long-finger sign—when the patient extends the wrist and fingers and pressure is applied to the third digit to resist extension, the extensor fascia tightens in the area of the radial tunnel, increasing symptoms. Weakness may be found in the extensors of the thumb, the abductor pollicis longus, and the extensor indicis.

9. What five tests are commonly used for the diagnosis of radial tunnel syndrome?
1. Compression over the radial tunnel
2. Long-finger test
3. Wrist extension
4. Resisted supination
5. Cuff test—a blood pressure cuff is applied above the source of pain in a peripheral nerve distribution and, by decreasing vascularity to the compression site, reproduces the pain syndrome

10. Which test for radial tunnel syndrome has the highest sensitivity?
The differential motor latency test conducted in pronation and supination has the highest sensitivity for radial tunnel syndrome, with a >0.3-ms difference between positions being sensitive for the detection of radial tunnel syndrome. Clinically, palpation for pain over the radial tunnel area has the greatest sensitivity, followed by resisted supination and the long-finger test.

11. What are the possible sites of compression in radial tunnel syndrome?
The fibrous edge of the proximal portion of the supinator muscle (arcade of Frohse), tendinous origins of the extensor carpi radialis brevis, the distal edge of the supinator, and the vascular leash of Henry are all possible compression sites.

12. The pronator teres is typically innervated by a motor branch from the median nerve before the nerve passes through the two heads of this muscle. Why is this anatomic relationship important when performing a manual muscle test (MMT) during clinical testing for a suspected pronator teres syndrome?
The pronator teres (PT) would not demonstrate any weakness during an MMT, while compression of the nerve within the muscle could result in weakness during an MMT by all or some of the muscles supplied by the median nerve distal to the PT, such as flexor carpi radialis, flexor digitorum superficialis (sublimis), flexor pollicis longus, flexor digitorum profundus (digits 2 and 3), pronator quadratus, abductor pollicis brevis, and opponens pollicis.

13. Could a compromise of the anterior interosseous nerve (AIN) result in wrist pain and numbness and tingling of the thumb?
The AIN (a branch of the median nerve) provides sensory fibers to the capsule of the wrist but not to the skin of the hand. Therefore, symptoms could include wrist pain but not numbness and tingling of the thumb.

14. How can pronator teres syndrome be clinically differentiated from anterior interosseous syndrome?
In patients with pronator teres syndrome, forearm pain is increased by resisted pronation. Patients also may have pain on palpation in the pronator teres area; weakness in muscles, supplied by the median
nerve; feeling of numbness in the hand, especially the second and third digits; and, occasionally, complaints of easy fatigue with the use of the hand muscles.

Patients with anterior interosseous syndrome show weakness only in the muscles supplied by the anterior interosseous nerve. Some clinicians suggest testing the pronator quadratus with the elbow flexed; resisting pronation puts the pronator teres at a disadvantage and demonstrates weakness in the pronator quadratus. Patients complain of aching in the forearm but no sensory changes. They are unable to make an OK sign by pinching the thumb and index finger together because of weakness of the flexor pollicis longus.

15. What are the common signs, symptoms, and EMG/NSC changes noted in carpal tunnel syndrome, anterior interosseous syndrome, and pronator teres syndrome?

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<th>Symptoms</th>
<th>Signs</th>
<th>EMG/NCS Findings</th>
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<tbody>
<tr>
<td>Carpal tunnel syndrome</td>
<td>Numbness and tingling in digits 1–3½</td>
<td>Decreased sensation of digits 1–3½</td>
<td>Distal latency prolongation of median nerve (motor and sensory) across wrist</td>
</tr>
<tr>
<td></td>
<td>Weak grip</td>
<td>Weakness of thenar muscles</td>
<td>Possible denervation of thenar muscles and lateral lumbricals</td>
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<tr>
<td></td>
<td>Hand weakness</td>
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<tr>
<td></td>
<td>Wrist and hand pain (especially at night)</td>
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<td>Weakness of pinch</td>
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<tr>
<td></td>
<td>Denies numbness and tingling</td>
<td>Normal sensation</td>
<td>Normal median motor and sensory nerve, distal latencies at wrist, and normal forearm conduction velocity</td>
</tr>
<tr>
<td>Anterior interosseous syndrome</td>
<td>Numbness and tingling in digits 1–3½</td>
<td>Weakness of FPL, PQ, and FDP (lateral)</td>
<td>Denervation of FPL, PQ, and FDP (lateral)</td>
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<tr>
<td></td>
<td>Weakness of pinch</td>
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<tr>
<td></td>
<td>Denies numbness and tingling</td>
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<td></td>
<td>May have anterior forearm pain</td>
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<tr>
<td>Pronator teres syndrome</td>
<td>Numbness and tingling in digits 1–3½</td>
<td>Weakness of thenar muscles</td>
<td>Decreased forearm conduction velocity (may have conduction block across elbow)</td>
</tr>
<tr>
<td></td>
<td>Deep anterior forearm pain</td>
<td>Questionable weakness of FPL, PQ, and FDP (lateral)</td>
<td>Possible median motor and sensory distal latency; prolongation at wrist</td>
</tr>
<tr>
<td></td>
<td>Increased symptoms with forceful pronation activities (eg, twisting off lids)</td>
<td>Decreased sensation of digits 1–3½</td>
<td>May have denervation in thenar muscles, lateral lumbricals, FPL, PQ, and FDP (lateral)</td>
</tr>
<tr>
<td></td>
<td>Hand weakness</td>
<td></td>
<td>Pronator teres is spared</td>
</tr>
</tbody>
</table>

16. At what sites may the superficial radial nerve be compressed?

Occasionally, patients have symptoms of sensory loss in the distribution of the superficial radial nerve without other evident problems with the radial nerve. Compression may occur when the superficial radial nerve emerges from beneath the brachioradialis muscle and enters the fascia, investing the extensor muscles of the forearm. Some dog handlers loop the leash of a dog that they are training over their forearm and hold the leash below the loop. When the dog pulls on the leash, the loop tightens around the distal aspect of the forearm, compressing the radial nerve under the loop (dog handler’s syndrome). Other names for this syndrome are cheiralgia paresthetica and handcuff palsy.
17. What is Saturday night palsy?
Saturday night palsy is a compression injury in the portion of the radial nerve between the radiospiral groove and lateral intermuscular septum. Typical causes are periods of relatively severe compression of the radial nerve in the region of the posterior aspect of the humerus. The typical patient is an inebriated person on Saturday evening who loses consciousness with the arm slung over the back of a chair—hence the name Saturday night palsy.

18. Describe the symptoms and signs of Saturday night palsy.
Patients typically have weakness of the triceps and loss of function of the wrist extensors as well as the finger and thumb extensors. Sensory loss varies, depending on the level of the lesion. If the compression site is above the branches for the superficial radial nerve, the patient has sensory loss in the radial nerve distribution. If the compression is below the site of the superficial radial nerve branches and above the site for innervation to the brachioradialis and wrist extensors, the patient reports loss of wrist extension but no loss of sensation. Saturday night palsy is considered a high radial nerve palsy because it occurs in the upper arm, not in the forearm. This distinction is important in differentiating Saturday night palsy from radial nerve compressions of the forearm described previously.

19. Can the radial nerve be compressed by fibrous bands at the level of the radial head?
Yes. The most proximal site of compression of the deep branch of the radial nerve is the point where it crosses close to the radial head. Compressions of the deep branch of the radial nerve may be confused with tennis elbow or lateral epicondylitis. The surgical literature reports no differentiation in terms of clinical symptoms. Neural tension testing (provocative tests for neural tension) is a relatively recent concept. No research relates compression of the deep branch of the radial nerve at the level of the radial head to a positive test for neural tension.

20. Discuss the relative frequency of the ulnar and median nerve entrapments of the elbow and forearm.
A compromise of the ulnar nerve at or near the elbow is the most common entrapment encountered when examining the elbow and forearm. Entrapment of the ulnar nerve proximal to the elbow at either the medial intramuscular septum or ligament of Struthers is rare. Entrapment of the median nerve at either the ligament of Struthers or within the pronator teres is infrequent. Although entrapment of the anterior intersosseous nerve by fibrous bands of either the flexor digitorum superficialis (sublimis) or flexor digitorum profundus is relatively rare, it may be distinguished more easily than the pronator teres syndrome and, therefore, may be diagnosed more frequently with certainty.

21. What are the surgical options and outcomes for cubital tunnel syndrome?
Surgical options include decompression alone, subcutaneous transposition, intramuscular transposition, submuscular transposition, medial epicondylectomy, and arthroscopic epicondylectomy. Positive results vary from 70% to 95% with few differences between procedures other than minor benefits from submuscular transfer with advanced-stage ulnar nerve compression.

22. How frequently is there loss of ulnar nerve function after a total elbow joint arthroplasty?
The incidence of ulnar nerve complications after a total joint arthroplasty varies considerably. Some reports carry no information about ulnar neuropathy after this procedure. Others have reported an incidence of up to 26%. Most of the literature seems to indicate a 6% to 10% complication rate of ulnar nerve problems after a total joint arthroplasty, with most of these resolving over time. Many patients have subclinical nerve changes before their surgery secondary to arthritis, synovitis, and swelling.

Bibliography


1. Describe the anatomy of the carpal tunnel.

**TRANSVERSE CARPAL LIGAMENT**
- Extends from proximal carpal row (pisiform and scaphoid tubercle) to distal row (hook of hamate and trapezial ridge) through proximal aspect of second through fifth metacarpals.
- Width = 22 mm
- Length = 26 mm
- Thickness:
  1. Proximal = 0.6 mm
  2. Midportion = 1.6 mm
  3. Distal = 0.6 mm

**CARPAL CANAL**
- Depth
  1. Entrance to canal = 12 mm
  2. Midportion = 10 mm
  3. Distal end = 13 mm
- Average cross-sectional area = 17 mm²
- Smallest cross-sectional area = 16 mm² (at distal carpal row)
- Volume = 5.84 mL
- Carpal tunnel release increases canal volume by 25% and changes the shape from oval to round. This is associated with a concomitant change in the shape of Guyon’s canal from triangular to a vertical oval.

2. What is the average pressure (in mm Hg) in the carpal tunnel at different wrist positions?

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>With Carpal Tunnel Syndrome</th>
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</thead>
<tbody>
<tr>
<td>Neutral position</td>
<td>2</td>
<td>32</td>
</tr>
<tr>
<td>Full flexion</td>
<td>31</td>
<td>94</td>
</tr>
<tr>
<td>Full extension</td>
<td>35</td>
<td>110</td>
</tr>
<tr>
<td>After carpal tunnel release</td>
<td>5</td>
<td>—</td>
</tr>
</tbody>
</table>

3. Name the 10 structures that pass through the carpal tunnel.

The eight tendons of the flexor digitorum superficialis (FDS) and flexor digitorum profundus (FDP) pass through the carpal tunnel. The FDS tendons to the long and ring fingers are superficial to the FDS tendons to the index and little fingers. The flexor pollicis longus and, of course, the median nerve also pass through the tunnel. The flexor carpi radialis tendon is not considered to be an occupant of the carpal tunnel because it passes through its own compartment.
4. Describe the relationship of the contents of Guyon’s canal.
   From radial to ulnar, the ulnar artery and ulnar nerve pass through the canal. The flexor carpi ulnaris tendon is most ulnar but lies outside Guyon’s canal.

5. What is the relationship between the digital nerves and arteries?
   The common digital arteries usually bifurcate 0.5 to 1.0 cm distally to the bifurcation of the common digital nerves in the palm. They are contiguous in the distal half of the fingers. The arteries are deep to the nerves. In general, if the artery is lacerated, so is the nerve.

6. Where are the vincula located and what is their purpose?
   The vincula are located on the dorsal half of the flexor superficialis and profundus tendons. They are longitudinal fibers visible when the FDS or FDP are lifted volarly during surgery. The vincula are partially responsible for the nutrition of the flexor tendons.
7. Describe the anatomy of the flexor sheath.

The pulleys are called annular (A) and cruciate (C), names derived from their respective configurations. They prevent the tendons from bowstringing when the fingers are flexed as well as improving the biomechanics of finger flexion.

- A1 pulley—on the volar plate of the metacarpal phalangeal joint
- A2 pulley—over the proximal portion of the proximal phalanx
- C1 pulley—over the midportion of the proximal phalanx
- A3 pulley—on the volar plate of the proximal interphalangeal (PIP) joint
- C2 pulley—over the proximal middle phalanx
- A4 pulley—at the midportion of the middle phalanx
- C3 pulley—on the distal aspect of the middle phalanx
- A5 pulley—attached to the volar plate of the distal interphalangeal (DIP) joint

The odd-numbered annular pulleys (A1, A3, A5) are located at the joints, and even-numbered pulleys (A2 and A4) are located over bone. After the two initial annular pulleys, the cruciate and annular pulleys alternate. The A2 and A4 pulleys are considered the most crucial.

8. What are the pulleys of the thumb, and which thumb pulley is the most responsible for prevention of bowstringing of the FPL?

1. A1—annular pulley at the level of the volar plate at the thumb MP joint
2. A2—annular pulley at the level of the volar plate at the thumb IP joint
3. Oblique pulley—located between the annular pulleys at the diaphysis of the proximal phalanx

The oblique pulley is most responsible for preventing bowstringing of the FPL.

9. Compare the flexor digitorum superficialis (FDS) and flexor digitorum profundus (FDP) of the fingers.

Both the FDS and the FDP are extrinsic muscles whose primary function is to flex the interphalangeal joints. The FDS flexes the PIP joint and the FDP flexes the DIP joint. The FDS has separate muscle bellies to each of the digits, allowing them to function independently of one another. The FDP has...
a common muscle belly to the long, ring, and small fingers, preventing independent flexion. (The FDS is absent to the SF in approximately 30% of the population and prevents isolated PIP flexion in the SF.)

Testing of the FDP is done by blocking the PIP in extension while bending the DIP; testing of the FDS of the MF, RF, and SF is performed by actively flexing one digit while maintaining the adjacent digits in extension.

10. **What is the total excursion of normal flexor and extensor tendons?**
   - EDC: about 50 mm
   - FDP: about 70 mm

11. **Where is Camper’s chiasm and what is its function?**
    Just distal to the PIP joint, the FDS tendon divides, allowing the deeper FDP to pass through to become the more superficial tendon. The two slips of the FDS wrap obliquely around the FDP and rejoin dorsally via the Camper’s chiasm. The Camper’s chiasm is not just one specific point but the “bed” for the FDP to glide upon, limiting hyperextension and increasing stability of the PIP joint.

12. **What are the anatomic landmarks for the zones of flexor tendon injury in the hand?**
    - Zone 1—distal to the insertion of the FDS
    - Zone 2—distal palmar crease, formerly called “no man’s land”
    - Zone 3—distal to the distal edge of the transverse carpal ligament
    - Zone 4—carpal tunnel
    - Zone 5—distal portion of the forearm

13. **What are the zones of injury of the extensor tendon?**
    1. DIP
    2. Middle phalanx
    3. PIP
    4. Proximal phalanx
    5. Metacarpophalangeal (MCP)
    6. Metacarpal
    7. Dorsal retinaculum
    8. Distal forearm

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**Zones of flexor tendon injury.**
14. Name the tendons in the six dorsal compartments of the hand.

The first compartment consists of the abductor pollicis longus and extensor pollicis brevis. The extensor carpi radialis brevis and extensor carpi radialis longus are in the second compartment. In the third compartment the extensor pollicis longus passes radially to Lister’s tubercle to insert on the thumb. The fourth compartment contains the four tendons of the extensor digitorum and the “fellow traveler” extensor indicis proprius. In the fifth compartment is the tendon for the fifth digit—the extensor digiti minimi. Finally, the extensor carpi ulnaris passes through the sixth compartment.

15. Describe the tension in the collateral ligaments in relation to joint position.

At the metacarpophalangeal joint, the ulnar and radial proper collateral ligaments are taut in flexion because they are stretched over the condyles of the metacarpal head, resisting radial and ulnar deviation. The MCP proper collateral ligaments are relaxed with MP extension.

At the interphalangeal joint, collateral ligaments and volar plate are stretched when held in extension because of the shape of the phalangeal head. The IP collateral ligaments are relaxed with flexion.

16. Describe the structure of the carpal ligaments.

The majority of the carpal ligaments are intracapsular. They can be categorized into two groups: intrinsic and extrinsic. The intrinsic ligaments begin and end on the carpal bones, and the extrinsic ligaments connect the radius and ulna to the carpus. The names of the carpal ligaments describe their origins and insertions. The volar ligaments are believed to be the strongest and most important. The major extrinsic ligaments are the radioscaphoid, radiocapitate, long radiolunate, ulnocapitate, short radiolunate, ulnotriquetral, and ulnolunate. Among the major intrinsic ligaments are the scapholunate interosseous, lunotriquetral, triquetral-hamate-capitate complex, and the numerous distal carpal row interosseous ligaments.
17. What is the significance of the palmaris longus tendon?
The palmaris longus originates from the medial epicondyle and inserts distally on the palmaris fascia. It is a tendon commonly used as a graft source for hand and upper extremity surgeries, such as in thumb CMC arthroplasty, flexor and extensor tendon reconstruction, and triceps repair. It can be harvested without deformity or decrease of strength to the wrist or hand. It is present in approximately 85% of the population. However, this number varies some among different ethnic groups. It can be elicited by flexing the wrist and opposing the pads of the small finger and thumb. If it is present, it will be visible midline, just proximal to the volar wrist crease.

18. Where is the most immobile part of the wrist and hand located?
The “fixed unit” of the hand is considered the distal row of carpal bones and the 2nd and 3rd metacarpal joints. This is the base of support for the more mobile structures of the hand, specifically, the 1st, 4th, and 5th digits.

19. What is the triangular fibrocartilage complex (TFCC)?
The triangular fibrocartilage complex (TFCC) is a structure of ligaments and cartilage located on the ulnar wrist. It includes the dorsal and volar radioulnar ligament, the central articular disc, the meniscus homologue, the UCL and the ECU subsheath, and the origin of the ulnolunate and ulnotriquetral ligaments. The TFCC is responsible for stabilizing the joint, providing cushioning with movement and impact, and promoting smoothness of motion, especially during grip and forearm rotation.

20. What is the normal range of motion of the wrist?
- Flexion—0 to 80 degrees (60% from midcarpal joint, 40% from radiocarpal joint)
- Extension—0 to 70 degrees (33% from midcarpal joint, 66% from radiocarpal joint)
Flexion is dominated by midcarpal joint motion and extension by radiocarpal motion.
- Radial deviation—0 to 20 degrees (scaphoid volar flexion, lunate volar flexion)
- Ulnar deviation—0 to 30 degrees (scaphoid dorsiflexion, lunate dorsiflexion)
- Total radial/ulnar deviation arc—50 degrees (60% from midcarpal joint, 40% from radiocarpal joint)
- Pronation and supination—0 to 80 degrees, 160 degrees total arc of motion

21. What is the functional range of motion of the wrist?
Opinions vary; one study suggests 10 degrees of flexion, 30 degrees of extension, 10 degrees of radial deviation, and 15 degrees of ulnar deviation. Others suggest 40 degrees of flexion, 40 degrees of extension, and a combined 40-degree radial/ulnar deviation arc.

22. Describe the kinematics of the wrist.
The distal carpal row interosseous ligaments are multiple and strong. Hence, the distal row moves as a unit. Excluding the pisiform, the proximal row has no tendinous attachments. Thus the distal row moves first, and the proximal row follows its lead. With wrist flexion the distal row flexes and the ulnar deviates. Extension causes the distal row to extend and deviate radially. With radial deviation, the distal row deviates radially, extends, and supinates. The exact opposite occurs with ulnar deviation. The proximal carpal row extends with ulnar deviation and flexes with radial deviation.
23. Describe the motion of the fingers and thumb.
- MCP joint of the thumb—0 to 90 degrees of flexion-extension with an average of 55 degrees
- IP joint of the thumb—85 to 90 degrees of flexion with slight pronation; usually allows 0 to 20 degrees of hyperextension
- Finger MCP joints—30 to 45 degrees of extension, 85 to 100 degrees of flexion, and 20 to 60 degrees of abduction/adduction
- PIP joints—no extension and 100 to 115 degrees of flexion
- DIP joints—10 to 20 degrees of extension and 80 to 90 degrees of flexion

24. Describe the blood supply of the scaphoid.
The scaphoid receives its blood supply through its ligaments. The main arterial supply enters around the midpoint (waist) of the scaphoid; additional vessels enter distally. The more proximal portion of the scaphoid receives nutrients in a retrograde fashion. This precarious situation can be disrupted by fractures and explains the relatively high incidence of avascular necrosis.

25. Describe the normal anatomy of the distal radius.
The distal radius tilts in two planes. It normally has approximately 11 degrees of volar tilt and 23 degrees of ulnar inclination. It consists of two facets—the lunate and scaphoid fossae.

26. What position of the wrist allows maximal grip strength?
Maximal power grip is achieved with 35 degrees of extension and 7 degrees of ulnar deviation. In full flexion only 25% of grip strength can be achieved.

27. Describe the force transmission across the radiocarpal joint with axial wrist loading.
With the wrist in neutral position during axial carpal loading:
- 80% across distal radius (60% through scaphoid facet, 40% lunate facet)
- 20% across distal ulna

28. Describe the musculature of the hand.

<table>
<thead>
<tr>
<th>Musculature of the Hand</th>
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<tbody>
<tr>
<td><strong>MUSCLE</strong></td>
</tr>
<tr>
<td>Abductor pollicis brevis</td>
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<tr>
<td>Flexor pollicis brevis</td>
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<tr>
<td></td>
</tr>
<tr>
<td>Opponens pollicis</td>
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<tr>
<td>Adductor pollicis</td>
</tr>
<tr>
<td>Transverse head: shaft of 3rd MC</td>
</tr>
<tr>
<td>Palmaris brevis</td>
</tr>
<tr>
<td>Abductor digiti minimi</td>
</tr>
<tr>
<td>Flexor digiti minimi</td>
</tr>
<tr>
<td>Opponens digiti minimi</td>
</tr>
</tbody>
</table>
29. What innervates each of the extrinsic wrist and digit muscles?

The radial nerve innervates all nine extrinsic extensor muscles of the wrist and digits.

The median nerve innervates all of the extrinsic flexor muscles, except for the FCU and the FDP to the ring and small fingers, which are innervated by the ulnar nerve.

30. What nerve innervates the intrinsic muscles?

The median nerve innervates the LOAF muscles: 2 radial lumbricals, opponens pollicis, abductor pollicis brevis, and the superficial head of the flexor pollicis brevis.

The ulnar nerve innervates the adductor pollicis, the deep head of the flexor pollicis brevis, the abductor digitii minimi, flexor digitii minimi, opponens digitii minimi, and all interossei.

31. What are the two possible communications (anastomosis or interconnection) between the median and ulnar nerves?

- Martin-Gruber anastomosis: In this anomaly nerve fibers that were destined to be with the ulnar nerve when the median nerve was formed (by contributions from the lateral and medial cords of the brachial plexus) have stayed with the median nerve until the proximal forearm, where they finally join the ulnar nerve. This nerve interconnection explains why some patients with high ulnar nerve lesion have retained function in an area that typically is innervated by the ulnar nerve.

- Riche-Cannieu interconnection: This less common anomaly occurs with a pattern similar to the Martin-Gruber anastomosis. In this case, however, motor nerves to intrinsic muscles have stayed with the median nerve rather than the ulnar nerve at the level of the brachial plexus and rejoin the ulnar nerve in the hand.

32. Describe the bony anatomy of the metacarpophalangeal joint.

It is a condyloid (triaxial) joint with a trapezoidal-shaped metacarpal head on axial cross section. This results in a more narrow width dorsally. In flexion, the wider head has greater bony contact with the proximal phalanx, resulting in greater stability. The metacarpal head is a near perfect circle in the sagittal plane. For 10% of patients, the thumb metacarpal head is flat, in which case the joint acts more like a hinge with mobility relying upon capsular laxity.
33. **Explain the cam effect of the metacarpophalangeal joint.**

The cam effect at this joint is due to the eccentric origin of the collateral ligaments, dorsal to the axis of rotation. Additionally, prominences are found volarly over which the collateral ligaments pass, increasing the tension from 60 to 90 degrees of flexion; therefore, the collateral ligaments tighten in flexion, giving stability to the MCP joint when gripping. In extension the collateral ligaments relax, allowing abduction-adduction motion, thus improving fine motor movements.

34. **Describe the anatomy of the proximal interphalangeal joint (PIP).**

It is a single-axis hinge joint with a bicondylar proximal phalanx head and intercondylar groove that articulates with the saddle-shaped median ridge of the middle phalanx base. This provides significant lateral stability. The PIP joint does not have a cam effect; therefore, the collateral ligaments have equal tension in flexion and extension.

35. **What is the clinical and anatomic significance of the thumb interphalangeal joint’s active extension versus hyperextension?**

All thenar muscles (except opponens pollicis) insert at least partially into the dorsal apparatus of the thumb and, therefore, act to extend the IP joint. They cannot, however, hyperextend the IP joint, which requires intact function of the extensor pollicis longus (EPL) muscle and tendon. Thus a patient with either radial nerve palsy or EPL tendon laceration may still exhibit thumb IP active extension but not hyperextension.

36. **Where is the extensor indicis proprius (EIP) tendon in relation to the extensor digitorum communis (EDC) tendon?**

The extensor indicis proprius (EIP) tendon almost always is ulnar to the extensor digitorum communis (EDC) tendon.

37. **Describe the insertion of the dorsal interossei (DI) tendons.**

The superficial belly inserts through the medial tendon to the lateral tubercle at the base of the proximal phalanx and acts as an abductor. The deep belly continues as the lateral tendon to the transverse fibers of the dorsal apparatus, acting to flex the MCPs and extend the IPs. The first DI tendon inserts 100% medially, the second DI tendon inserts 60% medially and 40% laterally, the third DI tendon inserts 6% medially and 94% laterally, and the fourth DI tendon inserts 40% medially and 60% laterally.

38. **What is the extensor mechanism of the digit?**

Finger extension involves a complex coordination of the extrinsic extensor muscles, intrinsic muscles of the hand, and the intricate retinacular structure responsible for organizing and transmitting tension to create movement. Other names referring to this same structure include the dorsal apparatus, extensor apparatus, extensor expansion, dorsal digital expansion, dorsal aponeurosis, and aponeurotic sleeve.

39. **Define the following components of the extensor mechanism:**

**Sagittal Bands:** At the level of the MP joint, the sagittal bands arise from the volar plate and span both sides of the MP joint to insert dorsally on the extensor tendon. The sagittal bands and volar plate completely surround the MP joint and assist the juncturae tendinum with anchoring and maintaining the extensor tendon over the MP joint. It also assists with extension of the PIP joint by transmitting the pull of the extrinsic tendon.

**Lateral Bands:** The lateral bands are composed of tendons from the intrinsic interossei and lumbrical muscles. The lateral bands are located on each side of the proximal phalanx and travel distally toward the PIP joint, contributing and receiving fibers along the way. At the distal end of the proximal phalanx, the lateral bands join with the lateral slips of the extrinsic extensor and become the conjoined lateral bands. The lateral bands transmit tension force from the EDC, lumbricals, and interossei to assist with MP flexion and IP joint extension.

**Central Slip:** The central slip is formed by the central division of the extrinsic extensor tendon in combination with medial fibers from the intrinsic tendons. The CS continues distally to insert onto the base of the middle phalanx, helping to initiate PIP extension.

**Conjoined Tendons:** The lateral slips of the extrinsic extensor merge with the lateral bands of the intrinsic muscles to form the conjoined lateral bands at the distal end of the proximal phalanx. The radial and ulnar conjoined tendons move distally and come together at the distal portion of the middle phalanx to form the terminal tendon, which goes on to insert on the base of the distal phalanx, allowing for extension of the DIP joint.
Oblique Retinacular Ligament (Landsmeer Retinacular Ligament): The ORL originates volarly on the flexor pulley at the PIP joint and inserts dorsally onto the lateral terminal tendon. This ligament links motion of the PIP and DIP.

The ORL is taut with PIP extension, creating passive tension that extends the DIP. Tightness of the ORL may limit DIP flexion.

Transverse Retinacular Ligament: The transverse retinacular ligament originates on the volar plate of the PIP joint and inserts distally onto the lateral edge of the conjoined tendons. During PIP flexion the TRL positions the lateral bands volarly and, during PIP extension, prevents dorsal displacement of the lateral bands.

Triangular Ligament: Located at the dorsum of the middle phalanx where the terminal tendons meet. It prevents volar subluxation of the conjoined lateral bands during flexion of the IP joint.

40. What is the function of the retinacular ligaments?
The transverse retinacular ligaments attach from the flexor sheath to the conjoined lateral bands, thus stabilizing the lateral bands. Lateral displacement of the bands may lead to a boutonniere deformity, whereas contracture and dorsal displacement may lead to swan neck deformity. The oblique retinacular ligament runs from the proximal volar aspect of the PIP to the dorsal terminal extensor tendon. This ligament links the movement of the DIP and PIP joints. PIP flexion allows for DIP flexion, whereas PIP extension promotes DIP extension.

41. Describe the cross section of the median and ulnar nerves.
- **Median**—6% motor, 94% sensory; motor branch may arise radial (60%), central (22%), or volar/radial (18%)
- **Ulnar**—45% motor, 55% sensory; motor branch arises dorsal ulnar

42. Describe the anatomy of the superficial branch of the radial nerve (SBRN).
The superficial branch of the radial nerve (SBRN) exits between the brachioradialis (BR) and the extensor carpi radialis longus (ECRL) at approximately 9 cm proximally to the radial styloid. It bifurcates 5.1 cm proximally to the styloid and develops into five dorsal digital nerves.

### Bibliography

### CHAPTER 50 QUESTIONS
1. What intrinsic muscles of the hand are innervated by the median nerve?
   a. Dorsal interosseous
   b. Palmar interosseous
   c. 1st and 2nd lumbricals
   d. 3rd and 4th lumbricals
2. What is the role of the lateral bands of the extensor mechanism?
   a. Distributes force from the FDS, lumbricals, and interossei to the middle phalanx
   b. Distributes force from the FDP, lumbricals, and interossei to the distal phalanx
   c. **Distributes force from the EDC, lumbricals, and interossei to the distal phalanx**
   d. Distributes force from the EDC, lumbricals, and interossei to the middle phalanx

3. What is the function of the dorsal interossei muscles?
   a. Flexes IP joints
   b. Adducts digits and assists with MCP flexion and IP extension
   c. Flexes the thumb
   d. **Abducts digits and assists with MCP flexion and IP extension**

4. Which of the following muscles uses the flexor retinaculum, scaphoid, and trapezium as their proximal attachment?
   a. Adductor pollicis
   b. **Opponens pollicis, flexor pollicis brevis, and abductor pollicis brevis**
   c. Medial lumbricals
   d. Opponens digiti minimi, flexor digiti minimi, and abductor digiti minimi
1. Describe clinical assessment of contracture of the oblique retinacular ligament.
   Measure distal interphalangeal (DIP) flexion with the proximal interphalangeal (PIP) joint both extended and flexed. An increase in DIP motion with the finger flexed indicates contracture of the oblique retinacular ligament (ORL). If DIP motion is unchanged, range of motion probably is limited by joint contracture. If DIP motion is worse with flexion, the contracture is probably in the lateral bands.

2. How is the flexor digitorum superficialis clinically isolated when testing flexion at the PIP joint?
   Passively extend all the fingers except the test finger. This maneuver prevents individual flexion of the flexor digitorum profundus (FDP) but allows function of the flexor digitorum superficialis (FDS).

3. What occurs anatomically during a positive scaphoid shift test?
   The scaphoid shift test assesses the integrity of the scaphoid-lunate ligament. The evaluator applies pressure to the volar surface of the scaphoid with the thumb while passively holding the wrist in ulnar deviation. The evaluator maintains pressure over the scaphoid with the thumb while passively moving the wrist into radial deviation and slight flexion. At this time the evaluator releases pressure from the deviation of the wrist. If the integrity of the scaphoid-lunate (SL) ligament is adequately disrupted, pressure from the evaluator’s thumb will cause the scaphoid to glide onto the dorsal rim of the radius. Release of the thumb from the scaphoid causes the scaphoid to fall back into volar flexion, sometimes eliciting an audible, painful “clunk.”

   • Ulnocarpal stress test—wrist is placed in maximal ulnar deviation, axially loaded, while passively pronating and supinating the forearm. Pain with performance can indicate possible ulnocarpal impaction syndrome, L-T ligament injury, TFC tear, and/or ulnocarpal arthritis.
   • Ulnar fovea sign—tenderness with palpation of the soft region around the distal ulna (between the ulnar styloid and FCU and between the ulnar head and pisiform) could indicate foveal disruption of the distal radioulnar ligaments and/or ulnotriquetral ligament injury.
   • Piano key test—downward pressure to the dorsal distal ulna while the wrist is held in pronation; if the distal ulna falls volar with pressure and then immediately returns to normal anatomic position, a positive test is indicated. A positive test may indicate possible DRUJ instability.
   • L-T ballottement (shuck test)—pain with compression of the triquetrum medially into the lunate may be indicative of possible lunotriquetral pathology.
   • Midcarpal shift test—used to detect midcarpal instability, the patient is positioned in pronation and 15 degrees of ulnar deviation, and the examiner's thumb is placed over the distal capitate with applied volar pressure and axial loading of the metacarpals. A positive test occurs when a painful clunk reproduces that patient’s symptoms.
   • Pisotriquetral grind test—performed to detect pisotriquetral arthritis, the examiner compresses the pisiform upon the triquetrum and rocks it back and forth in a radial and ulnar direction. Pain is indicative of a positive test.

5. Describe the Bunnell-Littler test.
   Measure PIP range of motion (ROM) with the metacarpophalangeal (MCP) joint flexed and extended. If the PIP has more ROM with the MCP flexed, the test is positive and indicative of intrinsic muscle tightness.
6. How can the intrinsics be clinically isolated when testing extension at the PIP joint? The patient is asked to extend the interphalangeal (IP) joints while the examiner holds the metacarpophalangeal (MP) joint flexed.

7. Describe the test for vascular integrity of the radial and ulnar arteries. The Allen test is used to evaluate vascular integrity. The examiner compresses the patient’s ulnar and radial arteries at the wrist and instructs the patient to open and close the hand several times so that the hand appears pale. The examiner then releases one artery and notes how long it takes for the fingers to recover their normal color (usually <5 seconds). The test is performed for each artery and is useful in evaluating patency.

8. Describe splinting techniques for various upper extremity nerve injuries.
   - Median nerve—must maintain thumb abduction
   - Radial nerve—must maintain wrist extension; may use outrigger for passive finger extension
   - Ulnar nerve—must attempt to avoid claw deformity; use a splint that keeps the MCP joints flexed and the IP joints extended

9. In general, what is the appropriate position of the MCP joints in splinting? Why? The MCP joints should be splinted in some degree of flexion and not in an extended position. In the extended position, the collateral ligaments are shortened, whereas in the flexed position they are stretched. Consequently, it is easier to regain full flexion when the MCP joints are splinted in the flexed position. Because of the cam effect of the metacarpal head, the collateral ligaments are lengthened and, therefore, stretched in flexion.

10. What is the best position to splint the hand after injury or surgery to prevent ligament shortening and possible fixed deformity? The hand should be positioned with the wrist in extension, the MCP joint flexed, the IP joint extended, and the thumb palmarly abducted. Rehabilitation can be achieved more quickly and easily from this so-called “safe” position.

11. What is currently thought to be the most important factor in increasing a stiff joint passive range of motion with regard to static progressive mobilization via splinting or casting techniques? The amount of time a stiff joint is placed into end range (TERT: total end range time) appears to have a directly proportionate relationship to the increased PROM of the desired motion. This approach has been found applicable to most synovial joints. The amount of force applied varies per the tolerance of the joint. It is important that the force applied not create a significant inflammatory response, thus potentially resulting in decreased ROM. Brand suggests that the joint be placed in a moderately lengthened position for an extended duration, resulting in growth/lengthening of the connective tissue.

12. Define the syndrome of the quadriga. If a surgical procedure or injury prevents the proximal excursion of a single flexor profundus tendon, the full flexion of the adjacent profundus tendon may be impaired. This phenomenon can occur only in the long, ring, and small fingers because of the anatomic arrangement of the flexor profundus tendons and their origin from a common muscle belly. If the excursion of one profundus tendon is limited, the muscle cannot move the other tendons to their full extent. Verdan coined the term quadriga from the Roman chariot in which the reins to four horses were controlled and operated by a single rider.

13. Explain extrinsic tightness with respect to the extensor tendons. If the extrinsic (long) extensor tendon or tendons are adherent, for eg, to the metacarpal after a fracture has healed, excursion distal to this point is limited. Adherence limits simultaneous flexion of the MCP andPIP joints. If the MCP joint is flexed, the PIP joint is pulled into extension by the adhered tendon, and if the PIP joint is flexed, the MCP joint is pulled into extension. The test for extrinsic extensor tightness is exactly opposite to the test for intrinsic tightness.

14. What is a mallet finger? How does it develop? A mallet finger may develop after an injury to the extensor mechanism at the DIP joint, resulting in “droop” of the finger into flexion at the DIP joint. Such injuries include tendon rupture at the distal phalanx, laceration at or proximal to the DIP joint, avulsion fracture of the extensor tendon, or any injury that results in the loss of integrity of the extensor tendon insertion at the base of the distal phalanx. Loss of integrity usually is caused by a force applied to the tip of an actively extended finger, which forces the DIP joint into flexion. A classification system of mallet fingers was developed by Doyle as follows: Type I (closed injury, with or without small dorsal avulsion fracture), Type II (open injury, with laceration of the tendon), Type III (open injury, with loss of skin, subcutaneous cover, and tendon substance), and Type IV (mallet fracture).
15. How are mallet fingers currently treated?

- Type I mallet fingers are typically treated nonoperatively via 8 weeks of immobilization in a dorsal extension fixation device (AlumaFoam, Stack Finger Splint, or custom-made orthotic), placing the DIP joint in slight hyperextension. The patient is encouraged to maintain PIP joint flexion during the immobilization.
- Type II–III mallet fingers are typically treated operatively via an open repair of the tendon and K-wire fixation of the DIP in extension. The K-wire is typically removed at 4 weeks postoperatively, followed by dorsal extension splinting full time for 2 weeks.
- Type IV mallet fingers are often treated nonoperatively, but operative treatment with K-wire fixation may be appropriate when a large fracture with volar subluxation is present. Patients treated nonoperatively are splinted in extension for 5 to 6 weeks.
- Active ROM of the DIP joint post immobilization is encouraged; however, passive stretching of the DIP is not recommended because this could lead to an elongation of the extensor tendon with a subsequent increase in extensor lag.
- Maximum DIP ROM will take months to achieve, typically with a residual extensor lag present. Treatment intensity should be adjusted according to the patient’s progression and the amount of extensor lag present.

16. What is a trigger finger?

A trigger finger is a “locked” position of flexion. If the flexor tendon cannot reenter the fibro-osseous canal at the level of the A1 pulley because of thickening of the A1 pulley and reactive inflammation of the synovium of the flexor tendon, it assumes a flexed or locked position which can snap or “trigger” on forced extension. Treatment is injection of corticosteroids into the tendon sheath or release of the A1 pulley.

17. What is Dupuytren’s contracture? Which structures in the hand usually are involved?

Dupuytren’s contracture is a familial disease characterized by the development of new fibrous tissue in the form of nodules and cords in the palmar and digital fascia of the hand. The fibrous tissue leads to flexion contractures of the digits. Dupuytren’s contracture is more common in northern Europeans, diabetic patients, alcoholic patients, patients with liver disease, and patients who smoke. Men outnumber women by about 9 to 1. Dupuytren’s contracture involves certain components of the palmar fascia, the pretendinous bands, the superficial transverse ligament, the spiral band, the natatory ligament, the lateral digital sheet, and Grayson’s ligament. Treatment is usually surgical. Active splinting improves ROM in 59% of patients who comply with the program.

Structures involved in Dupuytren’s contracture. (From the Christine M. Kleinert Institute for Hand and Microsurgery, Inc., with permission.)
18. When is collagenase clostridium histolyticum (Xiaflex) performed with Dupuytren’s contracture patients?

- Xiaflex is currently used to treat adults with Dupuytren’s contracture when a “cord” can be felt. Injections can be performed up to 3 times in one location at 30-day intervals to disrupt the cord and achieve a straight or nearly straight finger. Typically, only 1 to 2 injections are necessary.

19. What is the current therapy regimen post Xiaflex injection and manipulation with Dupuytren’s contracture patients?

After the performance of the injection and manipulation by the physician, patients are fitted with a hand-based splint, placing the patient’s involved digit in maximal extension to be worn at night. Patients are also instructed to perform active isolated digit extension, abduction/adduction, IP hooking exercises, and gentle passive extension exercises to the involved digit. The above exercises are encouraged to be performed 10 times each at 4 times per day. More success has been found with MCP joint contractures than with PIP contractures. However, current research indicates that increased success with PIP flexion contractures could occur with a structured hand rehabilitation protocol, consisting of a home exercise program (MCP, PIP, and DIP exercises performed hourly to every other hour), 4 to 6 weeks of constant PIP extension, splinting between exercise performance, followed by constant extension splinting at night for 6 months.

20. What is Kienböck’s disease?

Kienböck’s disease is avascular necrosis of the lunate, usually as a result of distant trauma. Treatment is radial shortening or ulnar lengthening.

21. What is a ganglion cyst?

Ganglion is a Greek word meaning “cystic tumor.” Ganglions are mucus-filled cysts that account for 50% to 70% of all soft tissue tumors of the hand and wrist; they are more prevalent in women (female to male ratio of 3:1). There is no occupational proclivity, although the tendency to develop ganglions is seen with repetitive wrist activity. Dorsal wrist ganglions are the most common and account for 60% to 70% of all ganglions. The next most common site is the radial volar wrist (20%), followed by the flexor sheath of the fingers and the DIP joint. When ganglions become painful or noticeably enlarged, aspiration and cortisone injection may be indicated. Surgical removal of the cyst, in most cases, provides reliable definitive treatment.

22. Define swan neck deformity.

Swan neck deformity describes the posture of a finger in which the DIP joint is flexed and the PIP joint is hyperextended, giving the overall appearance of a swan neck. This deformity can be flexible or fixed. The many causes of swan neck deformity include volar plate deficiency at the PIP joint, FDS tendon incompetence, intrinsic muscle contracture, chronic mallet finger, and excessive traction by the extensor apparatus. This deformity usually does not respond to conservative splinting or an exercise program and requires operative management.

23. What is a boutonnière deformity?

A boutonnière (buttonhole) deformity is opposite in appearance to the swan neck deformity; it describes the posture of a finger in which the DIP joint is hyperextended and the PIP joint is flexed. It is caused by “buttonholing” of the head of the proximal phalanx through the extensor mechanism at the PIP joint (central slip injury), which allows the lateral bands of the extensor mechanism to move volarly. The result is extension of the DIP joint with flexion of the PIP joint. In contrast to swan neck deformity, boutonnière deformity responds to a specific and conventional splinting and exercise program.
24. **What is a pseudoboutonnière deformity?**

A pseudoboutonnière deformity presents similarly to a boutonnière deformity in that the patient has a digit with a PIP joint flexion contracture. With a pseudoboutonnière deformity, there has been an injury to the volar plate of the digit, resulting in scarring of the digit into a flexed posture at the PIP joint. The pseudoboutonnière deformity does not result in tightness, as evidenced by passive mobility of the DIP joint with the PIP joint held in extension.

25. **What is de Quervain’s disease? How is it treated?**

De Quervain’s disease is the inflammation of tendons and synovium, specifically the abductor pollicis longus and extensor pollicis brevis tendons and their surrounding synovium. It is also called stenosing tenosynovitis of the first dorsal compartment of the wrist. Finkelstein’s test may help diagnose de Quervain’s disease by eliciting pain over the radial side of the wrist. The test is performed by ulnar deviation of the hand after a fist is made over the flexed thumb. Caution should be taken in interpreting a positive Finkelstein test. Many causes other than de Quervain’s disease can generate pain with this maneuver, including first carpometacarpal arthritis, Wartenberg’s disease, and arthrosis of the radiocarpal and intercarpal joints. Anomalous tendons, multiple slips of the abductor pollicis longus tendon, and multiple subcompartments within the first compartment have been implicated as the cause for failure of nonoperative treatments, such as use of nonsteroidal antiinflammatory drugs, local steroid injection, and thumb and wrist immobilization. If nonoperative treatment fails, surgical release of the first dorsal compartment provides the best result.

26. **Compare the success rates of splinting, splinting with injection, injection, and operative management for the treatment of de Quervain’s tenosynovitis.**

- Splinting (wrist in 20 degrees extension with thumb in MP and IP extension)—30%
- Splinting with injection—57%
- Injection—69% (50% to 90% success rate with 1–2 injections)
- Operative management—91%

27. **What type of resistive exercise has been found most effective in decreasing lateral epicondylitis rehabilitation? What is the frequency of performance?**

Studies indicate that isotonic eccentric exercises performed for 3 sets of 15 repetitions daily for 6 to 12 weeks have been most effective. Resistance should be increased per the patient’s tolerance. Mild pain with performance is appropriate; however, care should be avoided to not worsen the patient’s condition. As tolerated, exercise to the wrist extensors should be performed with the elbow in extension and the forearm in pronation, with the arm resting on a supported surface. A 1-minute rest period should occur between the sets. Currently it is unclear as to what speed the exercises should be performed. Some therapists believe that exercise should be performed progressively more quickly, while others believe that the exercises should be performed slowly to avoid reaggravation of the tissues.

28. **What long-standing rehabilitation problem may occur when proximal phalanx fractures do not allow for rigid fixation and early motion?**

When range of motion exercises must be delayed to await fracture healing, adhesion of the flexor and extensor tendons to the fracture callus site is common. Adhesion can prevent tendon excursion and sometimes leads to either flexion contracture or extensor lag at the PIP joint, depending on postfracture position and healing pattern.

29. **Describe the “lumbrical plus” finger. What causes it?**

A lumbrical plus finger results in paradoxical extension of the PIP joint during attempted flexion of the finger. It occurs when the FDP is ineffective because of laceration, scarring, or amputation. “Pull” of the FDP
30. What changes in the hand are commonly associated with rheumatoid arthritis?
- Extensor tendon rupture (usually in the ring and small fingers)
- Rupture of the flexor pollicis longus tendon (Mannerfelt syndrome)
- Synovitis and degeneration of the radioulnar and radiocarpal joints (with sparing of the midcarpal)
- Palmar and ulnar dislocation of the MCPs
- Swan neck deformities of the fingers
- Boutonnière deformity of the thumb

31. What structures compose the triangular fibrocartilage complex (TFCC)?
The TFCC consists of the articular disc, the meniscal homolog, the tendon sheath of the extensor carpi ulnaris, the disc-lunate and disc-triquetral ligaments, and the disc-carpal and ulnocapitate ligaments. The TFCC distributes force and stabilizes the ulnar wrist.

32. How are TFCC tears diagnosed?
Diagnoses are made by clinical examination and imaging studies. Ulnar-sided wrist pain may indicate TFCC. Arthrography has a 27% false-positive rate, and false positives may be as high as 50% with MRI. The sensitivity of both modalities is about 85%.

33. What is the total excursion of normal flexor and extensor tendons?
- EDC—about 50 mm
- FDP—about 70 mm

34. What extensor tendon injuries should be repaired?
Injuries in which >50% of the tendon is lacerated should be repaired.

35. When are extensor tendon repairs weakest?
Extensor tendons lose 10% to 50% of their strength between postoperative days 5 and 21.

36. How long should extensor tendon repairs be protected?
- Zones I and II—6 to 8 weeks
- Zones III and IV—6 weeks
- Zones V to VII—4 to 6 weeks

37. Describe the rehabilitation of an extensor tendon injury.
For zones V to VII, dynamic splinting is begun at postoperative day 3. The wrist is held at about 40 degrees of extension, with the MCPs and IPs at 0 degrees in an elastic outrigger. Active flexion with passive extension is done 10 times per hour. Dynamic splinting is discontinued by the third or fourth week, and active ROM is started. Finger extension exercises are started at week 4, finger flexion strengthening at week 6, and resistive exercises at week 7.

38. How are flexor tendons nourished in the synovial sheaths of the fingers?
They are nourished in two ways—through the vincula, which are small blood vessel networks, and by synovial fluid diffusion.

39. When should a flexor tendon be repaired?
When >60% of the tendon is lacerated, it should be repaired.

40. When are flexor tendon repairs weakest?
Flexor tendons are weakest between postoperative days 6 and 12.

41. How much gliding of flexor tendons does joint motion produce?
Each 10 degrees of DIP motion produces 1 to 2 mm of FDP gliding, whereas each 10 degrees of PIP motion produces about 1.5 mm of FDP and FDS gliding.

42. List and briefly describe the three rehabilitative approaches to the treatment of flexor tendons.
- Immobilization—a conservative treatment approach, immobilizing the patient for a duration of 3 to 4 weeks in a dorsal blocking splint with the wrist in 10 to 30 degrees of flexion, 40 to 60 degrees of MCP flexion, and full IP extension. This treatment approach is primarily used with children and other individuals who are unable to adhere to more complex protocols.
Early passive mobilization—a treatment approach having various subprotocols including, but not limited to, Kleinert, modified Duran, and Washington. These protocols exist on the theory that passive mobilization of the tendon will result in increased tendon excursion with fewer adhesions and increased healing of the tendon. The modified Duran protocol uses a DBS with a strap to maintain the hand against the back of the splint. PROM is performed to the digits in flexion. The IP joints are actively extended while holding the MPs in full passive flexion. The Kleinert and Washington protocols also use a DBS; however, they use rubber band traction with a palmar pulley, providing passive flexion to the digit(s). The patient performs hourly active extension within the brace. The splints are worn for 3 to 6 weeks as appropriate with treatment, progressing according to the patient’s progress.

Early active mobilization—another treatment approach having various subprotocols; developed for the treatment of zone II tendon repairs. Early active mobilization protocols apply a controlled amount of stress to the repaired tendons, encouraging increased tendon glide with fewer adhesions. Various subprotocols use varying techniques for applying the controlled stress, including, but not limited to, active contraction while using rubber band traction and active contraction in a tenodesis splint.

43. In general, what are the expected outcomes after flexor tendon repair?
On average, patients regain 75% of grip strength, 77% of finger pressure, 75% of pinch strength, 76% of PIP motion, and 75% of DIP motion.

44. Name the risks and benefits of early active tendon mobilization post flexor tendon repair.
- Risks include gap formation and tendon rupture.
- Benefits include early improvement of biological properties and improved excursion.
- It is important to note that improved surgical techniques and materials have decreased the risks of early rupture rates and gapping. A review of 34 articles that compared rehabilitation approaches and rupture rates indicated:
  - Passive protocol—4% rupture rate (57/1598 patients)
  - Early active protocol—5% rupture rate (75/1598)
- Of the 75 patients who ruptured with an early active protocol, 72 had a 2-strand core suture, 3 had a 4-strand core suture, and no patients ruptured with a 6-strand core suture.

45. Describe the pyramid of progressive force exercises and how they apply to the treatment of flexor tendon repairs.
The pyramid of progressive force exercises are nine exercises that progressively increase the tensile stress onto the flexor tendon repair in efforts to increase tendon excursion when a lag with treatment gains has occurred. The first four phases of the pyramid are performed with the wrist in a protected position: passive protected extension, place and hold, active composite fist, and hook and straight fists. Phases five through nine are performed with the wrist unprotected: isolated joint motion (blocking), splint discontinuation, resistive composite fist, resisted hook and straight fist, and resisted isolated joint motion. The pyramid allows a guide to make an objective decision with the recovery of a flexor tendon, rather than strictly adhering to a flexor tendon “cookbook” approach.

46. Describe the difference between the congenital anomalies camptodactyly and clinodactyly.
- Camptodactyly is a flexion deformity of a digit in the anteroposterior plane. It more commonly occurs bilaterally at the PIP joint of the small finger. However, other joints and digits can be affected. This flexion deformity is caused by tightening of the skin, ligaments, and tendons; abnormal musculature; and irregularly shaped bones.
- Clinodactyly is a curving of a digit in the coronal plane. It commonly occurs bilaterally at the middle phalanx of the small finger into radial deviation. However, other phalanges and digits can be affected. The deformity is caused by shortening of the phalanx, most often on the radial side of the digit.

47. Describe the benefits of pressure therapy in the therapeutic management of a burned hand.
Pressure therapy is an essential key to preventing or controlling hypertrophic scarring after a burn. Pressure garments applying approximately 25 mm Hg of pressure will help control scarring by decreasing circulation to the maturing scar tissue, thereby preventing excessive growth of the scar tissue. This
will help the scar to mature into a flat, soft, and pliable scar. Pressure garments are typically elastic customized garments worn over the affected area 24 hours a day.

48. What scar contractures can potentially occur after a burn to the dorsum of the hand? What scar contractures can occur after a burn to the palmar surface of the hand? Burns to the dorsum of the hand can potentially result in the following contractures of the hand: MP joint hyperextension, IP joint flexion or hyperextension, flattening of the transverse arch, ulnar rotation of the fifth digit, thumb extension and adduction, thenar contractions, and interdigital web space contractions. Burns to the palmar surface of the hand can potentially result in the loss of thumb and finger extension and abduction.

49. Transfer of a muscle-tendon unit will result in what change in muscle grade, using a 0 to 5 muscle grading scale? Tendon transfers do not automatically result in any loss of muscle grade. Other variables can affect and decrease the muscle grade of a transfer; however, loss is not automatic.

50. How does systemic lupus erythematosus (SLE) differ from rheumatoid arthritis (RA) with regard to arthritis and pathodynamics? SLE and RA are both autoimmune disorders that result in chronic inflammation of the body’s tissues. SLE attacks and breaks down the joint capsule, causing ligament and volar plate laxity and tendon subluxation. Subsequent deformities, including joint instabilities and subluxations, occur because of the lost integrity of the ligaments and tendons. RA, however, causes inflammation of the synovium, resulting in erosion of cartilage and bone. RA can develop into a multitude of joint deformities and loss of motion.

51. What are common wrist and hand deformities developed by patients with a diagnosis of systemic lupus erythematosus? Common wrist and hand deformities include MP joint ulnar drift, MP volar subluxation, swan neck deformities, boutonnière deformities, lateral IP deformities, inability to extend the MP joint, thumb MP flexion posture, type 1 thumb deformity (thumb flexion and IP joint hyperextension), radiocarpal and intercarpal subluxation or dislocation, and dorsal subluxation of the distal ulna.

52. What is Graded Motor Imagery (GMI), and how is it effective in the treatment of CRPS? GMI is the utilization of mirror therapy and motor imagery to improve cortical organization and active motor networks. It is a 3-phase process:
- Phase 1—reestablishing laterality (right-to-left orientation)
- Phase 2—motor imagery performed with or without a mirror box. The patient imagines the affected limb in a variety of positions without moving the affected hand.
- Phase 3—mirror therapy is used to project the illusion that the noninvolved limb is the involved limb, fooling the brain into believing that pain-free movement is occurring with the involved hand. GMI has been found to be most effective with the treatment of chronic CRPS; however, utilization of the mirror box during the motor imagery phase of treatment has been found increasingly effective in the treatment of nonchronic CRPS-I. In short, GMI is used to slowly reeducate the brain to tolerate movement without activating defensive and protective outputs.

53. Define Raynaud’s phenomenon and discuss its etiology, clinical presentation, and treatment. Raynaud’s phenomenon is a vasospastic disorder of unknown origin. It is often experienced by individuals with vascular disorders, including systemic lupus erythematosus and atherosclerosis, as well as with rheumatoid arthritis. It is also commonly seen in response to repeated digital trauma, vibration, and prolonged cold exposure. The presenting symptoms of Raynaud’s phenomenon often include a “triple response” of vascular changes, although not all individuals experience three color changes, and the order of the color changes varies. Typically the digit(s) will assume a blanched appearance (lack of blood flow because of vasoconstriction), then cyanosis (venous pooling), and then a reddening of the digit(s) as arterial blood flow returns to the digit(s). Raynaud’s phenomenon can also occur in the feet, nose, ears, and tongue. Only two color changes have to occur for Raynaud’s phenomenon to be diagnosed. Treatment for this disorder consists of surgical removal of the proximal obstruction, patient education on the effects of smoking and caffeine, avoidance of cold and vibration and of vasoconstrictive medications, biofeedback, and use of oral vasodilatory medications.
1. What test is commonly used to test the integrity of the scaphoid-lunate ligament?
   a. Scaphoid-lunate slide test
   b. Scaphoid-lunate shift test
   c. Piano-key test
   d. Finkelstein's test
2. What drug can be used to attempt to release a Dupuytren’s contracture when a cord is present?
   a. Norco
   b. Zeflex
   c. Xiaflex
   d. Xefolase

3. What is a common complication to immobilization or lack of AROM after a proximal phalanx fracture?
   a. PIP flexion contracture
   b. Tendon rupture
   c. Weakness
   d. Flexor and/or extensor tendon adhesions

4. What diagnosis is the pyramid of progressive force exercises primarily used for in the clinical decision-making process?
   a. Flexor tendon repair
   b. Lateral epicondylitis
   c. S-L ligament repair
   d. Distal radius fracture

5. What is the typical position of immobilization of a mallet finger?
   a. Neutral
   b. Slight hyperextension
   c. Flexion
   d. Maximal hyperextension
1. Define boxer’s fracture.
Typically, fractures of the metacarpal necks of the ring and small fingers are called boxer’s fractures. The name is derived from the mechanism of injury. The fracture usually occurs when a person strikes or punches. The fracture usually angulates the apex dorsally because the volar cortex comminutes and the intrinsic muscles cause a flexed position secondary to crossing the metacarpophalangeal (MP) joints volar to their axis of motion. Usually boxer’s fractures can be treated nonoperatively with closed reduction and casting. The acceptable degree of angulation is undecided, but most surgeons accept up to 10 to 15 degrees in the second and third digits, 30 to 35 degrees in the fourth, and 50 degrees in the fifth.

2. What is a baseball finger?
Baseball finger, another name for mallet or drop finger, is typically a flexion deformity of the distal interphalangeal (DIP) joint resulting from injury of the extensor tendon to the base of the distal phalanx. This injury usually occurs during catching a ball (hence the name) or striking something with the finger extended and the tendon tight. The extensor tendon can pull directly off the dorsal distal phalanx or be associated with a dorsal articular fracture. The usual treatment is splinting of the DIP joint for 4 to 6 weeks. Average extensor lag after stack splinting is 8 degrees. Late management includes tenodermodesis, Fowler’s tenotomy, or oblique retinacular ligament (ORL) reconstruction.

3. What is a jersey finger?
A jersey finger is avulsion of the flexor digitorum profundus (FDP) tendon from the distal phalanx. The result is inability to flex the DIP. Treatment is surgical reattachment. Some loss of extension is common. Surgery should be performed soon after injury, especially if the tendon is completely retracted to the palm.

4. Describe the usual angulation of proximal phalanx fractures.
The angulation of proximal phalanx fractures, like that of most fractures, depends on two factors: the mechanism of injury and the muscles acting as a deforming force on the fractured bone. Typically, proximal phalanx fractures present with apex volar angulation. The proximal fragment is flexed by the interossei, which insert into its base, and the distal fragment is pulled into hyperextension by the central slip, which inserts into the base of the middle phalanx.

5. What is the usual or ideal position of immobilization of phalanx fractures?
Stable fractures often can be treated with buddy taping and early movement. If a fracture requires reduction and immobilization, the best position is the position of function, with the MP joints in almost full flexion and the interphalangeal (IP) joints in full extension. The MP joints rarely become stiff in full flexion because of the cam effect of the metacarpal hands on the collateral ligaments. The proximal interphalangeal (PIP) joints are least likely to become stiff in full extension.

6. Describe Bennett’s fracture and Rolando’s fracture.
Both are fractures of the base of the thumb metacarpal. Bennett’s fracture typically results from an axial force directed against a partially flexed metacarpal (often in a fight). The smaller of the two fracture fragments stays in place, attached to the anterior oblique ligament. The rest of the digit is pulled dorsally and radially by the abductor pollicis longus, whereas the more distal attachment of the adductor pollicis contributes additional dorsal displacement. Rolando’s fracture involves more comminution with the two fragments; usually a third large dorsal fragment in a Y- or T-shaped pattern is also present.
7. Describe the diagnosis and treatment of lateral collateral ligament injuries of the PIP joint.

Lateral dislocations are caused by an abduction or adduction force across the extended finger, usually in such sports as basketball, football, and wrestling. The radial collateral ligament (RCL) is injured more often than the ulnar collateral ligament (UCL). The PIP joint is stressed radially and ulnarily between 0 and 20 degrees. Angulation >20 degrees is an indication of collateral injury. The injury is treated with buddy taping and motion. The length of treatment depends on the degree of injury (complete or incomplete).

8. What are the differences between a dorsal and a volar PIP dislocation?

Dorsal dislocation is more common and results from hyperextension of the joint. The volar plate usually is injured at its attachment to the distal phalanx. Such injuries usually are treated with buddy taping for 3 to 6 weeks. Volar PIP dislocations are much less common. The injured tissue is the central slip. If the dislocation is treated with buddy taping, a boutonnière deformity probably will result. Hence, volar dislocation should be treated with immobilization of the PIP joint in full extension.

9. Define gamekeeper’s thumb.

An injury to the UCL of the thumb MP joint is called a gamekeeper’s thumb because British gamekeepers often developed UCL laxity, resulting from their method of putting down wounded rabbits. Today, however, it is seen most commonly in skiers. On examination the thumb is most tender over the ulnar aspect of the MP joint. The MP joint is stressed in both flexion and extension and in comparison with the other side. Often radiographic stress views confirm the diagnosis.

10. What is a Stener lesion?

With a complete tear of the UCL, the adductor aponeurosis often will be found under the torn UCL. This is called a Stener lesion and can prevent the ligament from healing. For this reason, most physicians recommend surgical treatment of complete UCL ruptures.

11. How is gamekeeper’s thumb treated?

Acute partial ruptures can be treated with a thumb spica cast for 4 weeks. The treatment of complete ruptures is controversial. Most believe that it should be treated surgically. Tears in the middle of the ligament can be repaired directly. If the ligament is avulsed, it is reattached with a bone anchor or tied over a button.

12. How are metacarpophalangeal radial collateral ligament tears of the thumb treated?

These injuries occur by an impact on the radial thumb region. Partial tears are treated with 6 to 8 weeks of immobilization. Treatment of complete RCL tears remains controversial. The ligament rarely displaces, similar to a Stener lesion; therefore, immobilization can allow for complete ligament tears to heal. However, the EPL can pull the thumb MCP into ulnar deviation, causing the RCL ligament to heal in an attenuated position. Some are advocating surgical repair to avoid subjective instability and joint degenerative changes.

13. Describe the radiographic evaluation of the wrist.

1. Anteroposterior (AP): three smooth arcs should be visible on the normal AP radiograph—across the distal radius; across the distal scaphoid, lunate, and triquetral; and across the proximal capitate and hamate.

2. Lateral: the radiolunatocapitate should form a straight line with the third metacarpal joint.
   - Normal scapholunate (SL) angle—30 to 60 degrees
   - Normal capitolunate (CL) angle—0 to 30 degrees

3. Flexion, extension, radial deviation, and ulnar deviation views, along with the previously mentioned, are enough to diagnose 90% of wrist injuries.

4. Special views
   - Scaphoid-radial oblique (supinated posteroanterior view)—with the forearm pronated 45 degrees from neutral, a full profile view of the scaphoid is obtained.
   - AP with fist compression or passive longitudinal compression may accentuate scapholunate dissociation and widening of the scapulolunate interval.
   - Carpal tunnel view—for this view, the wrist is in maximal dorsiflexion with the beam directed 15 degrees toward the carpus.
14. Describe Colles’, Barton’s, and Smith’s fractures.
The most common of the three is Colles’ fracture, which is extraarticular with dorsal angulation, displacement, and shortening. Barton’s fracture is an intraarticular shear fracture that may be dorsal or volar. A Smith’s fracture is often called a reverse Colles’ fracture. It is an extraarticular fracture with volar displacement and angulation.

15. What are chauffeur’s and die-punch fractures?
A chauffeur’s fracture is an intraarticular, triangular-shaped fracture involving the radial styloid. A die-punch fracture describes a depressed fracture of the lunate fossa.

16. When is surgery indicated for distal radius fractures?
An unstable fracture (one that cannot be held in position with a splint or cast) is an indication for surgery. Radial shortening >5 mm, dorsal angulation >20 degrees, and articular step-off >1 to 2 mm are also reasons to consider surgery.

17. Name the five factors that may contribute to instability of a distal radius fracture after closed reduction.
- Initial angulation >20 degrees
- Dorsal metaphyseal comminution >50% of the width of the radius
- Intraarticular fracture
- Age >60 years
- Considerable osteoporosis

18. What are the outcomes from volar plating of distal radius fractures?
Flexion and extension average 55 to 60 degrees, pronation/supination averages 75 degrees, and grip is approximately 75% to 80% of the contralateral side.

19. What is the second most common fracture of the wrist?
Scaphoid fractures are the second most common wrist fracture after distal radius fractures. They usually result from a fall on a dorsiflexed wrist. The diagnosis is made from the patient’s history and from examination findings of pain and swelling in the anatomic snuff box. Of course, radiographs are taken, but pain and tenderness justify initiation of treatment.

20. Where is the scaphoid most commonly fractured?
Around 65% of scaphoid fractures occur at the waist, while 10% occur at the distal body, 15% through the proximal pole, and 8% at the tuberosity. Because of differences in blood supply, fracture location can determine healing rates and times to union. The average time to union for waist fractures is 10 to 12 weeks, and 90% heal. It takes 12 to 20 weeks for proximal pole fractures to heal, and only 60% to 70% heal with cast treatment. Tuberosity and more distal fractures almost always heal in 4 to 6 weeks.

21. What are the treatment guidelines for scaphoid fractures?
1. Nondisplaced fractures: long-arm thumb spica for 6 weeks, then short-arm cast until the fracture is radiographically healed
2. Displaced fractures (ie, 1-mm step-off, >60-degree scapulolunate angulation, or >15-degree lunatocapitate angulation):
   - With acceptable reduction (ie, <1-mm step-off, <25-degree lateral intrascaphoid angulation, or <35-degree anteroposterior angulation), use a long-arm spica cast.
   - With unacceptable reduction, use open reduction with Herbert or compression screw or staple fixation; cast for 2 to 3 weeks, then encourage early movement.

22. When is surgery indicated for ulnar styloid fractures?
Ulnar styloid fractures are relatively common fractures. They can present alone or in association with a distal radius fracture (seen in approximately 50% of distal radius fractures). Surgical treatment is only indicated if the fracture leads to DRUJ instability. Fortunately, the most common pattern seen is an avulsion fracture of the tip of the styloid. These rarely lead to DRUJ instability and can be treated conservatively. Fractures involving the base of the styloid have a higher incidence of DRUJ instability and may need surgical fixation with a tension band or screw fixation.

Kienböck’s disease is defined by the radiographic finding of avascular necrosis of the lunate. The exact etiology is uncertain, but the probable cause is some combination of a traumatic event, repeated microtrauma, and/or injury to the ligaments carrying blood supply to the lunate. It also has been associated with relative shortening of the ulna compared with the radius (ulnar negative variance).
24. What are the four stages of Kienböck’s disease?

- Stage 1—sclerosis
- Stage 2—fragmentation
- Stage 3—collapse
- Stage 4—arthritis

25. What are the treatment options for Kienböck’s disease?

Surgical treatment before lunate collapse (stage 1 and stage 2 disease) involves decreasing the load across the lunate and/or improving the vascular supply to the lunate. The load is decreased by a radial shortening osteotomy in ulnar negative variance or capitates shortening osteotomy in ulnar neutral or ulnar positive variant wrists. Revascularization is performed by inserting a vascularized bone graft or a blood vessel into the lunate to promote blood flow. Once significant collapse has occurred (stage 3 and stage 4 disease), salvage procedures are employed. If the articular surfaces of the capitate and lunate fossa of the radius are intact, a proximal row carpectomy can be performed. In the setting of significant degenerative changes, a heavy laborer, or failure of previous surgical procedures, a total wrist arthrodesis is recommended.

26. Describe the classification of carpal instabilities.

The loss of the normal carpal ligaments and/or normal bony anatomy can lead to wrist instability. Wrist instability is classified as dissociative (CID) or nondissociative (CIND).

- CID—Carpal instability dissociative results from loss of the intrinsic ligaments. There are two types of CID:
  1. Dorsal intercalated segment instability (DISI) results from disruption between the scaphoid and lunate, allowing the scaphoid to rotate into volar flexion. The remaining components of the proximal row, the lunate and triangular muscles, rotate into dorsiflexion because of loss of connection to the scaphoid. DISI is the most common clinical pattern of carpal instability. The SL angle is >60 degrees, and the CL angle is >30 degrees.
  2. Volar intercalated segment instability (VISI) results from disruption of the ligamentous support to the triangular and lunate and leads to volar rotation of the lunate and extension of the triangular. It is the second most common instability. The SL angle is <30 degrees, and the CL angle is >30 degrees.

- CIND—A tear of the extrinsic ligaments can cause midcarpal or radiocarpal instability and is called carpal instability nondissociative.

- CIC—Carpal instability combined is caused by disruption both within and between rows, as seen in a transscaphoid perilunate fracture dislocation.

- Axial carpal instability—This is usually caused by violent trauma that results in longitudinal disruption.

27. What is scapholunate dissociation?

A complete tear of the scapholunate ligaments may result from a hyperextension injury and can lead to scapholunate dissociation, which disrupts normal proximal row kinematics. The lunate and triquetrum extend abnormally, supinate, and deviate radially. The scaphoid tilts into flexion, pronation, and ulnar deviation. This abnormal positioning affects how the wrist bears loads and can lead to pain, weakness, and arthritis.


Watson’s test is used to discern scapholunate dissociation. The wrist is moved from ulnar to radial deviation while pressure is applied over the volar tuberosity of the scaphoid. A positive test results when a painful clunk is felt from the proximal pole of the scaphoid as it subluxates over the rim of the radius.

29. What is the Terry Thomas sign?

A posteroanterior radiograph of the wrist that shows a gap >3 to 5 mm between the scaphoid and lunate, especially in comparison with the other side, suggests scapholunate dissociation (SLD). It is named after the English comedian who had a space between his front teeth. A more familiar eponym might be the Alfred E. Newman sign.

30. How is SLD treated?

Acute SLD can be treated with closed reduction and percutaneous pinning or open reduction, internal fixation, and repair of the ligament. Less than acute injuries can be treated with repair or reconstruction of the ligament and reinforcement of the capsule. Chronic injuries can be treated with limited or complete fusion, proximal row carpectomy, styloidectomy, or total wrist arthroplasty.
31. How are thumb UCL avulsion fractures best treated?
Small avulsion fractures of the thumb ulnar collateral ligament with minimal (<2.0 mm) displacement are best treated with open reduction and internal fixation. Minimally displaced UCL avulsion fractures frequently have significant rotation, which prevents successful fracture healing even with prompt cast immobilization.

32. Describe the Galeazzi fracture-dislocation.
In the Galeazzi fracture-dislocation of the forearm, fracture of the radial shaft includes a distal ulna dislocation. This injury was termed a “fracture of necessity,” stemming from the inherent instability of the fracture-dislocation and the need for surgical intervention. A key element of treatment is to stabilize the radius with internal fixation and restore the length of the radius. The stability of the distal radioulnar joint (DRUJ) is assessed, and, if unstable, repair of the triangular fibrocartilage complex (TFCC) and/or the capsule of the DRUJ is recommended.

33. Describe the Essex-Lopresti injury.
Essex-Lopresti injuries are a variant of Galeazzi fractures, except that the radial head has an intraarticular fracture combined with a dislocation of the distal ulna, tearing the capsule of the DRUJ and/or the TFCC. Injuries that have less than 1 to 2 mm of articular step-off, have less than 30% of the radial head joint surface involved, and those that are angled less than 30 degrees can be treated nonoperatively by immobilizing the forearm in supination to help reduce the DRUJ for 3 weeks before starting range of motion exercises. Fractures not within these criteria are best treated with open reduction and internal fixation.

BIBLIOGRAPHY

CHAPTER 52 QUESTIONS
1. What form of carpal instability is seen with a chronic scapholunate ligament tear?
   a. Volar intercalated segment instability (VISI)
   b. Dorsal intercalated segment instability (DISI)
   c. Carpal instability non-dissociative (CIND)
   d. Carpal instability complex (CIC)
2. In a Stener lesion, what structure traps the UCL in a displaced position?
   a. Extensor pollicis longus
   b. Volar plate
   c. Abductor aponeurosis
   d. Adductor aponeurosis
3. What position should you immobilize phalanx fractures?
   a. MCP flexion and PIP flexion
   b. MCP flexion and PIP extension
   c. MCP extension and PIP flexion
   d. MCP extension and PIP extension
1. What is Wartenberg’s disease?
Wartenberg’s disease, also known as superficial radial nerve entrapment and cheiralgia paresthetica, occurs infrequently and is often confused with de Quervain’s disease. Because of its superficial location along the distal radius, the nerve is easily compressed between the brachioradialis and extensor carpi radialis longus tendons with pronation and ulnar deviation. Superficial radial nerve entrapment creates a pattern of pain, numbness, and tingling over the dorsal lateral aspect of the hand. Wrist movement or blunt trauma aggravates the symptoms.

2. How is de Quervain’s disease clinically differentiated from superficial radial nerve entrapment?
Finkelstein’s test may be positive for both disorders. They can be differentiated by percussion along the anatomic course of the nerve, visual inspection for the presence or absence of edema along the dorsal lateral aspect of the hand, and sensory testing. Dellon described a nerve traction test for the superficial radial nerve. The patient is asked to pronate the forearm for up to 1 minute. If numbness and tingling are elicited or exacerbated over the superficial radial nerve field, entrapment is suspected. In addition, a positive Tinel’s sign on resisted pronation is confirmative. Electrodiagnostic tests can confirm the abnormality by demonstrating an absent superficial radial sensory response when the median and dorsal ulnar cutaneous responses are normal.

3. How is median nerve entrapment at the wrist clinically differentiated from a C8 root level compromise?
Symptoms of median nerve entrapment at the wrist include daytime and nocturnal pain, reduced perceptions of sensation in the radial three and one-half digits, and intrinsic muscle weakness. Nontraumatic cervical root lesions have symptoms including vague neck complaints, digital numbness and tingling, fine motor skill limitations, and muscle weakness. Median nerve entrapments are made worse with repetitive use and prolonged wrist flexion. Median nerve sensibility is limited to its nerve field, whereas sensory changes associated with a cervical root level lesion are dermatomal. Manual muscle testing of C8 ulnar- and radial-innervated muscles compared with median nerve-innervated muscles may indicate global C8 muscle weakness, whereas isolated median muscle weakness localizes the level of pathology.

4. Describe the clinical manifestations of compression of the deep motor branch of the ulnar nerve.
The second, third, and fourth digits are unable to abduct because of deep motor branch nerve pathology. The fifth digit should abduct because the intact abductor digiti minimi is innervated by the superficial ulnar motor branch. Sensation in the ulnar nerve field should be intact. Visual inspection may reveal ulnar guttering of the deep motor branch intrinsic muscles rather than the hypothenar musculature. Last, manual compression applied by the examiner’s thumb and index finger to the first web space (first dorsal interosseous/adductor pollicis muscle group) elicits pain compared with the same test applied to the abductor digitii minimi or the opposite side. This simple provocative pinch test appears to be a sensitive but nonspecific test; it is often present with ulnar neuropathy at the elbow and other sites as well. The mechanism of injury is associated with long-standing pressure in the palm, often an occupational hazard associated with pipe cutters, mechanics, and cyclists.
5. A complete ulnar nerve lesion at the wrist may produce motor paralysis of which muscles in the hand?
The majority of the intrinsic hand muscles receive their motor innervation from the ulnar nerve. A complete lesion of the ulnar nerve at the wrist causes extreme motor weakness or atrophy of up to 1 1/2 muscles, listed below in the order of innervation sequence:
- One subcutaneous muscle (the palmaris brevis, which puckers the skin over the hypothenar muscle group)
- Three hypothenar muscles (abductor digiti minimi, flexor digiti minimi, and opponens digiti minimi)
- Two medial lumbricals (numbers 3 and 4, which are in the palm, just radial to and originating from the third and fourth flexor digitorum profundus tendons)
- Three palmar interosseous muscles that adduct the fingers
- Four dorsal interosseous muscles that abduct the fingers
- One and one-half thenar muscles (adductor pollicis, both oblique and transverse heads, and the deep half of the flexor pollicis brevis muscle)
- Total hand muscle/nerve scores: ulnar = 1 1/2, median = 4 1/2, radial = 0

6. What is the significance of a positive Froment’s sign?
Ulnar nerve lesions result in a significant loss in hand grip strength. Weakness of the adductor pollicis, flexor pollicis brevis, and first dorsal interosseous muscles sharply impairs the pinching power of the thumb against the index finger. A simple test is to ask the patient to pinch a piece of stiff paper between the thumb and index finger while the examiner attempts to pull it away. The patient with an ulnar-deficient hand substitutes the flexor pollicis longus, causing hyperflexion of the thumb DIP joint to hold the thumb opposed to the radial side of the index finger. As the patient tries harder, the thumb flexes more and the pinch becomes weaker and fails.

7. Describe the tunnel of Guyon and a related nerve entrapment.
The lateral border of the tunnel of Guyon is the hook of the hamate, and the medial border is the pisiform bone. The floor is the joining of the ulnar extension of the transverse carpal ligament and pisohamate ligament. The overlying palmar fascia and palmaris brevis form the roof. The principal contents of the tunnel include the ulnar nerve and ulnar artery. The flexor carpi ulnaris inserts on the pisiform, but no tendons are contained within the tunnel of Guyon. Ganglions, fracture of the hamate hook, displacement of the pisiform bone, anomalous muscles, repetitive trauma, hypothenar hammer syndrome, arthritis, ulnar artery thrombosis, or aneurysm can cause various patterns of ulnar nerve involvement, ranging from complete motor and sensory to partial motor or sensory-only symptoms.

8. What is the significance of the palmaris brevis sign?
The palmaris brevis muscle is located on the ulnar aspect of the hand, superficial to the hypothenar muscle mass. When it contracts, it causes puckering of the skin on the ulnar border of the hand. To contract the muscle, ask the patient to abduct the small finger, which should cause a wrinkle over the proximal hypothenar region. The muscle receives innervation by the only motor twig of the superficial branch of the ulnar nerve as it passes immediately out of the tunnel of Guyon. Ganglions, fracture of the hamate hook, displacement of the pisiform bone, anomalous muscles, repetitive trauma, hypothenar hammer syndrome, arthritis, ulnar artery thrombosis, or aneurysm can cause various patterns of ulnar nerve involvement, ranging from complete motor and sensory to partial motor or sensory-only symptoms.

9. Name underlying systemic pathologies that may present with carpal tunnel syndrome.
- Kidney disease
- Thyroid disease
- Liver disease
- Diabetes, both NIDDM and IDDM

10. What are important factors to consider when reading an electromyograph (EMG) and nerve conduction velocity (NCV) report for a patient with suspected carpal tunnel syndrome?
Median motor studies include stimulation of the median nerve proximal to the carpal tunnel with recording over the abductor pollicis brevis muscle. Median sensory studies can be antidromic, which means that the stimulus is opposite of the physiologic direction of response transmission. In a sensory antidromic study, the nerve is stimulated proximally with a recording over that same nerve distally. For the median nerve, a typical antidromic sensory study involves stimulation proximal to the wrist and recording 14 cm
distally in either digit I or digit II, or possibly digit III. A palmar segment can be studied to more closely analyze the carpal tunnel involvement by performing the same antidromic study with digital recording and stimulation in the palm. The distal portion (from the palm to the fingers) is subtracted from the entire 14-cm distance to calculate the nerve conduction velocity across the carpal tunnel. Another method of evaluating the median sensory nerve involves stimulation of the nerve distally in the hand or palm and recording over the median nerve at the wrist. A focal conduction can be calculated directly when the median nerve is stimulated in the palm and the recording is made at the wrist. The NCV component of the report should contain latency, distance, amplitude, nerve conduction values, and temperature values.

11. Describe the classic findings of median nerve compression at the wrist.
Median nerve compression at the wrist results in numbness or pain in the radial three and one-half digits. These complaints are noted particularly at night. Patients also may complain of referred pain in the forearm or as proximal to the shoulder. Patients note an increased frequency of dropping items, apparently attributable to sensory loss. Such symptoms are more common in women than men. Symptoms are exacerbated with sustained activity, such as cumulative trauma disorders or repetitive wrist flexion associated with assembly occupations. Objective features of median nerve compromise vary with acuity of the lesion. In the early stages of median nerve compromise, sensory changes are negative. Two-point discrimination may be reduced along the second and third digits and the radial aspect of the fourth digit. Tapping over the median nerve at the wrist crease may produce an electric shock sensation to the median-innervated digits. Tinel’s sign (the presence of electric shock) provides clarification of pathology when it is positive and generally is detected only with moderate to severe cases of median nerve entrapment. Phalen’s test (wrist flexion test) is conducted with the wrists in complete volar flexion for up to 60 seconds. It is positive with aggravation of median nerve signs and symptoms. Thenar eminence manual muscle testing reveals reduced strength in the abductor pollicis brevis in long-standing cases of median nerve entrapment with muscle atrophy. Long-standing cases also are associated with deterioration of manual dexterity as sensorium and muscle atrophy persist.

12. Are clinical examination tests valid for evaluating carpal tunnel syndrome?
Tinel’s sign is used clinically to evaluate the status of peripheral nerve function. A tingling sensation, paresthesia, or electrical shock felt distally to the tapping site in the median nerve distribution to the thumb, index, middle, or ring fingers is considered a positive Tinel’s sign. Reported values of specificity range from 55% to 95%, and sensitivity ranges from 45% to 75%. Tinel’s sign may be present in normal people and is not descriptive of abnormality; therefore, it may be more useful to rule out carpal tunnel syndrome when it is negative.

### Diagnostic Tests for Carpal Tunnel Syndrome

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## Diagnostic Tests for Carpal Tunnel Syndrome (Continued)

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### Tourniquet Test (Blood Pressure Cuff Test)

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13. What are the most sensitive electromyographic indicators for carpal tunnel syndrome when reading an EMG report?

Median sensory studies typically show the earliest abnormalities in carpal tunnel syndrome. Reports can be interpreted more easily when there is an indication both of the site of median sensory nerve stimulation and recording and of the distance traveled by the stimulus between onset and recording.

Lew et al. examined the sensitivity and specificity for several median sensory conduction tests in determining carpal tunnel syndrome. They examined the wrist to digit, palm to digit (subtracted from the wrist to digit), and palm to wrist median sensory studies in 44 normal and 136 symptomatic hands. They found that the short segment from the palm to wrist was the most sensitive (75%) for carpal tunnel syndrome.

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**Diagnostic Tests for Carpal Tunnel Syndrome (Continued)**

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Edx +, Use of electrodiagnosis as gold standard for diagnosis; PPV, positive predictive value; NPV, negative predictive value; NR, not reported.

*Indicates values that were not presented but calculated from published data.
†Negative hands of same 50 subjects, served as the controls.
‡Hypothetical population incidence rate of 0.50.

From Wiederien et al.: Carpal tunnel syndrome: A literature review for the effect of the median nerve compression test on median nerve conduction across the carpal tunnel, 1999, U.S. Army–Baylor University.
14. What is the clinical difference between an anterior interosseous nerve injury and median nerve injury at the wrist?

The anterior interosseous nerve, which innervates the flexor pollicis longus, pronator quadratus, and flexor digitorum profundus to the index and long fingers, may be injured traumatically or become inflamed spontaneously. Pain along the volar surface of the forearm may be associated with local trauma or heavy muscle exertion. As weakness develops, fine motor control suffers and pinching motion is reduced. Physical examination reveals absent or reduced flexion of the IP joint of the thumb and DIP joint of the index finger, caused by weakness of the flexor pollicis longus and flexor digitorum profundus muscles. Sensation to the volar surface of the forearm and median-innervated digits is intact. Percussion over the nerve may produce radiating pain along the path of the nerve distally to the pronator quadratus.

15. What are the normative EMG and nerve conduction values used to define pathology in carpal tunnel syndrome, and how are they applied in a patient case setting?

To control for the effects of temperature, some examiners use comparisons between nerves in the same limb. The following are some guidelines:

- Perform a median/ulnar sensory orthodromic study.
- Difference greater than 10 m/sec in velocity or 0.5 msec in latency between median and ulnar sensory studies in the palm-to-wrist segment is considered abnormal.
- Difference greater than 0.5 m/sec between radial and median sensory studies (stimulate at wrist and record from thumb) over a 10-cm distance is considered abnormal.
- Absence of median sensory response is abnormal.

If absolute values are used, the following are some commonly used criteria:

- Median motor latency greater than 4.2 msec is considered abnormal
- Sensory digital latency over a 14-cm distance greater than 3.6 msec is considered abnormal.
- Sensory palmar latency (orthodromic) greater than 2.2 msec over a 7- to 8-cm distance is considered abnormal.

16. What are the common risk factors associated with carpal tunnel syndrome?

There are several anthropometric characteristics associated with carpal tunnel syndrome to include the following:

- Body mass index/obesity
- Hand length-to-body height ratio
- Greater wrist width
- Wrist index greater than 0.69

The wrist index was described by Johnson et al. (1993) and is measured using calipers at the distal wrist crease to measure wrist width in centimeters in the anteroposterior (AP) and mediolateral (ML) planes. The AP measurement is then divided by the ML measurement to calculate the wrist index.

Other risk factors have been identified as:

- Smoking
- Alcohol use
- Kidney disease
- Thyroid disease
- Pregnancy
- Lactation
- Diabetes

17. What may produce carpal tunnel syndrome in children?

Scheie syndrome is a rare genetic lysosomal storage problem with an increased prevalence of carpal tunnel syndrome (CTS) in adolescence. Scheie syndrome also presents with hernia, coarse facial features, joint contracture, bone deformities, hepatomegaly, and clouding of the cornea. Hunter syndrome is another rare, inherited pathology with increased prevalence of CTS in children. CTS presentation in Hunter syndrome is typically bilateral, with a reported mean age of only 9.75 years. Other genetic conditions have been associated with childhood CTS to include familial CTS, hereditary neuropathy with liability to pressure palsy (HNPP), Schwartz-Jampel syndrome (SJS), melorheostosis, Leri’s syndrome, Dejerine-Sottas syndrome, and Weill-Marchesani syndrome. Other cases of CTS in children have been...
reported to be associated with thickening of the flexor retinaculum in familial cases and with scleroderma. Malformation of the palmaris longus, flexor digitorum superficialis, first lumbrical, and palmaris brevis muscles, or the presence of a vestigial or supernumerary muscle in the forearm or hand, has also been reported to have a link with childhood CTS.

18. Are there different classifications or degrees of carpal tunnel syndrome, and, if so, how would an electromyographer grade carpal tunnel syndrome?

Chang et al. (2008) established normal values from 50 normal subjects using means ±2 standard deviations to arrive at the upper and lower limits of normal. They defined mild carpal tunnel syndrome as having a prolonged median motor distal latency from 3.6 up to 5.4 ms, slow median sensory velocity between 30 and 37 m/s, reduced median CMAP of 3.9 mV and reduced median SNAP of 6.1 μV, and increased proportion of polyphasic motor units with EMG sampling of the APB. Moderate carpal tunnel syndrome was defined as a prolonged median motor distal latency between 5.4 and 7.2 ms, slow median sensory conduction velocity between 20 and 30 m/s, reduced median CMAP between 2.5 and 3.8 mV, reduced median SNAP between 4 and 6 μV, or a markedly increased proportion of polyphasic potentials or markedly increased motor unit duration with EMG sampling of the APB. Severe carpal tunnel syndrome was defined as a prolonged median motor distal latency of greater than 7.2 ms, slow median sensory conduction velocity of less than 20 m/s or absent, reduced median CMAP amplitude below 2.5 mV, reduced median SNAP amplitude below 4.0 μV, presence of retrograde degeneration of the median forearm segment, presence of fibrillations, and positive sharp waves or electrical silence with attempted APB muscle contraction upon EMG examination.

19. A patient complains of numbness and tingling in the small and ring fingers on only the palmar side of the hand with no complaints of numbness in the forearm or in the dorsal hand. What is your suspected location of injury and why?

The injury is to the ulnar nerve distal to or at the level of the wrist. The ulnar nerve supplies sensation to the small and ring fingers and is a derivative of the C8 and T1 roots, the lower trunk and medial cord of the brachial plexus. As such it is possible that a lesion in any one of these sections could produce tingling in the small and ring fingers; however, a proximal lesion at the level of the lower trunk or proximal medial cord would most likely produce numbness in the medial forearm via the medial cutaneous nerve of the forearm, which is a derivative of the medial cord. In addition, tingling would be present on the dorsum of the small and ring fingers in a lesion at the level of the midforearm or proximal to this location because the supply to the dorsal aspect of the small and ring fingers is from the dorsal ulnar cutaneous sensory branch of the ulnar nerve proper that exits the ulnar nerve approximately 10 cm proximal to the wrist.

**Bibliography**


1. Describe the blood supply to the spinal cord.
The spinal cord is supplied by 3 arteries. The anterior spinal artery supplies 80% of the spinal cord, and paired dorsal arteries supply the remainder. The anterior spinal artery is often mistaken as one contiguous artery. It actually is 3 separate anterior arteries, with the superiormost artery supplying C1-T3, the middle supplying approximately T3-T8, and the inferiormost anterior spinal artery supplying the area from T8 to the conus. The superior anterior spinal artery is fed by branches of the vertebral artery. The middle and inferior sections are fed by direct radicular branches from the aorta.

2. Describe the cross-sectional location and function of the lateral corticospinal tracts, the spinothalamic tracts, and the dorsal column tracts of the spinal cord.
   - The lateral corticospinal tracts are located laterally and slightly posteriorly. Within the column, arm function is located medially, truncate function in the middle, and leg function most laterally. This controls ipsilateral motor function.
   - The spinothalamic tracts are anterior and lateral. They transmit pain and temperature sensation from the contralateral side of the body.
   - The dorsal column tracts are located dorso medially. Within the dorsal column, arm function is most centrally located and leg function is most peripheral. These columns transmit light touch, proprioception, and vibration.

3. Describe the 6 major incomplete spinal cord injury syndromes and their characteristics.
   - Anterior cord syndrome: Injury to the anterior two thirds of the spinal cord either due to direct compression or disruption of the anterior spinal artery supply. Usually results in complete paralysis and spasticity (lateral corticospinal tracts are nonfunctional) but maintains proprioception and deep pressure (dorsal columns).
   - Central cord syndrome: This is the most common and results in motor injury in the upper extremities more than the lower extremities. Usually the result of a hyperextension injury in the cervical spine. This has the best prognosis.
   - Posterior cord syndrome: Injury to the dorsal columns, resulting in loss of proprioception.
   - Brown-Séquard syndrome: Injury to one half of the spinal column, resulting in loss of ipsilateral motor function and proprioception, with contralateral loss of pain and temperature.
   - Conus medullaris syndrome: Can result in urinary incontinence because of spastic bladder (high conus lesion) or flaccid bladder (low conus lesion). Usually results in asymmetric weakness and loss of sensation. May have a mix of upper and lower motor neuron syndromes.

4. Describe Fryette’s laws of spinal biomechanics.
   1. In the cervical spine, side-bending and rotation occur to the same side.
   2. When the lumbar and thoracic areas of the spine are in neutral position, side-bending and rotation occur to the opposite side.
   3. When the lumbar and thoracic areas of the spine are in extreme flexion, side-bending and rotation occur to the same side.
   4. In actuality, spinal movement is highly variable among different people and even in the same person in different regions of the thoracolumbar spine.

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*In honor of Dr. Harry N. Herkowitz.*
5. Describe the normal ranges of motion of each section of the spine.

- **C0-C1**—10 to 15 degrees of flexion/extension, 8 degrees of lateral flexion, minimal rotation
- **C1-C2**—10 degrees of flexion/extension, 45 degrees of rotation, little or no lateral flexion
- **C3-C7**—64 degrees of flexion, 24 degrees of extension, 40 degrees of lateral flexion, 40 degrees of rotation
- **T1-S1**—80 degrees of flexion, 25 degrees of extension, 45 degrees of rotation, 35 degrees of lateral flexion

In general, flexion/extension and lateral flexion increase from cranial to caudal. Rotation decreases from cranial to caudal.

6. List the important ligaments of the cervical and lumbar spine. Specify their origin, insertion, attachment, and function.

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<thead>
<tr>
<th>Name</th>
<th>Origin</th>
<th>Insertion</th>
<th>Attachment</th>
<th>Function</th>
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<td>Skull</td>
<td>Sacrum</td>
<td>Anterior surface of vertebral bodies</td>
<td>Limits extension</td>
<td>Atlantoaxial and anterior atlanto-occipital membrane</td>
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<tr>
<td>Posterior longitudinal ligament</td>
<td>Skull</td>
<td>Sacrum</td>
<td>Posterior surface of vertebral bodies</td>
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<td>Helps extend membrane</td>
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7. Describe the anatomy of the intervertebral disc.
Each motion segment has one disc, with the exception of C1-C2. The disc is an avascular structure composed of an outer annulus fibrosus, an inner nucleus pulposus, and a cartilaginous end-plate interface superior and inferior to the vertebral body. The jellylike nucleus pulposus acts as a shock absorber, and the annulus helps stabilize and transmit the loads transmitted to the nucleus pulposus by axial loading. The biomechanical vertical compression forces to which the nucleus is exposed are converted into horizontally directed forces that the tough outer annulus helps absorb and distribute to the motion segment. The fibers of the annulus are arranged in alternating perpendicular lamellar fibers, arranged at a 45-degree angle to the vertebral end plates. Disc height is larger anteriorly in the cervical and lumbar spine and shorter anteriorly in the thoracic spine, which accounts for the cervical and lumbar lordosis and thoracic kyphosis.

8. How does the disc obtain its nutrition?
Because the disc is avascular, the disc cells must obtain their nutrition through local diffusion. Diffusion of uncharged solutes, such as glucose, occurs primarily through the end plates. Negatively charged solutes diffuse through the annulus.

9. What is the effect of exercise on disc nutrition?
Exercise provides nutrition through pumping of the disc, which aids in solute transport and possibly promotes nutrition through increasing external local vascularity.

10. What changes occur in the disc with aging?
As the spine ages, degenerative changes occur, and the chemical composition of the nucleus pulposus changes. In the nucleus pulposus of a youth, type II collagen, proteoglycans, and water are abundant. Over time, the nucleus decreases proteoglycan production, loses water, and produces less type II collagen. It begins to resemble the annulus, which consists mostly of type I collagen. Whereas young discs maintain height, aging discs lose height with degeneration and water loss.

11. Describe the facet articulations of the spine.
- Occiput anterior (OA)—At the atlanto-occipital joint is an articulation between the condyles of the occipital bone and superior facets of the atlas (C1). The anterior and posterior occipital membranes and joint capsule support this articulation.
- Atlanto-axial (AA)—Great mobility is needed at C1-C2, where 50% of cervical rotation occurs. As a result of the strong coupling pattern at this joint, axial rotation is associated with vertical translation and contralateral side-bending. The facets of C1-C2 are horizontally aligned but biconvex in design. As a result, C1 is vertically at its highest position in neutral rotation and in its lowest position in full left or right lateral rotation.
- Cervical—The facet joints are angulated at 45 degrees to the vertical in the sagittal plane at C2-C7. This orientation allows increased mobility compared with the thoracic and lumbar portions of the spine, including the coupled axial rotation observed with lateral bending.
- Thoracic—In the thoracic spine, the facet joints are oriented at 60 degrees to the vertical in the sagittal plane. This orientation leads to increased rigidity in the thoracic spine, with decreased axial rotation in the lower thoracic spine compared with the upper thoracic spine. This decrease is secondary to transitioning of the facets to a more lumbar-type facet.
- Lumbar—In the lumbar spine, the facet joints are vertically oriented, and their configuration allows little rotation and flexion. The superior facets are oriented dorsomedially, almost facing each other. The inferior facet processes face ventrolaterally. This configuration allows for a locking-in of each articulation of the superior facets from the lower vertebrae with the inferior processes of the upper vertebrae.

12. How does the spine receive loads in different postures? What is the effect of a backrest or lumbar support?
Nachemson measured intradiscal pressure with pressure transducers placed at L3-L4 in normal patients at different postures. His research showed that the least loaded condition is lying supine. In vivo, loads increase sequentially with lying on the side, standing, sitting in a chair, standing with flexed spine, sitting in a chair with flexed spine, standing with flexed spine carrying a weight, and sitting in a chair with flexed spine carrying a weight. The relative loads are as follows:
- Lying on the side—25%
- Standing—100%
- Seated—145%
• Standing with forward bend—150%
• Seated with forward bend—180%

The loads on the lumbar spine are lower during supported sitting than during unsupported sitting because part of the weight of the upper body is supported by the backrest. Backward inclination and use of a lumbar support further reduce the loads.

13. What are the dimensions of the spinal canal? How does the canal size change in different areas of the spine?
The space available for the cord (SAC) is defined as the area posterior to the posterior longitudinal ligament and anterior to the ligamentum flavum. The normal canal opening is about 17 to 20 mm in the cervical spine. Stenotic symptoms often occur when this space decreases to <14 mm. The SAC is narrowest at T5. The spinal cord is approximately 42 cm long in women, 45 cm long in men, and 10 mm in diameter. Two levels of enlargement correlate with the levels of upper and lower extremity innervation: the C4-T1 level and the L2-S3 level. The end of the cord, the conus medullaris, starts at the T10-T11 disc level. The L1-L2 disc level marks the end of the conus medullaris and the start of the cauda equina.

14. How are the facet joints innervated?
The spinal nerve divides into ventral and dorsal rami. The dorsal primary ramus gives medial, lateral, and intermediate branches to the facet joints and paraspinal muscles. The medial branches are especially important in facet joint innervation. Two branches—superior and inferior—inervate the facet joint above and below the level of the nerve root. For example, the descending medial branch of L1 and the ascending medial branch of L3 innervate the L2-L3 facet.

15. Where is the nerve root in relation to the pedicle and disc in the cervical and lumbar portions of the spine?
In the cervical spine, the spinal nerve roots exit directly lateral from the spinal canal adjacent to the corresponding disc and superior to the inferior pedicle. The nerve roots are numbered for the cervical vertebra above which they pass. For example, the C4 nerve root exits beneath the C3 pedicle and above C4. Because there are eight cervical nerve roots but only seven cervical vertebrae, the numbering changes at C7-T1. Here the eighth cervical root passes. Thus the nerve root passing under the pedicle of T1 is the T1 nerve root.

In the lumbar spine, the nerve root passes directly under the pedicle for which it is named. For example, the L4 nerve root passes beneath the pedicle of L4 at the L4-L5 intervertebral level. The nerve root is usually superior to the disc at that level, whereas the cervical nerve roots exit at the level of the disc.

16. How does spinal movement affect the size of the intervertebral foramen?
Cadaver studies indicate that foramen size increases in flexion by 24% and decreases in extension by 20%. Changes caused by lateral bending and axial rotation are not as impressive.

17. What is Spurling’s sign?
Spurling’s sign is performed by extending the patient’s neck with lateral bending. This results in narrowing of the neuroforamen on the side that the head is located. A positive test results in the production or worsening of radicular symptoms on that side. This may indicate cervical radiculopathy.

18. Describe the function of the facet joints and their role in load bearing.
The facets are thought to protect the lumbar spine against torsional disc damage. They decrease the allowable rotation to which an intervertebral disc is exposed and share spinal load with the disc. Investigators debate the exact amount of load that the facets bear, but estimates range from 9% to 25%, depending on whether the spine is flexed or extended. If the spine is arthritic, the facets may bear almost 50% of the load.

19. Describe the form and function of the uncinate processes.
The uncinate processes, which are fully developed by age 18, are thought to prevent posterior translation as well as some degree of lateral bending. They are also thought to be a guiding mechanism for flexion and extension in the cervical spine.

20. What happens during the straight-leg raise test?
Investigators have shown that the L4, L5, S2, and S3 nerve roots run in a sigmoid course through the foramina, with slack that can be taken up. The S1 nerve root runs a relatively straight course through the foramen. During a straight-leg raise (SLR) test, the sciatic nerve trunk is drawn downward
through the greater sciatic notch, pressing tightly against the anterior bony structures. From 0 to 30 degrees, at 5 cm above the horizontal position, movement of the nerve at the greater sciatic notch already has begun. After a bit more elevation, the lumbosacral plexus is moving against the sacral ala, without root movement. From 35 to 70 degrees, the nerve roots begin moving. At 70 to 90 degrees, the nerve roots no longer move, but more tension is placed on all of the neural structures. The seated SLR has been shown in some studies to be more sensitive than the supine SLR.

21. Which muscles are recruited to initiate and complete lumbar flexion and extension? Flexion is initiated by the abdominal muscles and the vertebral portion of the psoas. With further flexion, the erector spinae muscles are recruited as the forward moment acting on the spine increases. As the spine is further flexed, the posterior hip muscles are activated. At full flexion, the erector spinae muscles become inactive and are at full stretch. These muscles and the posterior ligaments supply passive restriction to further forward flexion. To extend from this position, the pelvis tilts backward and the spine extends backward, using the above muscles in reverse sequence.

22. How effective are lumbosacral corsets for relief of spinal disc pressure? Maximal disc load reduction with tight corsets is approximately 20% to 30%. The use of an abdominal corset with a chair back brace also may be helpful in diminishing loads applied to the lumbar spine.

23. What changes in lumbar spine intervertebral flexion and extension can be expected after lumbar disc replacement surgery? A recent study has shown that at a mean follow-up of 8.7 years, at least 2 degrees of intervertebral motion is maintained in 66% of patients. The study found the mean range of motion for all discs to be 3.8 degrees.

24. List the ratios of disc height to vertebral body height in the cervical, lumbar, and thoracic areas of the spine.
   - Cervical—1:4
   - Thoracic—1:7
   - Lumbar—1:3

25. What active range of motion in the cervical spine is required to perform activities of daily living? In order to perform activities of daily living, 65 to 70 degrees of both rotation and flexion and extension is needed. In a recent study, shoe-tying required the greatest amount of flexion and extension (66.7 degrees), while driving a car in reverse (67.6 degrees) and crossing the street (~85 degrees) necessitated the greatest rotation.

26. Describe the effect on spinal loading of the double SLR, supine sit-up, trunk curl, and reverse curl.
   - A double SLR involves mostly the psoas muscle; little abdominal muscle function can be measured.
   - A supine sit-up with the knees and hips bent eliminates psoas recruitment and actively strengthens the abdominal muscles; however, because of greatly increased disc pressure, these exercises should be avoided.
   - A trunk curl or half sit-up, in which only the shoulder blades clear the floor, lessens lumbar motion, recruits abdominal muscle function, and lessens the load on the discs.
   - A reverse curl, in which the knees are brought to the chest and the buttocks are raised from the floor, activates the internal and external obliques as well as the rectus abdominus but with less disc pressure than sit-ups.

27. What lumbar pressures are involved in commonly used exercises and postures?

<table>
<thead>
<tr>
<th>Exercise</th>
<th>Maximal Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standing</td>
<td>100%</td>
</tr>
<tr>
<td>Sit-up</td>
<td>210%</td>
</tr>
<tr>
<td>Fowler’s position</td>
<td>35%</td>
</tr>
<tr>
<td>Reverse curl</td>
<td>140%</td>
</tr>
<tr>
<td>Bilateral SLR</td>
<td>150%</td>
</tr>
<tr>
<td>Prone extension</td>
<td>130%</td>
</tr>
</tbody>
</table>

28. What are the differences in lumbar spine muscle kinematics between patients with chronic low back pain and normal subjects? Chronic low back pain patients have been found to have earlier activation and significantly longer activation of their erector spinae musculature compared with normal controls during a lifting exercise.
Longer contraction may suggest that chronic low back pain patients have changed their motor program from an open to a closed loop system.

29. **What is the effect of age on cervical spine range of motion?**
   Average adolescent flexion-extension measures approximately 130 degrees whereas adult men (average age 37) have only 117 degrees of flexion-extension. Similarly, rotation decreased from 160 to 153 degrees for these same age groups. This exemplifies the importance of cervical spine range of motion exercises beginning in young adulthood.

30. **What are the effects of lumbar discectomy on trunk musculature?**
   At 2 months after surgery, patients undergoing lumbar spine discectomy were found to have 44% decreased trunk flexion strength and 36% decreased trunk extension strength compared with controls. This may indicate a need for formal trunk strengthening after lumbar spine surgery.

31. **What is the effect of leg length discrepancy on spinal motion during gait?**
   There is a significant asymmetric lateral bending motion in the lumbar spine during gait in patients with leg length discrepancies of 3 cm. This may lead to accelerated degenerative changes in the lumbar spine.

32. **How much nerve root movement occurs in the lumbar spine with SLR?**
   - L4—1.5 mm
   - L5—3.0 mm
   - S1—4.0 mm
   - Sacral ala—4.5 mm
   - Sciatic notch—6.5 mm

33. **How much nerve root movement occurs in the lumbar spine with forward flexion while standing?**
   - L1-L2—2 to 5 mm
   - L3—2.0 mm
   - L4—0 mm

34. **How much dural movement occurs in the cervical spine with flexion and extension?**
   - C5—Approximately 3 mm
   - C8— Approximately 9 mm
   - T1—Approximately 13 mm
   - T5—Approximately 7 mm
   - T10—Approximately 2 mm

35. **Describe key vertebral landmarks.**
   - L5—Smallest lumbar spinous process
   - L3—Largest lumbar transverse process
   - C7—Most prominent cervical spinous process
   - C6—Most inferior bifid cervical spinous process

36. **Discuss the 3-column model of the spine.**
   - The anterior column consists of the anterior longitudinal ligament to the mid-vertebral body.
   - This middle column includes the posterior vertebral body of the posterior longitudinal ligament.
   - The posterior column includes the pedicles, facets, laminal transverse processes, spinous processes, and interspinous ligament.

37. **Describe the sacroiliac joint.**
   The sacroiliac joint is an irregularly shaped articulation between the lateral-facing facet of the sacrum and the medial-facing facet of the ilium. The joint is supported by 3 very strong ligaments, including the posterior sacroiliac ligaments (strongest), the intersosseous ligaments, and the anterior sacroiliac ligaments. The curvature of the joint and the strong ligaments allow for very little motion of the sacroiliac joint.

38. **What is Patrick’s test, and how is it used to assess the sacroiliac joint?**
   Patrick’s test, also known as the FABER test (flexion-abduction-external rotation), is performed with the patient supine. The patient’s leg is flexed, abducted, and externally rotated at the hip. Next, the ipsilateral knee is pushed by the examiner posteriorly and laterally. This causes compression of the ilium onto the sacrum at the sacroiliac joint. A positive test elicits pain in the region of the joint and can indicate sacroiliitis.
BIBLIOGRAPHY


CHAPTER 54 QUESTIONS

1. Where does the greatest amount of nerve root movement occur with SLR?
   a. L4
   b. L5
   c. S1
   d. Sciatic notch

2. Lumbar pressures are greatest:
   a. Standing
   b. Laying on side
   c. Standing with forward bend
   d. Seated with forward bend

3. Cervical rotation is greatest at:
   a. C0-C1
   b. C6-C7
   c. C7-T1
   d. C1-C2
1. What is the role of bed rest in acute back pain?

Bed rest has a very limited role; evidence-based treatment guidelines for acute LBP and a review of thirteen national guidelines by Koes (2010) recommend early and gradual activation of patients and discourage bed rest. “Rest from activity but not from function” is a good adage to follow in this situation.

2. Describe the structure of the intervertebral disc.

Moving centrally from the outer neurovascular capsule are fibrous annular plates, often erroneously called rings (they do not circle the disc). Because the number of anterior and lateral plates is greater than the number of posterior plates, the nucleus in the lumbar spine is positioned slightly posteriorly within the disc. Between the fibrous outer annulus and the inner fluid nucleus is a transition zone consisting of a loosely arranged collection of fibrous tissue that is highly deformable and acts as a buffer between the nucleus and annulus. The nucleus pulposus is a mucoid protein that binds approximately three times its weight in water and allows for distribution of forces.

3. Describe the functions of the intervertebral disc.

Rather surprisingly, because of its extensive water content, the disc is not a shock absorber. Instead, the muscles of the spine are responsible for shock absorption. The functions of the intervertebral disc include the following: (1) It provides space and position for the segment to allow for the nerve root to pass through the foramen without compromise. (2) It permits, guides, and restrains motion in all directions, with the nucleus acting as an incompressible ball while the ligamentous annulus (90% type I collagen) restrains the nucleus and prevents excessive motion within the segment.

4. What position facilitates disc nutrition?

Side lying or lying on the back with the knees bent and the back flat facilitates nutritional pressure changes. Approximately 80% of the nutrition absorbed within a night’s rest occurs within the first hour of rest. Therefore, by resting during the lunch hour and again at the end of the workday as well as at night, it is possible to more than double the nutrition to the disc.

Side lying is of value if the knees are drawn up so as to flatten or slightly round the back. However, the moment the back assumes lordosis, it loads the posterior disc, restricting its ability to imbibe nutrient fluids through the cartilaginous end plate. Likewise, prone lying is not recommended unless there is a large, firm pillow beneath the abdomen to prevent the formation of lordosis.

5. Describe the innervation of the disc.

The recurrent sinu-vertebral nerve and a gray ramus communicans from the sympathetic chain innervate the disc. They penetrate the outer capsule and may extend as far as the second or third annular lamella.

6. What is the source of discogenic low back pain?

Peng et al. studied the histologic characteristics of the painful disc. They noted the formation of a zone of vascularized granulation tissue from the nucleus pulposus to the outer part of the annulus fibrosus. Nerve growth was found deep into the annulus fibrosis and nucleus pulposus following the zone of granulation tissue in painful discs. Immunoreactive nerve fibers (such as substance P, neurofilament 2000, and vasoactive intestinal peptide) were more extensive in painful discs than in control discs. Annular tears noted at the periphery of discs were associated with this increased vascular granulation tissue, and these fibers may be the source of discogenic low back pain.
7. What are some of the anatomic structures associated with mechanical dysfunction of the facet joint, and how might they be a source of mechanical pain?

With regard to the facet joint, there are five common conditions that can lead to pain and disability.

1. **ACUTE SYNOVITIS/HEMARTHROSIS**
   As with any synovial joint, an acute strain to the facet joint may result in effusion and even bleeding into the joint. The strained joint is painful, which causes its muscles to act as involuntary stabilizers, holding the joint against unguarded motion to facilitate initial healing. However, if the joint is held in this position for more than 1 or 2 days, because of pain or the fear of pain, the cross fibers of collagen in the capsule will begin to create capsular stiffness, resulting in a capsular pattern or restriction. Additionally, if there were a hemarthrosis present, adhesion can be expected to form from the fibrinogen in the resolving blood clot.

2. **STIFFNESS**
   As stated previously, stiffness can occur after an acute injury, resulting from collagen cross binding or the deposition of fibrous adhesions after the injury.

3. **MECHANICAL BLOCK**
   Occasionally, most typically at L4/5 in the male, a joint may become painlessly locked in side-bending, following stooping to pick up an object. The exact cause of this “locking” can only be speculated, but it could be caused by a torn or separated meniscoid (all lumbar facets have menisci), a free fragment of articular cartilage, or simply roughness between degenerative joint surfaces.

4. **PAINFUL CAPSULAR ENTRAPMENT**
   On occasion (more commonly in the cervical spine), a sudden awkward movement may result in an acute one-sided pain that prevents the patient from holding the spine erect. In fact, any movement toward the pain that slides the superior facet downward seems to cause acute discomfort. In these circumstances, one can only assume that the facet capsule has become “stuck” between the articular surfaces. The fact that an isometric contraction of the multifidus muscles or a rotation, gapping technique can often produce immediate relief tends to support this hypothesis.

5. **DEGENERATIVE ARTHROSIS**
   Joint degeneration is a fact of age, no doubt hastened by misuse and abuse as well as genetic factors. Arthritic joints are stiff and painful, especially in the early morning.
   It is of interest to note that some spinal segments are hypermobile and perhaps unstable. This should be considered mostly a ligamentous condition, although laxity of the facet capsules may play a small role.

8. Describe the articular receptor distribution in the spine.
   - **Type I**—Postural receptors such as Ruffini’s corpuscles (greatest in the cervical spine) sense joint position; they have a low threshold and are slow to adapt.
   - **Type II**—Dynamic receptors such as Golgi-Mazzoni fat pads (deep seated in synovium) sense movement; they have a low threshold but adapt rapidly.
   - **Type III**—Inhibitory receptors are found in the outer layers of the facet capsules, in associated ligaments, and in the deep layer of the multifidus.
   - **Type IV**—Nociceptive receptors have a high threshold; they are nonadapting and chemosensitive.

9. Which structure is most commonly involved in the patient with low back pain?
   Regardless of the primary source of pain—disc, facet, or sacroiliac—the muscles will always be involved, whether voluntarily in a protective manner or involuntarily to guard against low back pain. However, they may also be the primary source of pain after unaccustomed overuse (eg, the first day of spring gardening). The most common cause of initial low back pain would be injury of the facet joints, followed by ligamentous weakness, sacroiliac strain, and ligamentous pain from the outer annulus. Pain may also develop from ligamentous instability in an unstable segment that is often adjacent to a stiff segment.

10. Describe the outcomes of physical therapy for acute low back dysfunction.
   Only in the area of acute low back pain (with no specific diagnosis) have satisfactory outcomes been established. The treatments determined to be effective were, in descending order, manipulation, patient instruction, and exercise. Cook et al. (2013) compared the effectiveness of early use of thrust and nonthrust manipulation during the first two visits of physical therapy in patients with mechanical low back pain. Both groups improved with either thrust or nonthrust manipulation. Based on a systematic review of the evidence, spinal manipulation performed by physical therapists is safe and resulted in improved clinical outcomes for patients with low back pain.
11. How does a therapist determine when manipulation of the spine for mechanical low back pain is indicated?

Clinical research studies that have demonstrated the effectiveness of thrust and nonthrust manipulation for treatment of LBP used a clinical decision-making framework that incorporates an impairment-based approach. A framework to consider would be the following:

- Rule out red flags and assess for yellow flags
- Assess pain location and behavior
- Assess patient expectations
- Assess impairments—Mobility deficits with active spinal mobility and passive intervertebral motion testing and pain provocation with passive accessory intervertebral motion testing guide the therapist to determine the location of focus for the manipulation, direction, intensity, and speed of force application.
- Classify patient—Hypomobility with concurrent pain in the low back and/or buttock with or without symptoms in the thigh

12. Discuss the potential sources of pain associated with dysfunction of the disc.

Several researchers have found nerve endings in the outer two to three layers of the disc. Furthermore when the disc degenerates to the degree that it becomes engorged with blood vessels in an effort to repair the disc, sympathetic nerves accompany the blood vessels. Substance P, a pain facilitator, has also been found in degenerative discs.

Early back pain, particularly that associated with developing instability, is mostly from the disc, is usually felt in the back and buttocks, and is of a deep and vague nature, often poorly localized. When the disc herniates, one source of pain may be from the mechanical strain on the outer fibers of the annulus. If the prolapse places pressure on a nerve root, a sharper radicular radiating pain may pass from the back into the leg from compression of the dorsal root ganglia. With initial nerve root pressure, there is little pain because it appears that the nerves first have to become engorged and sensitized. Thus nearly 30 minutes may pass from the initial, sharp low back pain (tearing of the annulus) to the onset of radicular leg pain (pressure on the nerve root). Chemical irritation from inflammatory agents of the nociceptive fibers of the outer annulus may also cause pain. Other anatomic structures associated with the disc that are innervated and may cause nociception include the posterior longitudinal ligament (PLL), dural root sleeve, and dural sheath. Discogenic pain is mediated by the sinu-vertebral nerves; it reaches the rami communicans through the L2 spinal ganglion. The pain may also take another route through the sympathetic nervous system.

13. Does disc herniation result from weakness and damage to the annulus (outside in) or from pressure pushing the disc outward (inside out)?

The first change noted with discography is that the nucleus deforms and starts to "leak" or move laterally. The inner annulus has few fibers, like the loose-knit weave of a woolen sweater. It can be stretched considerably without tearing. The fibrous annulus, however, has many fibers. It is more like a cotton shirt, having little elasticity before tearing. Although the inner annulus may degenerate, tears begin at the outer annulus and spread inwardly, eventually allowing the nucleus to deform. The outer annulus is approximately three times as vascular as the capsule of the knee and thus can heal, as postmortem specimens have shown. Therefore, determining which patients have an outer annulus injury can aid in selection of the appropriate therapy to promote healing and prevent herniation. Glycosaminoglycan turnover within the annulus requires approximately 500 days; collagen turnover is even slower. Healing, if possible, is still remarkably slow.

14. At what levels do cervical spondylosis most typically occur?

The prevalence of cervical spondylosis is as follows: C5/C6 > C6/C7 > C3/C5 > C7/T1. These changes affect 70% of the population by age 70.

15. At what levels do lumbar disc prolapse most commonly occur?

The prevalence of lumbar disc prolapse usually occurs in the following order: L4/L5 > L5/S1 > L3/L4 > L2/L3 > L1/L2.

16. In the thoracic spine, what are the most common levels of dysfunction that present with clinical symptoms?

The junctional sites T1/2, T12/L1, and T4/5 are the most common levels of dysfunction.
17. Describe a classification of disc herniations.

**DISC PROTRUSION (ANNULAR FIBERS INTACT)**
- Localized annular bulge (usually laterally)
- Diffuse annular bulge (usually posteriorly and bilaterally)

**DISC HERNIATIONS (ANNULAR FIBERS DISRUPTED)**
- Prolapsed (nucleus has migrated through the inner layers but is still contained)
- Extruded (nucleus has broken through the outermost layer)
- Sequestered (nucleus has broken from the disc and is in the spinal or intervertebral canals)

18. Does spontaneous disc resorption occur? What are the proposed mechanisms?
Results reported by Kawaguchi et al. maintain that regression of herniated discs is a process of general tissue repair and remodeling observable in a range of disc herniations rather than a specific autoimmune response.

19. What is the effect of facet angle on disc herniation?
A study by Karacan et al. showed a positive correlation in patients with lumbar disc herniation and asymmetry to sagittalization of facet joints. They noted these alterations were more prominent in the taller patients. Park et al. found that the degree of facet tropism and disc degeneration might be considered a key factor when distinguishing the development of far lateral lumbar disc herniation from posterolateral lumbar disc herniation. A direct relationship between the extent of the degree of facet tropism and the extent of disc herniation was not seen. Other studies by Hagg and Farfan found an unclear relationship between facet tropism and disc degeneration.

20. What is the incidence of disc herniation?
The incidence of disc herniations cannot be answered for the simple reason that it is now believed that most disc herniations do not hurt. Computed tomography (CT) scans of the lumbar spine in asymptomatic subjects with no history of other than minor back discomfort indicate that the rate of disc herniation is 39%. A similar study by Weisel showed 50% of abnormalities on CT scans in asymptomatic hospital workers. Disc protrusions are seen in 24% of asymptomatic patients.

21. What are the common causes of radiculopathy?
Neurologic signs arising from the lumbar spine most commonly occur in middle age, are more prevalent in men, and are typically a result of disc herniations, whereas neurologic signs arising from the cervical spine occur later in life, are more prevalent in women, and result from lateral foraminal stenosis caused by osteophytes from the lateral interbody, osteoarthrosis of the facet joints, and perhaps some disc material along with shortening and thickening of the ligamentum flavum.

22. Describe the classic presentation of disc herniations at various spinal levels.

<table>
<thead>
<tr>
<th>Level</th>
<th>Nerve Root</th>
<th>Dermatome</th>
<th>Myotome</th>
<th>Reflex</th>
</tr>
</thead>
<tbody>
<tr>
<td>C2/C3</td>
<td>C3</td>
<td>Anterior neck and posterior neck</td>
<td>Lateral neck press</td>
<td>None</td>
</tr>
<tr>
<td>C3/C4</td>
<td>C4</td>
<td>Nape and anterior shoulder</td>
<td>Shoulder shrug</td>
<td>None</td>
</tr>
<tr>
<td>C4/C5</td>
<td>C5</td>
<td>Deltoid anterior arm to base thumb</td>
<td>Biceps</td>
<td>Biceps</td>
</tr>
<tr>
<td>C5/C6</td>
<td>C6</td>
<td>Lateral arm thenar eminance, thumb</td>
<td>Wrist extensors</td>
<td>Brachioradialis</td>
</tr>
<tr>
<td>C6/C7</td>
<td>C7</td>
<td>Posterior arm to index, long, and</td>
<td>Triceps</td>
<td>Triceps</td>
</tr>
<tr>
<td></td>
<td></td>
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<td></td>
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</tr>
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</tr>
<tr>
<td></td>
<td></td>
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</tr>
<tr>
<td>T12/L1</td>
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<td>Iliac crest and groin</td>
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<td>Psoas</td>
<td>None</td>
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<td>L2/L3</td>
<td>L3</td>
<td>Anterior lower thigh and shin</td>
<td>Quadriceps</td>
<td>Knee jerk</td>
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<tr>
<td>L3/L4</td>
<td>L4</td>
<td>Medial calf and big toe</td>
<td>Tibialis anterior</td>
<td>Knee jerk</td>
</tr>
<tr>
<td>L4/L5</td>
<td>L5</td>
<td>Lateral leg and anterior foot</td>
<td>Extensor hallucis</td>
<td>Extensor digitorum</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>longus</td>
<td>brevis</td>
</tr>
</tbody>
</table>
23. Describe the natural history of disc disease.
In 90% to 95% of patients, spinal pain (which often is disc-related) resolves in 3 to 4 months. Lumbar disc herniations are quite common, and most cases have a favorable prognosis. Approximately 45% of patients demonstrate resorption of the herniation over time. In Norway, Weber randomly denied surgery to half of the patients selected for surgery by good and fair criteria (not as liberal as in the United States). At the end of the first year, those who had surgery scored twice as well on assessment as those who did not. By 3 years, however, there was no significant difference between the two groups. Five-year follow-up examination also found no difference.

24. Which is more successful for acute disc herniation—surgery or conservative care?
Evidence-based treatment guidelines for acute LBP and a review of thirteen national guidelines by Koes (2010) recommend early and gradual activation of patients, the discouragement of prescribed bed rest, spinal manipulation, and screening for psychosocial factors that would be risk factors for chronicity. Surgery has not been determined to be more successful than conservative care, as indicated by guideline recommendations and published literature.

25. Is disc degeneration associated with low back pain?
In the past, studies of asymptomatic individuals have demonstrated that there is little or no correlation between low back pain and disc pathology. More recently, several large population studies have demonstrated that disc degeneration is associated with LBP. Cheung et al. demonstrated by visualization on MRI that the risk of back pain increased directly with increasing disc degeneration. De Schepper et al. showed a similar association with annulus collapse from disc narrowing visualized on x-rays. Other degenerative changes, such as radial fissures, end plate damage, and disc extrusions, have been linked with pain. The variations in the literature may be based on the variety of structural changes that can occur with degeneration such as annular tears, herniation, annular collapse, Schmorl’s nodes, dehydration, and annular bulging.

26. Describe the Impairment-Based Classification System (IBCS) and International Classification of Functioning (ICF) and associated findings in a patient likely to present with discogenic pain.
1. IBCS: Lumbar and related leg pain that centralizes with repeated movements (specific exercise)  
   ICF: Low back pain with related (referred) lower extremity pain
   Findings:
   • Low back and leg pain that moves below the knee
   • Extension syndrome
     Symptoms centralize with lumbar backward bending
     Symptoms peripheralize with lumbar forward bending
   • Lateral shift
     Visible frontal plane deviation of the shoulders relative to the pelvis
     Symptoms centralize with side glide and backward bending

2. IBCS: Lumbar radiculopathy that does not centralize with repeated movements  
   ICF: Classification: Acute, subacute, or chronic low back pain with radiating pain
   Findings:
   • Low back with associated radiating leg pain that tends to travel beyond the knee
   • Poor tolerance to weight-bearing postures (ie, sitting or standing)
   • Symptoms alleviated with traction
   • Lower extremity paresthesias, numbness, and weakness may be reported
   • No lumbar movements centralize symptoms
   • No directional preference noted with history or clinical examination to alleviate lower leg pain
27. Discuss the role of manipulation and manual therapy in the treatment of disc herniation.

Manual therapy has no direct role in the reduction of disc herniations because neither traction nor manipulation has been shown to reduce the disc. However, manual therapy has been demonstrated to be effective by relaxing the muscles and allowing for movement in the segment.

Manipulation has not been shown to reduce disc herniation. However, Maitland grade I and II oscillations may help reduce discomfort and pain, thereby promoting return to active function. More physical techniques involving stretching and thrust may be of value at the neighboring stiff segments to increase motion and thus improve overall function of the spine, lessening the strain on the level with the disc herniation.

28. What is the effect of rehabilitation after disc surgery?

A 2011 systematic review of the literature by Wilco et al. provided the following conclusions:

- Exercise programs starting four to 6 weeks post surgery seem to lead to a faster decrease in pain and functional disability compared with no treatment, and high-intensity programs lead to a faster decrease in pain and functional disability than low-intensity programs.
- There is no evidence that these active programs increase the reoperation rate or that patients need to have their activities restricted after first-time lumbar surgery.
- It is still unclear the exact components that should be included in rehabilitation programs.
- High-intensity programs seem to be more effective, but they could also be more expensive.
- Therefore, cost-effectiveness analysis should be performed to assess whether intensive rehabilitation programs, if started early after surgery, lead to a reduction in costs in terms of less health care utilization or earlier return to work.
- Future research should also focus on the implementation of rehabilitation programs in daily practice.
- The effect of active rehabilitation on the main outcome of functional status is clinically significant in the short term and over the long term.

29. How does exercise relieve back pain?

1. Repetitive motion gates pain. For example, repetitive motion (eg, pendulum exercises) centralizes the pain to the shoulder, relaxes spasm, enables more motion, and hastens recovery.
2. If the pain is from an intradiscal source, repetitive motion may alter the chemical balance. T2-weighted MR studies showed a definite increase in disc water content after repetitive backward bending but no reduction in the size of the protrusion.
3. Extension places a higher stretch on the facet joint capsules than forward bending. Placing the hands in the small of the back and using them as a fulcrum mobilize the facet joints.
4. Repetitive motion enables a patient to get over “fear of movement” and undoubtedly relaxes muscle splinting, thus improving function, decreasing load on the disc, and allowing for earlier return to function.
5. Motion performed repetitively may reduce swelling around the nerve and thus the pressure that may cause ischemia.

30. What is the definition of spinal instability?

According to Panjabi, spinal range of motion can be divided into neutral- and elastic-zone components. Instability is thought to occur when neutral-zone motion increases beyond normal (thus decreasing the elastic zone). Stabilization of the spine is thought to revolve around three subsystems: passive, active, and neutral. When either of these subsystems is impaired, aberrant movement can occur and lead to pain or disability. Rehabilitation should be directed at all three of these systems to increase function and decrease spine pain.

31. Describe the innervation of the facet joints and types of afferent nerve fibers.

The innervation of the facet joints is a branch of the posterior primary ramus, which supplies the skin and muscles to the back. A deep branch arises near the facet joint and innervates that joint, with a larger branch supplying the joint below and another branch traveling to the level above (perhaps only in the lumbar spine). Thus, the facet joints on their larger posterior surface have in
common with most other joints a triple level of innervation. The anterior innervation is by the recurrent branch of the sinu-vertebral nerve that arches over the intervertebral foramen to supply the ligamentum flavum and the anterior facet joint capsule.

32. What muscles increase abdominal tone and pressure for stabilization of the lumbar spine?
The oblique and transverse abdominal muscles are important contributors to abdominal tone, while the multifidus muscle provides stabilization for the posterior spinal structures.

33. Discuss the significance of the multifidus muscle.
The multifidus arises from the mamillary process just lateral to the facet joint and then passes upward and medially, attaching to the adjacent facet joint capsule and to the capsule above before inserting into the spinous process one and two levels above. Acting unilaterally, it tends to bend the spine to the same side and rotate it to the opposite side. Acting bilaterally, it extends the spine. Because the multifidus inserts into the capsules of the facet joints, it tends to pull the capsule out of the way, helping to prevent capsular impingement. As one of the deepest muscles in the back, it is considered to be a primary stabilizer. The multifidus may be damaged during laminectomy or fusion. Even at 5 years after surgery, extensive damage may still be present. It is important that clinicians are able to target the multifidus when prescribing motor control exercises because it plays a significant role in lumbar stability. Decreased lumbar multifidus muscle activation is associated with the presence of factors predictive of clinical success with a stabilization exercise program. Hebert et al. studied the degree of transversus abdominis and lumbar multifidus activation by ultrasound imaging while looking at the prognostic factors associated with a successful stabilization exercise program. The factors included a positive prone instability test, age < 40 years, aberrant movements, straight leg raise > 91°, and presence of lumbar hypermobility. Significant relationships were identified between decreased LM muscle activation and the number of prognostic factors present. A positive prone instability test and segmental hypermobility were associated with decreased LM muscle activation. These findings provide evidence for the clinical importance of targeting the lumbar multifidus muscle for motor control exercises.

34. What are the effects of dynamic lumbar stabilization exercise programs after discectomy?
One study demonstrated that after a microdiscectomy, a 4-week postoperative exercise program can improve pain relief, disability, and spinal function. The exercise program, designed by a physical therapist, concentrated on improving the strength and endurance of the back and abdominal muscles and the mobility of the spine and hips. The program included aerobic exercise and strengthening exercises, such as curl-ups and leg lifts, to strengthen the erector spinae musculature. A prospective randomized clinical trial by Yilmaz et al. demonstrated with controls that dynamic lumbar stabilization exercises are an efficient and useful technique in the rehabilitation of patients who have undergone microdiscectomy. Outcomes were good for relief of pain and for functional parameters such as strength of the trunk, abdominal, and lumbar spine muscles.

35. What are the effects of disc herniation and surgery on proprioception and postural control?
Leinonen studied proprioception and postural control in patients before and after discectomy. These variables were found to be diminished when comparing postoperative patients with chronic low back pain caused by disc herniation versus healthy controls.

36. What are the functional results and risk factors for reoperation after disc surgery?
It has been documented that factors including sedentary occupations, exposure to considerable vibration (such as from driving a motor vehicle), cigarette smoking, previous full-term pregnancies, physical inactivity, increased body mass index (BMI), and a tall stature are associated with symptomatic disc herniations. Increased fitness levels and strength have been noted to reduce the risk of disc rupture. Lack of regular physical exercise was a significant predictor for reoperation, while gender, age, BMI, occupation, or smoking did not hold as much significance as regular exercise. The reoperation rate within 5 years for patients having disc surgery has varied in studies from 7% to 35.3%.

37. What are the effects of surgery on pain, spine mobility, and disability?
In a prospective cohort study from the Maine Lumbar Spine Study (Atlas et al.), 400 patients with sciatica caused by lumbar disc herniation were treated either surgically or nonsurgically, and then assessed in 10-year follow-up visits. Changes in the modified Roland back-specific functional status scale
favored surgical treatment throughout the follow-up period. However, work and disability status at 10 years did not demonstrate a difference between those treated surgically from those treated nonsurgically. A cross-sectional survey by Hakkinen et al. reviewed the results of patients’ status post lumbar disc herniation surgery. They found that 2 months after the operation median leg pain had decreased by 87% and back pain by 81%. However, moderate or severe leg pain was still reported in 25% and back pain in 20% of the patients. Hakkinen noted that pain, decreased trunk muscle strength, and decreased mobility were still present in a considerable proportion of patients 2 months after surgery.

38. What are the effects of low back pain, disc herniation, and surgery on the lumbar multifidus?

Functional instability with motor coordination impairments of the core musculature, including the multifidus, has been the clinical assumption after an episode of low back pain because of disc herniation or other impairments as well as surgery. In patients with first-episode low back pain, ultrasound measurements indicate that multifidus muscle recovery does not occur spontaneously when the low back pain resolves. Disc herniation has been associated with selective atrophy of type I fibers while the atrophy of type II fibers was more frequent and severe. Findings such as decreased size of type 2 muscle fibers and core/targetoid and/or moth-eaten changes in the type 1 muscle fibers have been noted. Selective type 2 muscle fiber atrophy has been found during intraoperative muscle biopsies. Pathologic changes were present in 88% of patients before surgery. Rantanen et al. reviewed the intraoperative biopsies of patients with disc herniation and 5-year follow-up biopsies. Results showed that patients who have a positive outcome have positive changes in the structure of the multifidus. After a posterior surgical approach, biopsies of the multifidus showed significantly more signs of denervation in the tissue than before surgery. Clinicians should progress patients with a spinal stabilization and conditioning program with emphasis on retraining the motor control of the deep abdominal and multifidus muscles.

BIBLIOGRAPHY


1. Which of the following is associated with impairment-based classifications in patients with discogenic low back pain?
   a. Poor sitting tolerance
   b. Poor standing tolerance
   c. LBP and buttock pain that does not move below the knee
   d. Symptoms centralize with forward bending

2. What finding that will predict clinical success of a stabilization program is associated with the following prognostic factors: positive prone instability test, age <40 years, aberrant movements, straight leg raise >90°, and presence of lumbar hypermobility?
   a. Decreased transversus abdominus function
   b. Normal transversus abdominus activation
c. Decreased multifidus function
d. Normal multifidus activation

3. Which of the following statements is true regarding rehabilitation after lumbar disc surgery?
   a. Exercise programs starting 4 to 6 weeks post surgery lead to faster pain reduction and less functional disability compared with no treatment.
   b. Low-intensity programs lead to a faster decrease in pain and functional disability than high-intensity programs.
   c. Specific components of exercise, such as stabilization, have been shown to be more effective than general aerobic conditioning.
   d. There is significant evidence that active exercise programs increase the reoperation rate after first-time lumbar surgery.
1. **What is lumbar spinal stenosis (LSS)?**
   LSS can be defined as any narrowing of the lumbar spinal canal, nerve root canals, and/or intervertebral foramina that may encroach on the nerve roots of the lumbar spine. LSS can become a painful and potentially disabling condition in affected individuals.

2. **How is LSS classified?**
   There are two means of classification commonly used to describe patients with LSS; one is based on the anatomic location of the narrowing, the other on the etiology of the narrowing.
   
   **ANATOMIC CLASSIFICATION**
   - Lateral stenosis—narrowing that occurs within the lumbar intervertebral foramina and/or the nerve root canal, causing encroachment around the spinal nerve as it exits
   - Central stenosis—narrowing that occurs within the spinal canal, causing encroachment around the nerve roots of the cauda equina housed within the dural sac
   
   **ETIOLOGIC CLASSIFICATION**
   - Primary stenosis—narrowing caused by a congenital malformation or defect in postnatal development. Only about 10% of cases of lumbar stenosis can be considered to be primary stenosis.
   - Secondary stenosis—narrowing resulting from acquired conditions such as degenerative changes, spondylolisthesis, fractures, and postsurgical scarring. The most common cause of secondary stenosis is degenerative changes. Secondary stenosis may occur in individuals who already have a degree of primary stenosis.

3. **What are the most common structural changes associated with LSS?**
   The majority of cases of LSS occur secondary to degenerative changes. Facet joint arthrosis and hypertrophy, bulging and thickening of the ligamentum flavum, loss of disc height and posterior/lateral bulging of the intervertebral disc, and degenerative spondylolisthesis are the most common changes contributing to LSS. Other, less common causes of secondary stenosis include fractures, postoperative fibrosis, tumors, and systemic diseases of the bone, such as Paget’s disease.

4. **Is lumbar stenosis a common problem?**
   Yes; LSS is a common cause of low back pain, particularly in older adults. It is the most common reason for undergoing spinal surgery in individuals over the age of 65. Because of increases in life expectancy and improved diagnostic technology, rates of diagnosis of LSS and rates of surgery have increased substantially in the past several decades.

5. **How will the typical patient with lumbar stenosis present clinically?**
   In general, because degenerative changes are the predominant cause leading to LSS, affected individuals are typically older than age 50 with a long history of low back pain. Most patients will have symptoms of pain and/or numbness in one or both legs. Chronic nerve compression may lead to diminished lower extremity reflexes and strength or sensation deficits. Lumbar range of motion, particularly in extension, will be limited and painful, often reproducing leg symptoms. Symptoms tend to be posture-dependent, worsening with spinal extension and improving with flexion. Because of this, patients will generally feel better in a sitting position and worse when standing or walking.

   Several authors have provided information that helps quantify the impact of these various clinical indicators on the ultimate diagnosis of LSS. Katz et al. and Fritz et al. identified several clinical findings with associated sensitivity (Sn) and/or specificity (Sp) values for establishing the diagnosis of LSS. These findings are as follows: age over 65 — Sn = 0.77; pain below buttocks — Sn = 0.88; no pain
when seated — $Sp = 0.93$; pain with flexion — $Sn = 0.88$; sitting is the best position — $Sn = 0.89$; standing/walking are worst positions — $Sn = 0.89$.

Sugioka et al. sought to develop a score-based prediction rule to assist with diagnosis of LSS from self-report items only. The final predictors of LSS and associated risk scores are shown in Table 56.1. Scores of 7 or more yielded positive likelihood ratios of 1.90 to 3.91.

<table>
<thead>
<tr>
<th>Predictive Variable</th>
<th>Assigned Risk Score</th>
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<tr>
<td>Age:</td>
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<tr>
<td>60–70</td>
<td>2</td>
</tr>
<tr>
<td>&gt;70</td>
<td>3</td>
</tr>
<tr>
<td>Onset of symptoms &gt;6 months</td>
<td>1</td>
</tr>
<tr>
<td>Symptoms:</td>
<td></td>
</tr>
<tr>
<td>Improve with bending forward</td>
<td>2</td>
</tr>
<tr>
<td>Improve with bending backward</td>
<td>–2</td>
</tr>
<tr>
<td>Exacerbate while standing up</td>
<td>2</td>
</tr>
<tr>
<td>Intermittent claudication present</td>
<td>1</td>
</tr>
<tr>
<td>Urinary incontinence present</td>
<td>1</td>
</tr>
</tbody>
</table>

Last, Konno et al. also developed a score-based prediction rule for diagnosing LSS. In this analysis, the researchers also included items from the physical examination. Their final predictors for LSS and associated risk scores are shown in Table 56.2. For this prediction rule, total risk scores of 7 or higher yield the following diagnostic indices: sensitivity = 0.93, specificity = 0.72, positive likelihood ratio = 3.31.

<table>
<thead>
<tr>
<th>Predictive Variable</th>
<th>Assigned Risk Score</th>
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<tbody>
<tr>
<td>Age:</td>
<td></td>
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<td>&gt;70</td>
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<td>Absence of diabetes</td>
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<td>Symptoms:</td>
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<tr>
<td>Improve with bending forward</td>
<td>3</td>
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<tr>
<td>Exacerbate while standing up</td>
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<tr>
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<td>Examination:</td>
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<tr>
<td>Symptoms induced by having patients bend forward</td>
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</tr>
<tr>
<td>Symptoms induced by having patients bend backward</td>
<td>1</td>
</tr>
<tr>
<td>Good peripheral artery circulation</td>
<td>3</td>
</tr>
<tr>
<td>Abnormal Achilles tendon reflex</td>
<td>1</td>
</tr>
<tr>
<td>SLR test positive</td>
<td>–2</td>
</tr>
</tbody>
</table>

6. Why do patients with LSS feel worse when standing than when sitting? Standing places the lumbar spine in a position close to full extension. Extension of the spine causes further narrowing of the spinal canal. In individuals without stenosis, this narrowing is tolerated without difficulty; however, patients with stenotic narrowing tend to have worse symptoms when standing or when standing and walking. Sitting causes flexion in the spine and therefore will generally reduce the symptoms of individuals with LSS.

7. Are there other factors that exacerbate symptoms for patients with LSS? Axial compression, as is experienced during weight-bearing, also creates increased narrowing of the spinal canal and may exacerbate the symptoms of LSS. Research has demonstrated that the narrowing effects of axial compression are similar in magnitude to those of spinal extension. This helps explain why walking can be difficult for patients with lumbar stenosis. Walking involves extension of the spine and creates increased compressive forces.
8. What is neurogenic claudication?
Neurogenic claudication is defined as poorly localized pain, paresthesias, and cramping of one or both lower extremities of a neurologic origin; symptoms are worsened by walking and relieved by sitting. There can be many causes of claudication; therefore the key distinguishing feature of neurogenic claudication is its neurologic origin—mechanical irritation of the cauda equina. The symptoms of neurogenic claudication are often the reason that an individual with LSS is prompted to seek medical treatment.

9. Are there other conditions that might be confused with lumbar stenosis?
Other conditions that have been confused with LSS include osteoarthritis of the hip, vascular claudication (with peripheral arterial disease), unstable spondylolisthesis, and lumbar intervertebral disc herniation. Other frequent concomitant problems that may mandate additional differential diagnosis for those with lumbar stenosis may include diabetic neuropathy, other peripheral neuropathies, other lower extremity (LE) disorders, iliacus arterial involvement, and spinal tumors.

10. How can LSS be differentiated from other conditions with a similar presentation in the clinic?
The postural-dependency of symptoms (ie, better with flexion or sitting; worse with extension, standing, and walking) is a unique characteristic of patients with LSS. A thorough history, including exacerbating and relieving positions or activities, will often reveal this posture-dependent nature of symptoms (see question 5 for more details).

Clinical tests have also attempted to capitalize on the postural-dependency of stenosis symptoms to differentiate spinal stenosis from other conditions with similar symptoms. A “bicycle test” has been described in which the patient first pedals in an upright, seated position with the lumbar spine in extension and then with the spine in a flexed position. If the individual pedals farther in the spine-flexed position, the test is considered positive for LSS. Walking tests have also been described for use in differential diagnosis. The patient walks on a level surface in an upright posture and also in a slumped or flexed posture. If the patient can walk farther with the spine flexed, the test is considered positive for LSS. A variation on this test, called the Two Stage Treadmill Test (TSTT), compares walking on a level treadmill versus an inclined treadmill (15% incline). The incline of the treadmill causes the patient to flex the spine while walking and usually will improve walking capacity in patients with LSS. Earlier onset of symptoms and prolonged recovery with level treadmill walking yields a specificity (Sp) of 0.95 and a positive likelihood ratio (+LR) of 14.5. A longer total walking time on the inclined treadmill versus the level treadmill yields an Sp of 0.92 and a +LR of 6.5.

Additionally, a thorough lower extremity neurovascular assessment should be performed, often including assessment of lower extremity muscle strength, reflexes, sensation, and pulses. The clinician should also look for trophic changes of the skin and nails and may opt to perform an ankle-brachial index test. This neurovascular assessment will help with differentiation from other neuropathic or vascular conditions.

11. Are diagnostic imaging studies or electrodiagnostic studies helpful in confirming a diagnosis of LSS?
Diagnostic imaging modalities are generally used to confirm the diagnosis of LSS. The most commonly used tests are the following:

- Magnetic resonance imaging—MRI is one of the most commonly used imaging studies to confirm a diagnosis of LSS. The anterior-posterior diameter of the spinal canal or the cross-sectional area of the dural sac can be measured to determine the extent of narrowing.
- CT scan—the CT scan is also commonly used to assess the diameter or cross-sectional area in the same manner as described for the MRI.

It is important to remember that the presence of stenosis with imaging is prevalent in the asymptomatic aging population; therefore correlation of imaging findings with clinical presentation is essential.

Examination with electrodiagnostics is occasionally used to assist in the diagnosis of LSS. According to Haig et al., the following factors can help with this diagnosis:

- Miniparaspinal mapping with a one side score >4: sensitivity = 0.3; specificity = 1.0
- Fibrillation potential in limb muscles: sensitivity = 0.33; specificity = 0.88
- Absence of tibial H-wave: sensitivity = 0.36; specificity = 0.92
- Composite limb and paraspinal fibrillation score: sensitivity = 0.48; specificity = 0.88
12. Are plain film x-rays helpful in the diagnosis of LSS?
Plain film x-rays can show the degenerative changes such as osteophytes and disc degeneration that are often the cause of LSS. Lateral views can demonstrate the diameter of the intervertebral foramina. However, plain film x-rays are limited in their usefulness by their inability to image the central spinal canal and the soft tissue changes that may contribute to LSS.

13. What are the most common impairments and functional limitations found in patients with LSS?
The most common impairments found during the examination of the patient are restrictions in spinal range of motion. Side-bending is often limited bilaterally; lumbar extension may be quite limited and reproduce or intensify the patient’s symptoms. Lumbar flexion is also frequently limited in range but will often somewhat relieve the symptoms. Deficits in vibratory or pinprick sensation in one or both lower extremities can occur, along with strength or reflex deficits. Many patients will have a positive straight leg raise test. Another common area of impairment is the hip joint. Restricted range of motion, particularly in extension, and weakness of the hip extensors and abductors are common findings. The most widespread functional limitation in patients with LSS is diminished walking tolerance because of leg symptoms that are relieved by sitting, as well as limitation in prolonged standing postures.

14. Describe the surgical procedure for a patient with LSS.
Surgical treatment of LSS is performed to relieve compression on the contents of the central and lateral spinal canals. The most common surgical procedure for patients with LSS is a decompression laminectomy in which portions of the vertebral arch are removed to reduce compression of the lumbar spinal nerves. Sometimes a fusion, with or without instrumentation, will also be performed, although this is usually only done if there is evidence of spondylolisthesis along with the spinal stenosis.

15. Should a patient with LSS have surgery?
The decision to pursue surgery should be determined based on careful and shared decision making between providers and patients, with heavy emphasis on patient preferences.
Clinical outcomes are generally good for surgical treatment, with high percentages of patients expressing satisfaction and reporting improvement early postoperatively. However, these outcomes tend to deteriorate with time, with only about 60% to 70% of patients satisfied after 4 to 7 years and between-group clinical outcomes for those treated with surgery versus conservative care diminishing within 2 to 8 years after surgery.
Sufficient literature describing positive clinical outcomes from various nonsurgical approaches to care is available and often with no reported adverse outcomes. These nonsurgical approaches typically include treatments such as aerobic exercise, stretching, strengthening, mobilization/manipulation, and patient education.
Based on the known risks of surgical complications and the stable nature of spinal stenosis, current recommendations are that patients should be offered a rigorous trial of physical therapy care before pursuing surgery. Patients who do not improve should be well informed of the potential risks and benefits of surgery, including the fact that benefits from surgery will most likely diminish over time.

16. Will the symptoms of lumbar stenosis continue to worsen over time?
Research to date shows that LSS is a generally stable condition. Although some patients will deteriorate over time, this is not inevitable, and large percentages of patients can maintain or improve their condition with time.

17. Will epidural steroid injections help patients with lumbar stenosis?
Epidural steroid injections have been frequently recommended as a nonsurgical option for patients with LSS. Some patients will receive short-duration benefits from epidural steroid injections. The effectiveness of injections in reducing symptoms beyond a couple of weeks, however, is less likely.

18. What is the best physical therapy treatment for patients with lumbar stenosis?
Numerous treatment options have been proposed for use by physical therapists in the treatment of patients with LSS. Flexion-oriented exercises are advocated to capitalize on the postural dependency of symptoms of spinal stenosis. General conditioning activities are useful and may include stationary cycling, aquatic exercise, and walking as tolerated by the patient. Any strength or flexibility deficits identified
during the physical examination should be addressed. Manual therapy (including mobilization, manipulation, and stretching) targeting the thoracic and lumbopelvic spine regions and hips may also be helpful.

19. Should traction be used in the treatment of patients with LSS?
Pelvic traction has been recommended for the treatment of LSS in an attempt to relieve compression that results from the pathology. Although traction may be helpful for pain reduction in some patients, it should be combined with more active forms of therapy to improve function.

20. Can deweighted treadmill ambulation help patients with LSS?
Deweighted treadmill ambulation uses a harness-and-traction device to provide a vertical traction force during ambulation on a treadmill. The traction force reduces the axial compression associated with weight bearing and may permit some individuals with LSS to walk with reduced symptoms of neurogenic claudication. This treatment technique may hold promise for selected patients with stenosis because it provides the benefit of traction while keeping the patient active and exercising. However, in a randomized trial by Pua et al. that compared exercise plus cycling versus exercise plus deweighted treadmill walking, both groups achieved similar pain and disability outcomes at 6 weeks. Therefore the use of deweighting should be considered as a potential rehabilitation tool on a case-by-case basis, based on clinical response, and, in general, should not be viewed as superior to cycling as a part of a comprehensive treatment program.

21. Is it possible to identify patient-centered factors that predict better versus worse outcomes from surgery for lumbar stenosis?
Although many researchers have investigated this question, there is some conflicting information in the literature. Some identified predictors of worse surgical outcomes to date include depression, worse emotional health, smoking, Workers’ Compensation, anxiety, life dissatisfaction, higher BMI, longer duration of leg pain/symptoms, cardiovascular comorbidity, scoliosis, other disorders influencing walking ability, predominant back pain (>leg pain), prior lumbar surgery, history of psychiatric disease, female gender, and low baseline disability.

In addition to not having these identified predictive factors for worse outcomes, the following factors have been identified as predicting better surgical outcomes: greater central canal stenosis, good or above average self-rated health, younger age, lower duration use of analgesics preoperatively, greater preoperative disability, more ambitious preoperative expectations related to pain and functional improvements, and no lifting required at work.

22. Are there published studies documenting patient outcomes with defined physical therapy treatment approaches?
Many studies are now available demonstrating positive clinical outcomes of care for patients treated with interventions often provided by physical therapists, including aerobic exercise, stretching, strengthening, aerobic exercise, and mobilization/manipulation. Some studies also include traction, physical modalities, or lumbopelvic orthoses. Selected studies are described as follows:

- Simotas et al. reported on the results of 49 patients treated with a program of physical therapy (flexion-oriented exercises) and epidural steroids. After 3 years, 9 patients (18%) had undergone surgery, 12 patients (24%) reported no change in symptoms, 23 patients (47%) had some amount of improvement, and 5 patients (10%) experienced worsening of symptoms.
- Murphy et al. reported clinically meaningful long-term improvements in disability and pain for 57 patients treated with manipulation, neural mobilization, flexion exercises, and lumbar stabilization training in a prospective observational cohort.
- Pua et al. conducted a trial that included 68 patients. Both treatment groups received lumbar flexion exercises, lumbar traction, and thermal modalities. One group also performed deweighted treadmill walking, and the other performed stationary cycling. Although there were no between-group differences, both groups improved from baseline to 6 weeks.
- Ammendolia and Ngai reported on clinically meaningful improvements in pain and disability for a cohort of 49 patients after a 6-week program, including manual therapy (soft tissue and neural mobilization, manipulation, lumbar flexion-distraction, and muscle stretching), home exercises, and self-management strategies.
- Cambron et al. included 60 patients in a pilot RCT that investigated optimal dosages of spinal manipulation. The treatment groups receiving a total of 12 and 18 manipulations over 6 weeks
demonstrated significant within-group improvements up through 6 months in symptom severity, and the group receiving 18 manipulations had significant within-group improvements in physical function.

- Whitman et al. conducted an RCT with 58 patients that compared an individualized approach (impairment-based manual therapy interventions to the thoracic spine, lumbopelvic spine, and lower extremities; deweighted treadmill walking; and abdominal retraining exercises) to walking, a standardized flexion exercise, and a subtherapeutic ultrasound program. A greater proportion of patients in the pragmatic, individualized program reported recovery at 6 weeks versus the flexion exercise/walking group. Although both groups demonstrated positive outcomes over the 24-month follow-up, improvements in disability, satisfaction, and treadmill walking tests favored the individualized treatment group at all follow-up points.

- Delitto et al. conducted a multisite, randomized controlled trial that included 169 surgical candidates with lumbar stenosis. Patients were treated with either surgical decompression or a 6-week, well-defined, twice weekly physical therapy (PT) intervention program, including lumbar flexion exercises, patient education, general conditioning (cycling or treadmill walking), and individualized lower extremity strengthening and flexibility exercises. Those patients undergoing physical therapy intervention had similar outcomes to those treated with surgical decompression at the 2-year follow-up.

23. Should patients with lumbar stenosis wear a brace or corset?

The use of a rigid corset to limit spinal extension or a soft corset for general support has been recommended. A soft corset may provide a measure of relief for patients. A more rigid brace, although effective in limiting or preventing extension, is often cumbersome and restrictive for the patient and should likely be reserved for those individuals not responding to other forms of nonoperative treatment.

24. How should the outcomes of treatment for patients with lumbar stenosis be measured?

Measuring the effectiveness of any treatment for LSS is an important consideration. Patient-reported measures, such as the Oswestry or Roland Morris disability scales, as well as the condition-specific Swiss Spinal Stenosis Questionnaire, are useful for documenting functional limitations and disability. The measurement of walking tolerance, usually conducted on a treadmill or with a 6-minute walking test, is an important assessment and monitoring tool because it measures the most common and troublesome functional limitation in these patients.

25. Does stenosis occur in the cervical spine as well?

Yes; stenotic narrowing can and does occur in the cervical spine. Similar to the lumbar spine, the narrowing may occur laterally, in the intervertebral foramen, or centrally, in the spinal canal. The etiology may be primary (ie, congenital), secondary to degenerative conditions, or a combination of these two factors. The presence of congenital stenosis of the central canal in the cervical spine is a particular concern for participants of collision sports, such as football. The normal sagittal plane diameter of the spinal canal in the cervical region is 17 to 18 mm. The diameter of the spinal cord is about 10 mm. If the sagittal plane diameter of the canal is diminished, the safety margin within the canal is compromised, and symptoms of compression of the spinal cord may result. As with lumbar stenosis, the presence of cervical stenosis on imaging may be present in absence of clinical symptoms, therefore mandating corroboration of clinical and imaging findings.

26. What symptoms will a patient with cervical stenosis exhibit?

The symptoms of lateral and central cervical stenosis differ substantially. Lateral cervical stenosis typically results in compression of the cervical nerve root and produces symptoms of radiculopathy. Central cervical stenosis may compress the spinal cord, resulting in a condition termed cervical myelopathy. Symptoms of radiculopathy include neck and upper extremity pain and paresthesia in a dermatomal pattern. There may also be complaints of upper extremity muscle weakness in the affected arm. Symptoms of myelopathy are often subtler, particularly in the early stages. Neck pain is not always present. Unsteadiness in gait or clumsiness is often an early symptom. Wasting of the intrinsic hand muscles is common. An extrasegmental distribution of paresthesia in one or both hands and feet may be present, followed by a perception of weakness. Gait disturbances can become severe, significantly interfering with functional activities and safety.
27. What is the typical clinical presentation for patients with central cervical stenosis?
The signs of central cervical stenosis (myelopathy) are those of upper motor neuron, or long tract, disorders. Clinical signs may include weakness with spasticity, clonus, present Hoffmann and Babinski signs, hand withdrawal reflex, and an inverted supinator sign. Vibratory sensation is typically diminished in the lower extremities, and both upper and lower extremity reflexes may become hyperactive. CervICAL range of motion is typically restricted in all planes. Lhermitte’s sign (spinal pain and/or radiating extremity pain and paresthesias with forced cervical flexion or extension) may be present. Spurling’s sign is expected to be negative, and manual cervical traction will not have any effect on symptoms. According to Cook et al., at least 3 of the following five clinical tests helps rule in cervical myelopathy: 1) gait deviation, 2) present Hoffmann’s test, 3) + inverted supinator sign, 4) present Babinski test, 5) age >45 y (specificity = 0.99; CI = 0.97–0.99; + LR = 30.9; 95% CI = 5.5–181.8).

28. Is treatment by a physical therapist helpful for cervical myelopathy?
Nonsurgical care is often recommended for patients with mild myelopathy, although scant evidence exists investigating the impact of treatment by a physical therapist for this disorder. Intermittent cervical traction is often recommended as a potentially helpful intervention. In a small case series, Browder et al. reported improvements in pain and disability for 7 patients with cervical myelopathy who were all treated with intermittent cervical traction and thrust manipulation targeting the thoracic spine. No adverse events or outcomes were reported.

29. Is surgery recommended for patients with cervical myelopathy?
The disorder is considered to be progressive in nature and potentially disabling, but those with cervical myelopathy (CM) often experience long periods of stable neurologic status between episodes of exacerbation. Conservative nonsurgical treatment is often recommended for patients with mild cervical myelopathy. In contrast, surgical management is typically considered for those with moderate to severe CM. Although one prospective RCT demonstrated no significant differences between groups (surgery versus no surgery) at 10 years, several other studies conclude that those with moderate to severe CM who undergo surgery experience improved outcomes over those who do not. Performing surgery early in the course of the condition is believed to lead to a better long-term outcome. Laminotomy or laminoplasty is typically performed to increase the dimensions of the central spinal canal and may be accompanied by cervical fusion.

BIBLIOGRAPHY


CHAPTER 56 QUESTIONS

1. Current best evidence leads us to include the following in our physical therapy–related care for patients with lumbar spinal stenosis:
   a. Double and single knee to chest exercises, quadruped “cat and camel” (flexion and extension) exercises, hot packs, and electrical stimulation.
   b. Repeated flexion exercises, double and single knee to chest exercises, and abdominal (“core”) retraining
   c. Aerobic exercise (cycling and/or walking), manual therapy to the lower quarter, lower quarter and abdominal muscle stretching and strengthening, and lumbo-pelvic flexion exercises
   d. Traction, electrical stimulation, cycling, and thermal modalities
   e. None of the above

2. Which findings are most helpful in making a clinical diagnosis of lumbar spinal stenosis?
   a. Standing and walking aggravate symptoms, and sitting eases symptoms; younger age; low back pain only (none in the lower extremities)
   b. Standing and walking aggravate symptoms, and sitting eases symptoms; older age; presence of pain, paresthesia, and/or cramping into one or both lower extremities below the buttocks
   c. Sitting aggravates symptoms and standing and walking ease symptoms; older age; low back pain only (none in the lower extremities)
   d. Sitting aggravates symptoms and standing and walking ease symptoms; younger age; presence of pain, paresthesia, and/or cramping into one or both lower extremities below the buttocks

3. Which answer below includes disorders that may present similarly to lumbar spinal stenosis in the clinical exam? Pick the best answer.
   a. Deep vein thrombosis, spondylitis of the lumbar spine, post-polio syndrome
   b. Femoral neck fracture, mechanical low back pain, hip and knee osteoarthritis
c. Iliacus arterial disorder, fibular nerve adverse neural dynamics, mechanical low back pain

d. Hip osteoarthritis, peripheral arterial disease, lumbar radiculopathy, peripheral neuropathy, spinal tumors

4. What are common symptoms for a patient with cervical stenosis?

a. Neck pain and headache, referral of symptoms into the thoracic region, absence of symptoms below the acromioclavicular joint

b. Unsteadiness and clumsiness in gait, lower motor neuron changes in the upper and lower extremities, and an absence of neck pain

c. Neck and upper extremity pain and paresthesia, intrinsic muscle wasting of the hands, upper extremity weakness, absence of symptoms beyond the upper quarter

d. Neck and upper extremity pain and paresthesia, upper extremity muscle weakness, unsteadiness in gait, wasting of the intrinsic muscles of the hands

5. Which answer below includes a list of clinical signs that are most helpful in identifying the presence of central cervical stenosis?

a. Gait deviation, present Hoffmann’s test, positive inverted supinator sign, present Babinski test, age > 45 yrs, hand withdrawl reflex

b. Age range of 20–40, present Hoffman’s test, absent Babinski test, positive inverted pronator sign, hand withdrawl reflex, relief with manual cervical traction

c. Gait deviation, present Babinski test, absent Hoffman’s test, abnormal lower extremity neural dynamics tests, positive inverted pronator sign

d. Age > 45 yrs, neck pain and headache, referred symptoms to the medial scapular border, lower motor neuron findings in both the upper and lower extremities
1. How is spondylolisthesis measured and graded?
   Anterior slippage of one vertebral body on an adjacent body is graded I, II, III, and IV, according to the percentage of slippage of the posterior margin of one vertebral body relative to the next inferior body (25%, 50%, 75%, and 100%, respectively). For example, a grade II spondylolisthesis indicates a 25% to 50% subluxation of the vertebral body, a grade III indicates a 50% to 75% translation, etc. Grade V spondylolisthesis indicates the superior vertebral body slips entirely forward on and anterior to the subjacent body, known as spondyloptosis. These measurements are made on a standing lateral radiograph. Subsequently, Taillard has described a method that expresses the slippage in terms of percentage of the anteroposterior diameter of the distal segment (measurement of forward displacement of the anterior aspect of one vertebral body on the one below, divided by the anteroposterior dimension of the distal vertebral body). This method is considered to be more accurate and more reproducible than the Meyerding method. Both methods, however, continue to be commonly used.

2. What is sacral inclination?
   Sacral inclination, also known as sacral tilt, is the angle of displacement of the sacrum from the vertical. It is the measurement of the angle between a line drawn along the posterior margin of the first sacral vertebra and its bisection with the true vertical. This angle is measured on a standing lateral radiograph. The sacrum is angled anteriorly in normal upright standing postures, but the angle tends to decrease as the listhesis increases. The sacrum becomes more vertical with progressive listhesis.

3. What is the slip angle?
   Also known as sagittal roll, sagittal rotation, and angle of kyphosis, the slip angle is considered to be the most sensitive indication of potential segmental instability. This angle is measured between a
line drawn perpendicular to the S1 and S2 vertebral bodies (through the disc space) and a line drawn along the superior end plate of the L5 body. The inferior end plate can also be used; however, the inferior end plate is more commonly deformed with degenerative changes and is more difficult to consistently identify than the superior end plate. This measurement is critical because it is felt to be the most sensitive measurement to predict progression of the listhesis.

4. What are the types (classifications) of spondylolistheses and the etiologies of each?

![Types of spondylolistheses. Also see Table 57.1.](image)

5. What is the rate of occurrence of isthmic spondylolisthesis?
The incidence of spondylolysis or spondylolisthesis was found to be 4.4% at age 6. These children were followed into adulthood, where the incidence increased to 6%. The degree of slip was seldom found to progress after adolescence because listhesis generally occurs concurrently with fatigue fracture. Interestingly, the spondylolysis was never found to be symptomatic in the population studied by Fredrickson et al. yet was reported by Micheli to be the most common causes of low back pain in adolescents.

6. Does spondylolysis always progress to spondylolisthesis?
No; nearly 50% of patients who present with isthmic spondylolysis do not progress to spondylolisthesis. Generally speaking, listhesis occurs at the time of fatigue fracture. If the anterior translation has not occurred during childhood or adolescence, it seldom occurs in adulthood. In the longitudinal study by Fredrickson et al., progression of listhesis was found to be unusual after it was initially appreciated in childhood or adolescence. Degenerative spondylolisthesis can occur without isthmic defect because of long-standing segmental instability and/or intervertebral disc degeneration. In a similar fashion, dysplastic spondylolisthesis can occur without a disrupted pars interarticularis. Some cases of dysplastic spondylolisthesis occur with intact, but attenuated posterior elements.

7. Should neurologic compromise be anticipated with spondylolisthesis?
Lower extremity radicular pain in the child is said to be more representative of dysplastic spondylolisthesis, suggesting irritation of the L5 or S1 nerve root, although isthmic spondylolisthesis can present similarly. Isthmic defects are often filled with fibrocartilaginous tissue that is formed in response to the stress fracture and resultant listhesis. The exiting nerve root then is stretched across this fibrous defect, causing nerve root irritation and associated lower extremity radicular symptoms.
### Spondylolysis and Spondylolisthesis

<table>
<thead>
<tr>
<th></th>
<th>Congenital</th>
<th>Isthmic</th>
<th>Degenerative</th>
<th>Traumatic</th>
<th>Pathologic</th>
<th>Iatrogenic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td>Females &gt; males, 2:1</td>
<td>Males &gt; females, 2:1</td>
<td>Females &gt; males, 5:1</td>
<td>Seldom seen</td>
<td>Data unavailable</td>
<td>Data unavailable</td>
</tr>
<tr>
<td><strong>Age at onset</strong></td>
<td>Congenital</td>
<td>Adolescents</td>
<td>Generally after 40 years</td>
<td>Seldom seen</td>
<td>Data unavailable</td>
<td>Data unavailable</td>
</tr>
<tr>
<td><strong>Comorbidities</strong></td>
<td>Questionable accelerated DDD; SBO</td>
<td>Questionable accelerated DDD; SBO</td>
<td></td>
<td>Seldom seen</td>
<td>Local or systemic bone disease</td>
<td>Lumbar stenosis</td>
</tr>
<tr>
<td><strong>Most common level</strong></td>
<td>L5–S1</td>
<td>L4–5</td>
<td>L4–5</td>
<td>Seldom seen</td>
<td>Data unavailable</td>
<td>Data unavailable</td>
</tr>
<tr>
<td><strong>Progressive or not?</strong></td>
<td>Yes, often caused by attenuation of posterior elements</td>
<td>Uncommon after stress fracture occurs</td>
<td>Gradually, occasionally will autofuse, related to degenerative changes</td>
<td>Data unavailable</td>
<td>Data unavailable</td>
<td></td>
</tr>
<tr>
<td><strong>Associated symptoms</strong></td>
<td>Radicular pain is common, associated with nerve root stretch and irritation</td>
<td>LBP, may present with radiculopathy in adulthood; “crisis” LBP at onset possible</td>
<td>Neurogenic claudication associated with stenosis</td>
<td>Data unavailable</td>
<td>Data unavailable</td>
<td></td>
</tr>
<tr>
<td><strong>Cause</strong></td>
<td>Dysplastic pars interarticularis, dysplastic sacral facets may have sagittal or axial orientation, attenuates with weight bearing</td>
<td>Stress fracture of pars interarticularis, more common in young gymnasts and football linemen</td>
<td>Degenerative lumbar spine and inter-vertebral disc changes</td>
<td>Traumatic, producing fracture other than at pars interarticularis that follows listhesis</td>
<td>Tumor, infection, osteoporosis</td>
<td>Excessive decompression of facets at time of surgery</td>
</tr>
</tbody>
</table>

DDD, Degenerative disc disease; SBO, spina bifida occulta; LBP, low back pain.
Neurologic signs can occur in the form of lower extremity weakness, paresthesia, and occasional bowel or bladder incontinence. Cauda equina symptoms are most commonly associated with degenerative spondylolisthesis as the nerve roots are stretched across the defect as they exit the sacral foramina. Degenerative spondylolisthesis often results in neurogenic claudication signs consistent with associated spinal stenosis.

8. Does the isthmic pars defect heal when treated?
If diagnosed early and treated with rigid bracing for up to 6 months, the results have been favorable according to radiographic evaluation, clinical improvement in symptoms, and bone scan criteria. Bone scan evaluation is typically used to determine whether the fatigue fracture is sufficiently acute to warrant immobilization. Steiner and Micheli describe 78% clinical results as good or excellent with the use of a modified Boston brace in grade I spondylolisthesis. The brace was used for 6 months full time, while allowing for a flexion exercise program and sports participation within limits of pain complaints. Other reports indicate that the pars defect rarely heals, but clinical results tend to be favorable in response to bracing for the acute spondylolytic crisis. Early in the immobilization period, aggressive abdominal strengthening and stabilization exercises are begun, with return to activity, including sports, as tolerated. It should be noted that literature indicates that clinical results and return-to-activity orders are not significantly different in patients who demonstrate solid bony healing compared with those whose stress fractures go to fibrous healing. Clinical results are similar.

9. What associated morbidity is seen with spondylolisthesis?
When isthmic spondylolisthesis occurs at the L5–S1 level, local instability is rarely seen. However, when it occurs at L4–L5, instability is more common because of the absence of the contribution of the iliolumbar ligament to segmental stability. The fiber direction of this ligament allows for it to offer strong support against anterior translation of the L5 vertebra, but it has no insertion to L4, leaving the L4 body without this strong passive support. This level has been shown to be hypermobile or unstable into the third or fourth decade of life. Degenerative changes occurring over the next several years tend to stabilize the progressive isthmic spondylolisthesis but may lead to degenerative spondylolisthesis later in life.
There is some belief that intervertebral disc degeneration occurs more rapidly in the presence of isthmic spondylolisthesis than in a population without spondylolisthesis. Studies indicate a more rapid rate of degenerative processes after age 25 in patients with isthmic spondylolisthesis than in those without the disorder. Evidence of spina bifida occulta is seen four times more often in patients with isthmic and dysplastic spondylolisthesis compared with uninvolved control populations. Reported incidence of spina bifida occulta in dysplastic spondylolisthesis is 40%, with the normal incidence in adults without dysplastic spondylolisthesis being 6%. Spina bifida contributes to the predisposition to isthmic defects in involved patients by the dysplastic posterior elements not forming completely, leaving the posterior ring inherently weak. Transitional anatomy (sacralization of the L5 segment or lumbarization of the S1 segment) is four times more likely in those with degenerative spondylolisthesis than in age-matched controls.

10. How is spondylolisthesis diagnosed radiologically?
Standard x-rays are adequate in diagnosing a spondylolisthesis. A lateral lumbar spine film will demonstrate the listhesis of one segment on the next distal segment. Studies have confirmed that the anterior translation is greater in standing, weight-bearing films than in supine, nonweight-bearing films. Therefore some authors suggest both views be taken to demonstrate intersegmental motion. Lumbar spine oblique views are used to evaluate the integrity of the pars interarticularis. The well-described “Scotty dog” sign shows the presence of the fatigue fracture by a radiolucent area across the “neck” of the Scotty dog. Bone scan technology is used to diagnose an acute fatigue fracture of the pars or to differentiate local tumor or infection as the cause of symptoms. Neuroimaging studies (MRI or CT scan) are used to confirm suspicion of nerve root impingement associated with disc degeneration or the listhesis itself. Serial radiographs can be taken to assess for progression of listhesis. Repeat films are taken at 6- to 12-month intervals when spondylolisthesis is initially diagnosed and then after a greater interval if no progression is identified. Spondylosis, however, can be more difficult to diagnose before a stress fracture occurs. The single-photon-emission computed tomography (SPECT) scan is considered to be the most sensitive in detecting early metabolic changes that may preclude stress fracture. An uptake noted on this study indicates bone turnover, a response to stress reaction.

11. What are the basic principles of conservative management of spondylolisthesis?
Isthmic spondylolisthesis often presents with a “spondylolytic crisis”—acute low back pain—in a child or adolescent. When confirmed radiographically, bracing is recommended in the acute case, defined by...
active findings on CT or SPECT bone scan. When worn continuously for 3 to 6 months, the brace provides the pars defect an opportunity to heal. While still in the brace, specific trunk stabilization exercises are performed. The purpose of the exercises is to aggressively and functionally facilitate abdominal muscle contraction without causing segmental lumbar spine movement, which is undesirable during the healing stage because it may disrupt the healing pars. Attempts to restore “normal” lordosis through aggressive repeated extension activities, either standing or prone, are not indicated in treatment. A patient with spondylolisthesis may demonstrate a compensatory reduction in lumbar lordosis as a mechanism to limit the anterior translation stress involved with upright postures. Repeated lumbar extension exercises increase this stress, and have been shown to increase pain complaints in spondylolisthesis patients. As with many sources of mechanical low back pain, balance of flexibility of lower extremity muscles should be assessed and addressed. Patients with spondylolisthesis often demonstrate inadequate hamstring flexibility, resulting in increased shearing forces through the lumbar spine with movement. These shearing forces across an inherently weaker pars intraarticularis may contribute to low back pain symptoms.

12. What is the role of flexibility exercises in conservative treatment of spondylolisthesis?
Lower extremity flexibility exercises are an integral part of any complete low back rehabilitation program. Hamstring flexibility is often limited in patients with symptomatic spondylolisthesis. Hamstrings become tight reactively to produce and maintain a posterior pelvic tilt and subsequent reduction in lumbar lordosis, thereby reducing the anterior shear force of the lumbar spine vertebral body. An anterior pelvic tilt may be adopted, allowing the ilioptosa and rectus femoris to adaptively shorten. These opposing forces of reactively shortening lower extremity musculature increase the overall stress and tension within the muscular system of the lumbosacral spine and pelvis, resulting in increased symptoms of pain and dysfunction. Hamstring spasm that is unresponsive to conservative measures is often relieved by decompression of the L5 or S1 nerve roots at the time of surgery.

13. What are the surgical indications in the child or adolescent with spondylolisthesis?
Surgical indications for children and adolescents with spondylolisthesis are fairly well established. According to Amundson et al., surgical indications include:
- Persistence or recurrence of major symptoms in spite of aggressive conservative management for at least 1 year
- Tight hamstrings, persistently abnormal gait, or postural deformities that are unrelieved by physical therapy
- Sciatic scoliosis, or lateral shift
- Progressive neurologic deficit
- Progressive slip beyond grade II spondylolisthesis, even when asymptomatic
- A high slip angle (greater than 40–50 degrees), because high slip angles are considered to be the most sensitive indicator for progressive listhesis and instability
- Psychological problems associated with postural deformity, or gait deviations associated with a high-grade listhesis

Outcomes from in situ fusion in adolescents with spondylolisthesis have been well documented with very favorable results. Children and adolescents generally fare well after posterolateral fusion procedures, usually returning to unrestricted activity. It is interesting to note that most symptoms associated with spondylolisthesis in the child and adolescent are associated with segmental instability; therefore in situ fusion can adequately control the symptoms without requiring nerve root decompression. Current recommendations are that decompression without fusion should not be performed “in patients under age 40, and is rarely needed in the child and adolescent years.”

14. List the surgical indications for adults with spondylolisthesis.
- Isthmic spondylolisthesis that becomes symptomatic as an adult
- Segmental instability or neurologic compromise post trauma
- Associated with progressive degenerative changes
- Degenerative spondylolisthesis associated with progressive symptoms
- Persistent symptoms lasting more than 4 months, interfering with patient’s quality of life
- Progressive neurologic deficits
- Progressive weakness
- Bowel/bladder dysfunction
- Sensory loss
- Reflex loss
15. *What types of surgical interventions are available for treatment of spondylolisthesis?*

In situ fusion has long been the procedure of choice for symptomatic spondylolisthesis, both in adolescent and adult populations. Commonly, reduction procedures have been complicated by nerve root symptoms, radiculopathy, and occasional motor deficits from disrupting the nerve root during surgery. There is further controversy regarding the need for nerve root decompression accompanying posteriorlateral fusion in the adult with isthmic spondylolisthesis. Some authors claim decompression is necessary in the presence of any neurologic deficit, while others claim that decompression is effectively accomplished by a successful fusion. Wiltse et al. claim that the fibrocartilage mass decreases in size with successful posterolateral fusion, effectively decompressing the nerve root. All authors, however, agree that the presence of bowel or bladder dysfunction and a motor deficit that is significant enough to cause loss of normal ambulation are reasons to decompress the offending nerve root during surgery. Decompression without fusion is often proposed in the treatment of degenerative spondylolisthesis as well. However, wide laminectomy and involvement of the facet joints with decompression without fusion may result in an increased prevalence of associated instability and could lead to iatrogenic spondylolisthesis. As a result, substantial debate continues regarding the efficacy of decompression alone versus decompression with fusion in degenerative spondylolisthesis.

16. *Are athletes more prone to spondylosis than others?*

Generally speaking, the prevalence of spondylosis is not greater in athletes than in nonathletic populations; however, there are some sports in which there appears to be a higher incidence. Various reports have suggested a higher incidence of spondylosis in sports such as gymnastics, diving, wrestling, weight lifting, throwing sports, and volleyball. Note all of these sports include frequent, loaded, or extreme ranges of motion of the lumbar spine (flexion or extension). These end range, externally loaded, and repeated ranges of motion are felt to contribute to the higher incidence in these athletes than in other populations.

17. *By what criteria can an athlete or nonathlete return to activity after being diagnosed with spondylolisthesis?*

There is no absolute agreement on criteria for return to sport or other activity with diagnosis of spondylolisthesis. Various authors have suggested functional or time-based criteria (see Hopkins and White). Others determine activity more clinically. Presence of increasing or peripheralizing pain with activity and persistent or increasing weakness are indications to limit or reduce activity. Most literature suggests that bracing in the presence of an acute injury (metabolically active scan) is indicated, generally allowing activity within the brace. A Boston or other brace is felt to be unnecessary in absence of positive findings on bone scan but may be used if helpful in controlling symptoms associated with activity. Postoperatively, these patients are managed as other postlumbar fusion patients are, following the same physiologic guidelines as any patient status post instrumented lumbar spine fusion.

**Bibliography**


1. What are the major types of scoliosis?
   - Functional scoliosis—This may be caused by muscle spasm (secondary to lumbar or thoracic injuries) or leg length discrepancy (which causes a lateral shift in the spine). Functional scoliosis resolves with healing of the lumbar or thoracic injuries or correction of the leg length discrepancy.
   - Structural scoliosis—This type of scoliosis is usually idiopathic.
   - Congenital scoliosis—This type is caused by vertebral anomalies and is much less common than the other two types of scoliosis.

2. What is the incidence of idiopathic structural scoliosis?
   Idiopathic scoliosis affects 1 to 4 people per thousand. Curves >20 degrees are 7 times more common in females than in males, and curves >30 degrees have a 10:1 female-to-male ratio. The incidence drops to about 0.3% overall for curves >20 degrees. Idiopathic scoliosis usually occurs in adolescents between 11 and 14 years of age.

3. What are the possible causes of idiopathic scoliosis?
   The role of genetics has been debated. Family history is not helpful in determining curve magnitude. Some form of multifactorial or autosomal dominant inheritance seems to be involved, although most recent research suggests a polygenic inheritance pattern. The proprioceptive system and equilibrium imbalances, possibly related to asymmetry in the brainstem, also may be implicated.

4. Describe the clinical presentation of idiopathic scoliosis.
   Curves do not straighten when the trunk is flexed forward (Adam’s test). Structural curves exhibit rotatory components during forward flexion, and the patient’s symptoms usually include rib hump or asymmetry in the trunk, referred to as the angle of trunk rotation (ATR). The ATR is easily measured with the scoliometer.

5. What types of initial screening processes appear as most effective in determining whether aggressive active treatment, such as bracing or surgery, is needed?
   The most common method for determining the presence and severity of scoliosis is Adam’s test, combined with the use of the scoliometer. Moiré photography is moderately effective in screening for scoliosis but is much less cost-effective. Two-tier screening programs, which include both an initial screener and a secondary screener, tend to be the most effective in reducing false-positive diagnoses.

6. When is further evaluation of idiopathic scoliosis advisable?
   In general, patients with curves >15 to 20 degrees and a 5- to 7-degree ATR usually are referred for further follow-up by an orthopedist. Current data, however, recommend at least a 20-degree curve and 7-degree ATR.

7. Describe the Risser classification.
   The Risser classification uses ossification of the iliac epiphysis to grade remaining skeletal growth. Ossification starts laterally and runs medially. Ossification of the lateral 25% indicates Risser type 1; of 50%, Risser type 2; of 75%, Risser type 3; complete excursion, Risser type 4; and fusion to the ilium, Risser type 5. Growth in females is usually complete in Risser type 4.

8. Describe the King classification system.
   The King classification system describes curve types in idiopathic scoliosis, and the system helps determine surgical treatment.
   - Type I—primary lumbar and secondary thoracic curves
   - Type II—primary thoracic and secondary lumbar curves
   - Type III—thoracic curves only
   - Type IV—large thoracic curves extending into the lumbar spine
   - Type V—double thoracic curves
Recent studies have demonstrated some reliability problems with the King classification system. A newer system—the Lenke classification of adolescent idiopathic scoliosis—uses three components: curve type, lumbar spine modifiers, and sagittal thoracic modifiers. It is the most common system in use today for determining surgical intervention treatments. The Lenke system has recently been shown to be much more reliable than the King system.

9. Describe the rate of progression of idiopathic scoliosis.
Curve progression depends on curve size and Risser sign. For curves <20 degrees that are Risser type 0 or 1, progression occurs in 22% versus only 1.6% for curves above Risser type 2. For curves of 20 to 30 degrees and Risser type 0 or 1, progression occurs in 68% versus only 22% for curves above Risser type 2.

10. What treatment options are available for progressive idiopathic scoliosis?
Surgery and bracing have been the gold standards of treatment. There has recently been some research that shows that progressive inpatient rehabilitation programs, concomitant with the development of ongoing home programs and derived from this inpatient program, have been successful in controlling the progression of scoliotic curves.

11. When should bracing be considered?
Curves <20 degrees generally do not require bracing, particularly when patients are more mature (Risser types 3–5). Curves <30 degrees that progress 5 degrees or more over 12 months should be braced. For curves >30 degrees, bracing should be initiated immediately. Bracing is not indicated in skeletally mature patients.

12. Describe the bracing used for scoliosis. How long should the brace be worn?
The first brace, developed immediately after World War II by Blount et al., was named the Milwaukee brace. It was fairly cumbersome, made with stainless-steel bars, and fitted with side straps to reduce lateral deflection and rotation of the spine at the specific points of apexes of curves. Newer, more comfortable braces include the Boston brace (thoracolumbosacral orthosis [TLSO]), which appears to be the most effective; it is made of molded plastic and fitted to the patient. Boston braces enhance adherence to treatment protocols because of ease of use. Generally they must be changed once every 12 to 18 months, depending on the patient’s growth and body changes. Braces are most effective when worn 23 hours per day until skeletal maturity is achieved. The effectiveness of bracing is time-dependent: the more the brace is worn, the better the outcome.

13. What forces in braces reduce progression of scoliotic curves?
Computer evaluation of braces determined that the primary correction forces in braces are lateral. Muscle forces and longitudinal traction play minimal roles, if any. Reduction in hyperlordosis also is needed to reduce the curve.

14. What are the outcomes of major brace types in treating idiopathic scoliosis?
The Boston brace, Milwaukee brace, and Charleston bending brace are used most commonly to treat idiopathic scoliosis. Recent studies show that the quality of life scores are higher for Milwaukee and Boston braces than for the Charleston brace. For most curves, the Boston brace appears more effective at preventing curves from progressing, as defined by a lower rate of surgery. Surgical rates for the Charleston brace appear to be approximately 50% higher than for either the Milwaukee or the Boston brace. The greatest difference in outcome is found in King type III curves. King type I and II curves have fairly equal results with Charleston and Boston braces. Boston braces are most appropriate for curves with the apex below T8. Milwaukee braces are best used for curves with the apex above T7. Recent strides have been made in developing strap tension systems with strap transducers instrumented to the Boston brace. These tension systems allow for optimal prescribed levels of tensioning, so the patient may achieve the best curve correction along with a reduction in curve progression.

15. What curves respond best to bracing?
Curves without severe lumbar hyperlordosis, thoracic lordosis, or hyperkyphosis respond best to bracing. Risser type 0 curves respond best, whereas Risser type 4 or 5 curves rarely respond well. Double major curves respond less favorably to bracing than other curves.

16. How effective is bracing?
Over the years, the efficacy of bracing has been one of the most intensely debated subjects in the treatment of idiopathic scoliosis. Recent reports, however, indicate that the efficacy may be as high
as 74% to 81% in halting the progression of idiopathic structural scoliosis. In contrast, only 33% of patients do not progress without the use of bracing. Recent studies also show that wearing braces did not affect the quality of life in adolescents compared with observed counterparts. Other recent studies show that brace compliance and a high initial correction are strong indicators for bracing success.

17. What are the indications for surgical intervention?
   - Curves >50 degrees in skeletally mature patients
   - Curves progressed beyond 40 degrees in skeletally mature patients
   - Curves >30 degrees with marked rotation
   - Double major curves >30 degrees

18. Define “crankshaft phenomenon.”
   In a patient with an immature spine, correction of scoliosis with successful posterior fusion may be complicated by continued anterior vertebral body growth, which can increase the curve and vertebral rotation. This problem may be corrected with combined anterior and posterior fusion procedures if a skeletally immature patient must undergo surgery.

19. What type of correction can be expected with surgical intervention?
   Surgery in idiopathic scoliosis generally reduces the major coronal curve by approximately 50%, vertebral rotation by approximately 10%, and apical translation by an average of approximately 60%.

20. What is the most common form of surgical intervention in idiopathic scoliosis?
   Segmental instrumentation with multihook systems (eg, Cotrel-Dubousset system) is the most common approach. Fixation is posterior. For more advanced and rigid curves, both anterior and posterior fusions may be incorporated. Patients should be evaluated on an individual basis.

21. List the complications of surgical intervention for idiopathic scoliosis.
   - Migration of rods
   - Neurologic damage
   - Pseudarthrosis
   - Renal failure
   - Psychological stress
   - Blood loss
   - Failure of fixation
   - Infection
   - Respiratory distress

22. What types of treatments other than surgery or bracing have been shown to be effective?
   Numerous studies have demonstrated that lateral electrical stimulation (LES) and exercise, either in or out of the bracing, are ineffective. To date, no research has shown that chiropractic care is effective. Physical therapists have recently been used in progressive inpatient and immediate post-inpatient rehabilitation programs for scoliosis.

23. Describe the role of the physical therapist in screening and treating scoliosis.
   The physical therapist may train screeners, screen patients, and oversee preoperative and postoperative conditioning programs and progression in patient rehabilitation programs. Pain management, either before or after bracing or surgery, also may be needed.

24. Compare the costs of bracing and surgery.
   Most research shows that the costs of bracing and surgery are somewhat comparable. At the start of the new millennium, total surgical costs, which include preoperative and postsurgical care and bracing as well as other medical care, average approximately $50,000. These costs do not include screening. Overall costs would be decreased if screening were used with bracing. Cost estimates do not include loss of income, welfare, social programs, or other direct or indirect medical costs associated with surgical intervention.

25. What are the long-term curve progressions for surgical-treated versus brace-treated curves?
   After 22 years, brace-treated curves progressed 7.9 degrees versus 3.5 degrees for surgically treated curves.
26. What are the long-term (20 years or more) quality-of-life outcomes for surgery versus bracing treatment?
No correlation exists between curve size after treatment, curve type, total treatment time, or age at completion of treatment. Approximately 49% of those undergoing surgery, 34% of those treated with braces, and 15% of controls will have some limitation of social activities, mostly because of physical participation in activities or self-consciousness about appearance. Patients treated for scoliosis have about the same health-related quality of life as the general population.

27. What is the natural history of patients with untreated idiopathic scoliosis?
Untreated people with scoliosis are productive and function at a high level at 50-year follow-up. Back pain occurs in 61% compared with 35% of controls. However, of those with pain, 68% describe it as minor or moderate.

BIBLIOGRAPHY
1. What is the prevalence of thoracic spine pain and disability in children and adolescents?
   Prevalence estimates for thoracic spine pain vary widely. The point prevalence in adolescent 13- to 20-year-olds ranged from 4.0% to 41.0% and in children from 4.0% up to 72.0%, seemingly making children more likely to experience thoracic spine pain. There also appears to be a higher prevalence of thoracic spine pain in females as has been shown in other reports of musculoskeletal pain.

2. Describe the normal range of motion (ROM) of the thoracic spine.
   The rib cage and sternum attachments limit ROM of the thoracic spine. Inclinometry of T1–T12 indicates that the total range of sagittal plane motion is approximately 36 degrees (16 degrees of flexion and 20 degrees of extension from neutral posture). Frontal plane motion is approximately 44 degrees (24 degrees of right-side bending and 20 degrees of left-side bending from neutral posture).

3. Describe the preferred side-bending and rotation-coupling pattern of the thoracic spine.
   Systematic review of the literature to date shows variability in the coupling patterns of the thoracic spine. Study design and methodology variations may be to blame for some of the differences in findings. No consistent coupling pattern has been recognized whether the thoracic spine is flexed, extended, or in neutral position or whether axial rotation, side-bending, or upper extremity movement was initiated first. Even with recent advances in the accuracy of three-dimensional computed tomography assessments, consistency could only be found in the upper thoracic spine (T1–6), where axial rotation was coupled with side bending to the same side. The middle and lower thoracic spine (T6–L1) side bending occurred to the same and opposite direction as the axial rotation.
   Past premise has been that when the spine is neither flexed nor extended, side bending and rotation were coupled in opposite directions (right-side bending with left rotation). It had also been suggested that the coupling pattern was sensitive to which plane of movement was introduced first; Lee suggested that rotation and side bending couple to the same side in the thoracic spine when rotation is introduced first.

4. How accurate can we be with manual examination of the thoracic spine?
   Similar to the lumbar and cervical mobility assessment, intrarater-reliability findings are often variable but can reach substantial agreement in some studies while interrater reliability rarely exceeds fair. Passive physiologic intervertebral motion examination has shown agreement between 63.4% and 82.5%. Palpation for tenderness or pain typically increases the reliability measures. However, we should be cautious of false positives when relying heavily on the subjective report of pain provocation, especially in those patients with chronic widespread pain presentations.

5. What effects has thoracic spine manipulation been found to have?
   There is a growing body of evidence emerging on the study of treatment directed at the thoracic spine. A systematic review by Walser et al. in 2009 screened 242 articles pertaining to thoracic spine or cervicothoracic manipulation. Quality research is showing that thoracic spine manipulation has a positive effect on patients with shoulder or neck pain and disability. The exact mechanism by which these effects occur has not been determined, but accepted theories include a regional interdependence model that may also include neurophysiologic and other nonspecific effects.
6. **How many articulations are present on the typical thoracic vertebra?**

A typical thoracic vertebra has 12 separate articulations: 4 zygapophyseal articulations, 2 costotransverse articulations, 4 costovertebral articulations, and 2 body-IV disc-body articulations. At present, individual passive assessment of these components is likely to be fraught with difficulty and poor reliability.

7. **Describe the typical pattern of rib cage motion.**

The typical upper rib motion during respiration is termed pump handle (sagittal plane elevation), whereas lower rib motion is termed bucket handle (frontal plane flaring). Lee’s model suggests that during spinal flexion, the rib rotates anteriorly; posterior elements move superiorly and anterior elements move inferiorly. This pattern is termed internal torsional movement. During spinal extension, the opposite movement is proposed, with the rib rotating posteriorly; posterior elements move inferiorly and anterior elements move superiorly. This pattern is termed external torsional movement. This model has not been validated with in vivo motion studies. Various authors and one case report have outlined the potential clinical presentation and significant loss of this movement.

8. **Describe the cervical rotation lateral flexion (CRLF) test.**

The CRLF determines the presence of first rib hypomobility in patients with brachialgia. The test is performed with the patient in a sitting position. The cervical spine is rotated passively and maximally away from the side being tested (ie, rotation to the left to test the right side). In this position, the spine is gently flexed as far as possible, moving the ear toward the chest. A test is considered positive when lateral flexion movement is blocked. Lindgren and colleagues reported excellent interrater reliability ($K = 1.0$) and good agreement with cineradiographic findings ($K = 0.84$).

9. **Define thoracic outlet syndrome.**

Thoracic outlet syndrome (TOS) is perhaps the most controversial symptom complex in surgery. Even the use of established operation criteria before surgery results in the relief of symptoms in only 28% of patients undergoing first-rib resection. Diagnoses using the traditional positional provocation tests of the upper extremity are unreliable and result in a large number of false positives. Conservative therapy aimed at restoring function to the upper thoracic aperture in patients with TOS decreased symptoms and returned patients to work after intervention and at a 2-year follow-up. Therefore conservative management is advocated. Lower-level evidence (case series) describes treating a “subluxation” of the first rib with manual therapy techniques with subsequent reduction of symptoms attributed to TOS.

10. **Describe the typical pattern of movement and positional dysfunction of the thoracic spine and rib cage.**

In general, the upper two segments of the thoracic spine often have restricted ability to extend fully, resulting in a flexed (kyphotic) posture in this region. The T3–T7 segments often have restricted ability to flex and concurrent external rib torsional dysfunction, resulting in an extended (flat) posture in this region. The T8–T12 segments often have restricted ability to extend, resulting in a flexed (kyphotic) posture in this region.
11. Describe a classification system for thoracic spine and rib cage dysfunction.

Patients in whom specific mobilization is indicated have primary single segmental restriction of either flexion or extension, torsional rib cage dysfunction, and/or first-rib restriction. The immobilization category includes patients who require motion restriction. The rib subluxations are the primary candidates for this treatment, which is geared at using the patient’s muscle activity to restore normal symmetry and to avoid movement stresses in directions that promote asymmetry. Segmental thoracic hypermobility or instability also is placed in this category.

The nonspecific mobilization category does not imply gross mobilization but rather the treatment of multiple segments in the neutral (neither flexed nor extended) spine. Rib cage restrictions in either inhalation or exhalation also fall into this category.

12. Does osteoporosis frequently involve the thoracic spine?

Osteoporosis is associated with loss of bone mass per unit of volume. Loss of bone mass in the axial skeleton predisposes vertebral bodies to fracture, which results in back pain and deformity. An anterior wedge compression fracture is manifested by a decrease in anterior height, usually 4 mm or greater, compared with the vertical height of the posterior body.

13. What are the symptoms of thoracic osteoporosis? How are they treated?

Symptomatic osteoporosis presents as midline back pain localized over the thoracic or lumbar spine, the most common location for fractures. The treatment of osteoporosis is often complex and, in severely affected patients, should be coordinated with an endocrinologist. Treatment should include exercise, which has been shown to increase or slow the decline of skeletal mass. Weight-bearing activities should be emphasized.

14. What is the incidence of musculoskeletal dysfunction mimicking cardiac disease in the emergency department (ED)?

Musculoskeletal chest wall syndromes have been reported in as many as 28% of patients admitted to the ED with acute chest pain but without acute myocardial infarction.

15. A 35-year-old man presents with pain and stiffness in the thoracic region, which is worse in the morning. On physical examination you note limited chest expansion.

What should your differential diagnosis include?

Ankylosing spondylitis (AS) is a chronic inflammatory disease characterized by a variable symptomatic course. Back pain and stiffness are the initial symptoms in 81% of patients. In the thoracic spine, AS causes decreased motion at the costovertebral joints, reduced chest expansion, and impaired pulmonary function. Chest expansion is measured at the fourth intercostal space in men and below the breasts in women. The patient raises both hands over the head and is asked to take a deep inspiration. Normal expansion is ≥2.5 cm.

16. A 44-year-old man presents with pain in the right T7–T9 region just below the inferior lateral angle of the scapula. Further questioning reveals that the symptoms are worse 2 to 3 hours after a meal. What should your differential diagnosis include?

Pain from cholecystitis (inflamed gallbladder) typically occurs 1 to 2 hours after ingestion of a heavy meal with severe pain peaking at 2 to 3 hours. Pain from gallbladder disease is generally transmitted along T8 and T9 nerve segments. Right upper quadrant or epigastric pain is characteristic, but pain often is referred to the angle of the scapulae on the right side.
17. Can thoracic spine and rib cage musculoskeletal dysfunction mimic anginal pain?  
The T4–T7 thoracic segments frequently have been implicated as the source for initiation of pseudoanginal pain. The primary evidence is in the form of case reports and case series. Hamburg and Lindahl reported 6 cases of “anginal” pain relieved by manipulation of the midthoracic segments. In many cases, the primary symptoms of diabetic thoracic radiculopathy are severe abdominal and anterior chest pain with minimal back pain.

18. What is Scheuermann’s disease? Is it safe to use manual therapy in affected patients?  
Scheuermann first described the radiographic changes of anterior wedging and vertebral end-plate irregularity in the thoracic spine associated with kyphosis. The disease also is known as juvenile kyphosis, vertebral osteochondritis, and osteochondritis deformans juvenilis dorsi. Disc material herniated into the vertebral bodies (Schmorl’s nodes) is a common associated finding. Patients benefit from even slight increases in motion of the posterior elements at the involved segments. Despite the fact that the basic deformity is not “corrected,” maintenance and improvement in range of motion and function may be achieved.

19. Do postural abnormalities of the cervical and thoracic spine contribute to pain?  
Poor upper quadrant posture has been implicated as a source of neck and shoulder pain. Patients with more severe postural abnormalities of the thoracic, cervical, and shoulder regions have a significantly increased incidence of pain. In particular, patients with thoracic kyphosis and rounded shoulders reportedly have an increased incidence of cervical, interscapular, and headache pain.

20. Define T4 syndrome.  
T4 syndrome describes a group of patients with dysfunction within the T2–T7 segments. The clinical presentation includes various combinations of pain in the upper limbs and in the neck, upper thoracic, and scapular regions with cranial headaches. However, the T4 segment is nearly always involved. In addition, patients may report glove-like paresthesias and numbness in one or both hands, often nocturnal in nature. Differential diagnoses include systemic illness, polyneuritis, and nerve root compression. Typical examination findings include tenderness, asymmetry, and limited segmental range of motion and tissue thickening. Furthermore, posteroanterior pressure over the involved thoracic segment reproduces the symptoms. McGuckin (not peer-reviewed) reported 90 cases in which the syndrome occurred more frequently in women (4:1) than in men, with a typical presentation between 30 and 50 years. DeFranco and Levine reported two cases of apparent T4 syndrome of 6 to 12 month’s duration that were treated successfully by two sessions of T3–T4 manipulation. Treatment includes localized segmental mobilization and/or manipulation.

21. What role can the thoracic spine play in headaches?  
Dysfunction of the thoracic spine, in particular the upper five segments, has been implicated as the primary generator of headaches. Examination of the upper thorax in patients with headaches is warranted. Treatment using segmental mobilization and/or manipulation has been advocated. The mechanism for the referred pain to the head is unknown.

22. What symptoms may arise from or at least be affected by treatment directed at the thoracolumbar region?  
Dysfunction at the thoracolumbar spine has been described as thoracolumbar junction syndrome as early as 1974 by Maigne. There may often be clinical manifestations of lower lumbar pain, pseudovisceral pain, and pseudopain on the posterior iliac crest, as well as irritable bowel symptoms. The lateral branches of the dorsal rami of lower thoracic and upper lumbar segments become cutaneous over the buttocks, iliac crest, and greater trochanter. Symptoms may also be perceived in the area of the inguinal ligament, groin, and testicles. Symptoms in any of these regions can be referred from the lower thoracic and upper lumbar spine, and the thoracolumbar junction should be examined thoroughly in their presence.

23. During the history portion of the examination of patients over 50 years of age with thoracic spine pain that is not associated with trauma, why is it important to identify red flags associated with cancer?  
Metastatic lesions in the skeleton are much more common than primary tumors of bone (overall ratio = 25:1). The presence of metastases increases with age. Patients who are 50 years old or older are at greatest risk of developing metastatic disease. Metastases occur more commonly in the axial
skeleton than in the appendicular skeleton. The thoracic spine is the area of the spine most frequently affected by metastases. Breast cancer is the most common site of tumor origin. In addition, skeletal metastases from tumors of prostate, lung, thyroid, kidney, rectum, and uterine cervix are quite common.

24. Describe the clinical presentation of postherpetic neuralgia.
Postherpetic neuralgia is pain that persists for longer than 1 month after the rash of acute herpes zoster (reactivated chickenpox virus) resolves. The pain can be lancinating or manifest as a steady burning or ache along a thoracic dermatomal pattern. The involved skin area is often hypersensitive to light touch. Postherpetic neuralgia can mimic thoracic radiculopathy or referred pain of thoracic spine origin.

25. Define costochondritis. What can the physical therapist do about it?
Costochondritis is an inflammation or irritation of the costochondral junction. Frequently it is referred pain from thoracic or rib dysfunction, probably in the corresponding vertebral level. Examination of the thoracic spine and posterior chest wall is warranted. Treatment using segmental mobilization and/or manipulation has been advocated.

26. If the patient demonstrates inhibition or difficulty in activating the lower trapezius muscle, what should the therapist consider?
The therapist should screen the T8–T12 segments for extension restrictions. Segmental mobilization or manipulation to improve extension often results in immediate improvement of lower trapezius muscle activation. The mechanism is unclear; it may be secondary to localized pain that inhibits maximal muscle firing.

27. If the patient demonstrates inhibition of the serratus anterior muscle or has difficulty in stabilizing the scapula during arm movements, what should the therapist consider?
In the absence of long thoracic neuropathy, the therapist should screen the T3–T7 vertebral segments for flexion restrictions. Segmental mobilization or manipulation to improve flexion often results in immediate improvement of serratus anterior muscle activation. The mechanism is unclear; it may be secondary to localized pain that inhibits maximal muscle firing.

28. What areas of the cervical spine typically refer pain to the thoracic region?
- C2–C3—posterior skull and suboccipital region
- C3–C4—suboccipital region to the nape of the neck
- C4–C5—nape of the neck to the upper trapezius region
- C5–C6—nape of neck, over entire upper trapezius region, and upper medial border of the scapula
- C6–C7—over entire upper trapezius region, extending inferiorly over the entire scapula

BIBLIOGRAPHY


**CHAPTER 59 QUESTIONS**

1. The prevalence of thoracic spine pain is the greatest in which of the following groups?
   a. Soccer players
   b. TOS
   c. Children
   d. Males

2. Which of the following is said to be true regarding the side-bending and rotation-coupling patterns of thoracic spine motion?
   a. In an extended thoracic spine, side-bending and rotation are coupled in opposite directions.
   b. In a flexed thoracic spine, side-bending is coupled with rotation to the opposite side.
   c. In a neutral thoracic spine, side-bending and rotation are coupled in the same direction.
   d. None of the above

3. Which of the following is not a differential diagnosis for thoracic spine pain and dysfunction?
   a. Postherpetic neuralgia
   b. Scheuermann’s disease
   c. Asherman’s syndrome
   d. Ankylosing spondylitis
1. How common is trauma to the spinal column?
There are over 1 million spine injuries per year in the United States alone; 50,000 of these injuries include fractures to the bony spinal column. Males outnumber females 4 to 1 for spinal trauma. Injury is most common at the cervicothoracic and thoracolumbar junctions. The improvement in automobile restraint systems has increased survival rates from major spinal column injuries.

2. How many spinal cord injuries occur per year in the United States?
An estimated 16,000 people sustain spinal cord injuries each year, with 11,000 of the injured surviving to reach the hospital. Overall, 10% to 25% of spinal column injuries are associated with at least some neurologic changes. These changes are more common with injuries at the cervical level (40%) than at the lumbar level (20%).

3. What are the most common modes of spinal column injury?
Almost half (45%) are related to motor vehicle accidents (MVAs). Falls account for another 20%. In children falls account for only 9% of significant spine injuries, whereas in older patients, they account for 60%. Sports injuries account for another 15%. Of these, diving injuries are the most common. Trampoline, ice hockey, and wrestling are other frequent culprits. Organized football accounts for 42 cervical fractures and 5 cases of quadriplegia per year. This statistic has decreased from 110 and 34, respectively, in 1976 (before the spear tackling rules were enacted). Another 15% of spinal column injuries are related to acts of violence.

4. In what scenarios are spinal column injuries most likely to be missed?
Worsening neurologic deficits occur in only 1.5% of patients diagnosed early but in 10% of patients with missed injuries. Injuries are most commonly missed in patients with a decreased level of consciousness, intoxication, head trauma, or polytrauma. Two, separate noncontiguous spinal injuries occur in as many as 20% of cases. The presence of one obvious spinal injury increases the chance of missing another, subtler injury. Red flags to alert the practitioner to subtle spine injury are facial trauma, calcaneus fracture, hypotension, and localized tenderness or spasm. Significant injury is also more likely in patients with osteopenia or neuromuscular disease.

5. What is the long-term prognosis of a spinal cord–injured patient?
The average 10-year survival rate in all patients with spinal cord injury is 86%. In patients over 29 years of age, this number drops to 50%. Pneumonia and suicide are the chief causes of death.
6. What are incomplete cord syndromes, and how do they affect rehabilitation?
Incomplete cord syndromes reflect injuries in which only part of the cord matter is damaged. Although severe, some function below the level of injury is preserved.

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>MOI/Pathology</th>
<th>Characteristics</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central</td>
<td>Age &gt;50 y, extension</td>
<td>UE &gt; LE, M + S loss</td>
<td>Fair</td>
</tr>
<tr>
<td>Anterior</td>
<td>Flexion-comp (vert art)</td>
<td>Incomplete motor, some sensory</td>
<td>Poor</td>
</tr>
<tr>
<td>Brown-Séquard</td>
<td>Penetrating trauma</td>
<td>Ipsilateral motor, contralateral pain/temp</td>
<td>Best</td>
</tr>
<tr>
<td>Root</td>
<td>Foraminal comp/disc</td>
<td>Based on level, weakness</td>
<td>Good</td>
</tr>
<tr>
<td>Complete</td>
<td>Burst, canal comp</td>
<td>No function below level</td>
<td>Poor</td>
</tr>
</tbody>
</table>

MOI, Method of injury; UE, upper extremity; LE, lower extremity; M + S, motor and sensory; vert art, vertebral artery injury; comp, compression.

7. How is the pediatric spine differently susceptible to trauma?
For children older than 8 to 10 years, the spine behaves biomechanically like an adult’s. Younger children have more elastic soft tissues that make multiple, contiguous fractures much more common than in adults. The large size of the child’s head, relative to the body, places the fulcrum for spinal flexion at C2–C3 in children. For children older than 8 years, the fulcrum is at C5–C6. Younger children are therefore far more likely to have upper cervical spine injuries (occiput to C3).

8. What is SCIWORA?
The marked elasticity of the pediatric spinal column is greater than the elastic limit of the cord. Therefore in rare cases, the Spinal Cord can be Injured Without Obvious Radiographic Abnormality (SCIWORA). More than half of these children will have delayed onset of neurologic symptoms, and therefore close and repeated examinations are needed. In recent years, the concept of SCIWORA has been challenged. In any case, the ready availability of MRI makes the concept less critical than in years past.

9. How are gunshot wounds to the spine treated?
Because there is little ligamentous injury associated with civilian weapons, most can be treated closed with external immobilization. As bullet removal often worsens neurologic deficits, surgery is recommended only if the neurologic deficit is progressive, a CSF fistula ensues, or lead poisoning occurs. Surgical indications after colonic perforation are controversial.

10. Describe appropriate steps in the early evaluation of spinal column injury.
In trauma patients, the spine is assumed to be unstable until a secondary survey and radiographs have been performed. Directly examine the back by log-rolling the patient while maintaining in-line traction on the neck. Ecchymosis, lacerations, or abrasions on the skull, spine, thorax, and abdomen suggest that force was imparted to underlying spinal elements. Deformity, localized tenderness, step-off, or interspinous widening warrants further evaluation.

11. Describe appropriate steps in the early management of spinal column injury.
First, immobilize the spine on a backboard with sandbags and a hard collar. After radiographs and a secondary survey have excluded major instability, transfer the patient to a regular bed. Maintain a hard cervical collar until the cervical spine has been formally cleared. Until definitive stabilization can be undertaken, patients with significant thoracolumbar injury should be transferred to a rotating frame or other protective bed. For unstable cervical trauma, traction may be required. High-dose steroid protocols are no longer considered the standard of care in the acute management of spinal cord injury.
12. How is the level determined in spinal cord injury?

Because the cord ends at the L1–L2 disc space, the level of injury to the spinal column may not match the level of cord injury. The cord level is defined as the lowest functional motor level, that is, the lowest level with useful motor function (grade 3 of 5, or antigravity strength). In some cases, a given cord injury will be described as “T8 motor and T12 sensory.”

13. Are there any radiographic clues that an injury might be unstable?

The spine is divided into three columns—anterior (the anterior two thirds of the body and disc), middle (the posterior one third of the body and PLL), and posterior (the posterior elements). Injury to two or more columns renders the spine unstable. Other radiographic parameters have also been defined but vary by spinal level and remain controversial. Clues include significant loss of vertebral height (perhaps >50%), marked or progressive spinal angulation (in some studies, segmental kyphosis >20 degrees), or more than 3 to 4 mm of spondylolisthesis.

14. Why is the level of injury important?

The room available for the cord and the native stability of the spinal column vary significantly from the occiput to the sacrum. In the upper cervical spine, the bony elements are highly mobile, and stability comes from the ligaments. Also, the ratio of the size of the canal to that of the cord is large. This extra room allows for more displacement before cord injury. In the lower cervical spine, the narrow canal leaves little room for translation before cord compression.

The rib cage and sternum render the thoracic spine inherently more stable than the rest of the spine. Yet here the canal is narrowest versus cord size. The transition zone between the fixed thoracic and mobile lumbar spine subjects the thoracolumbar junction at higher risk for injury.

The mobile lower lumbar spine has a large canal with ample room for the nerve roots. Nerve roots are more resilient than the spinal cord, so injuries at this level tend to be less neurologically devastating.

15. How are spinal column injuries classified?

There are hundreds of classification systems for spinal trauma in general and injuries to certain vertebrae in particular. There is no widespread consensus as to which system to use. Mechanistic classifications divide injuries into groups based on the force that caused them. The groups are divided into grades to signal increasing severity.

16. What common force vectors cause spinal column injury?

When a car hits a tree, the seat belt holds the passenger back but inertia keeps the skull moving. An accident of this type imparts force to the cervical spine. A distraction vector, for example, lengthens the spinal column by tearing its ligaments. If the patient’s head then hits the windshield, a compression vector shortens the vertebral column by fracturing its bones. Flexion (forward and lateral), extension, and rotation are the other major vectors. In reality, most injuries result from multiple simultaneous forces with one vector predominating.

17. What types of injuries are caused by compression-flexion moments?

MVAs or diving accidents often impart compression and flexion vectors to the spinal column. Early, the anterior column fails in compression. Later, the posterior and middle column ligaments fail in distraction. When the ligaments fail, the fractured level slides posteriorly over the underlying intact vertebra. These injuries are most common in the midcervical spine (C4–C5 and C5–C6).

Compression fractures represent early-stage injuries with no significant ligamentous failure and heal with 8 to 12 weeks of immobilization. Torn ligaments rarely heal without surgery. Therefore higher energy compression-flexion injuries require operative stabilization.
The Ferguson-Allen classification of cervical spine trauma. Spinal injuries are divided into subtypes based on the vector of force that produced them. Group A represents compressive flexion injuries of increasing severity. Group B includes types of vertical compression injuries. Distractive flexion injuries are part of group C. Group D represents compression-extension patterns. The distractive extension patterns are found in group E, whereas lateral flexion injuries are shown in group F.

18. What is a flexion teardrop fracture?

The most severe flexion-compression injury—the flexion teardrop fracture—is the most devastating of all cervical spine injuries compatible with life. Most patients will have either anterior cord syndrome or a complete cord injury. The lateral radiograph demonstrates a large triangular fragment of anteroinferior vertebral body with marked kyphosis at the injured level, leading to subluxation or dislocation of the facets. Complete disruption of the disc and all the ligaments at the level of injury leads to translation and rotation of the involved vertebrae. Surgical stabilization is usually required.
19. How are vertical compression injuries differentiated from compression-flexion injuries?
If an MVA or diving accident leads to a blow to the top of the head rather than flexion, both the anterior and middle columns fail in compression (i.e., a burst fracture). With increasing force, vertebral arch fractures become more common. In cervical spine trauma, this is the only mechanism wherein the bony injury is more important than the ligamentous injury. The absence of ligamentous disruption allows for some of these injuries to heal in a halo. In higher level injuries or those with neurologic injury, anterior decompression and fusion are recommended.

20. What is the most common type of cervical spine injury?
Distractive flexion injuries account for 61% of all subaxial spine injuries. In early stages, only the posterior ligaments fail (i.e., a flexion sprain). Later, the middle and, finally, the anterior columns fail. As the spine displaces, the superior end plate of the subjacent vertebra may compress, but this should not be confused with flexion-compression injuries. The key differences are marked kyphosis with mild bony collapse and displacement between the fractured vertebra and its cranial neighbor.

21. How are distractive flexion injuries treated?
Low-energy injuries disrupt only the posterior column, resulting in facet subluxation only. Collar immobilization allows for complete healing. Increasing trauma leads to facet dislocation that merits reduction with skull tongs (Gardner-Wells tongs) followed by a posterior fusion to prevent late deformity, chronic pain, or worsening neurologic injury.

22. What are the characteristics of compressive extension injuries?
Accounting for almost 40% of cervical spine trauma, these injuries may result from a downward blow to the forehead. They may occur anywhere but are concentrated at C6–C7. Most are stable. At higher energy levels, tension shear failure through the middle and anterior columns allows the superior vertebra to move forward on the subjacent vertebra, leaving the posterior elements behind. In injuries without displacement, halo immobilization yields acceptable healing rates. Injuries with translation are best treated with operative stabilization.

23. What is an odontoid fracture?
Also called the dens, the odontoid is a peg of bone extending from the body of C2 into the arch of C1. This unique geometry maintains stability while allowing for significant rotation. In younger patients, odontoid fractures are associated with high-energy trauma. Patients report pain and a sense of instability; occasionally, the patient’s presenting symptoms include holding the head with the hands. In children under age 7, the fracture passes through the growth plate and is treated with reduction and a halo or Minerva cast for 6 to 12 weeks.

In adults, dens fracture subtypes associated with poor healing and late instability have been identified. For example, the injuries through the cortical waist of the dens (type II fractures) have poor blood supply and a higher nonunion rate. Type III fractures pass through the cancellous bone of the C2 body and are more likely to heal. A trial of halo immobilization is attempted. However, in severely displaced injuries, early stabilization is recommended.

Recently the significant cardiopulmonary and circulatory compromise engendered by halo-vest management in the elderly has led to increased emphasis on rigid, operative stabilization of dens fractures in these otherwise frail patients.

24. What is a hangman’s fracture?
Also known as traumatic spondylolisthesis of the axis, a hangman’s fracture represents a bilateral fracture of the C2 pars interarticularis. Because bilateral pars fractures enlarge the canal, neurologic injuries are rare. Minimally displaced injuries are immobilized in a Philadelphia collar. Displaced injuries benefit from reduction and halo immobilization. If significant subluxation of C2 on C3 is noted, a posterior stabilization procedure is required.

25. What is a Jefferson fracture?
A Jefferson bursting fracture (of the atlas) is a relatively uncommon injury, usually seen in the context of another spine injury, particularly an odontoid fracture or hangman’s fracture. Classically, this injury encompasses bilateral fractures in both the anterior and posterior arches of the C1 ring. Most isolated Jefferson fractures heal in an orthosis. With increased loading, the fragments displace more widely. Beyond 5–8 mm lateral displacement, the transverse atlantal ligament ruptures or avulses, rendering the C1–C2 motion segment unstable. If the CT scan suggests bone avulsion, traction for reduction followed by
halo immobilization may allow for adequate healing. Rupture of the midsubstance of the ligament necessitates C1–C2 fusion. Minimally displaced or isolated single or double fractures through the C1 ring may be treated with a Philadelphia collar.

26. What is whiplash?
Whiplash is a poorly understood clinical syndrome in which seemingly inconsequential trauma leads to chronic neck pain. This injury complex, also called acceleration injury, cervical sprain syndrome, or soft tissue neck injury, usually follows a rear-end collision. Patients treated for whiplash are commonly involved in accident-related litigation. For some of these patients, economic incentives interfere with clinical improvement.

27. How is whiplash different from other cervical spine trauma?
Most cervical spine trauma results from contact force (eg, striking the head on the dashboard, leading to an extension injury). Whiplash, on the other hand, results from inertial forces applied to the head. Anatomic structures including the sternocleidomastoid and longissimus colli muscles, intervertebral disc, facet capsule, and anterior longitudinal ligament have been implicated as pain generators.

28. Who tends to be susceptible to whiplash?
Although there are 4 million rear-end collisions per year, only 1 million result in reported whiplash injuries. Of those involved in these injuries, 70% are women, usually between 30 and 50 years of age. The injury is more common in those with low physical activity jobs.

29. What are the typical symptoms of whiplash?
Most patients report neck pain and/or occipital headaches. These headaches can be dull, sharp, or aching and are usually worse with movement. The pain is associated with stiffness and often radiates to the head, arm, or between the scapulae. Some patients report vertigo, auditory or visual disturbances, hoarseness, temperature changes, fatigue, depression, and sleep disturbances. These symptoms are often provoked or exacerbated by emotion, temperature, humidity, or noise and variably have been attributed to cranial nerve and sympathetic chain disruption.

30. Describe the physical examination and radiologic signs of whiplash.
On examination, decreased range of motion and spasm are noted; however, other objective findings are absent. Similarly, various radiologic modalities have a poor correlation with symptoms. Often a loss of normal cervical lordosis is noted. Preexisting degenerative disease of the spine is associated with a worse prognosis in whiplash. An MRI scan usually appears normal and is rarely indicated.

31. What is the natural history of whiplash?
Symptom onset usually occurs within 2 days. Of patients diagnosed with whiplash, 57% recover completely in 3 months, and 8% remain so severely affected that they are unable to work. For the remaining 35% of patients, a partial recovery occurs. Maximum improvement is usually reached by 1 year.

32. How is whiplash treated?
The goal of treatment is to reengage patients in their normal activities as soon as possible. In mild cases, an immediate return to work is warranted. Otherwise, a 3-week respite to allow for pain control may be advised. Nonsteroidal anti-inflammatory medications are usually recommended. Muscle relaxants and narcotics are not recommended. A collar should be used only for the first few days after the injury. The critical element in treatment is active mobilization. Short-arc active motion is used for pain and spasm. Gentle passive range of motion can be employed to counteract stiffness. After 48 hours, progression to active motion is suggested. After the acute pain subsides, proceed with isometric strengthening to tolerance. Other modalities are commonly employed, including traction, ultrasound, manipulation, massage, heat, and ice. If significant pain continues after 3 months, a multidisciplinary pain clinic approach has been found to be useful.

33. How are injuries to the thoracolumbar spine classified?
A number of classification schemes have been devised for the thoracolumbar spine. Some are descriptive; some are mechanistic. In general, however, the same principles apply as for the cervical spine. One useful classification, devised by Denis, divides injuries into major and minor types.

34. What might be considered a minor injury of the thoracolumbar spine?
Minor injuries account for 15% of thoracolumbar fractures. They include isolated fractures of the spinous and transverse processes, pars, and facets. They may be caused by direct trauma or violent muscular contraction in response to injury.
35. How are these minor injuries evaluated?
Obtain radiographs of the remainder of the spine to exclude other injuries. Then further assess the affected level for subtle injury with axial CT slices. If the CT is negative, flexion-extension views are important to exclude dynamic instability. For example, a pars fracture may be the only plain film evidence of a flexion-distraction injury. Assuming these tests are negative, the patient can be mobilized without braces or restrictions, except as needed for the relief of symptoms.

36. What are the broad types of major injuries of the thoracolumbar spine?

37. What are compression fractures, and how are they treated?
Compression fractures represent almost half of all major thoracolumbar spinal injuries. They result from a compression failure of the anterior column with the middle and posterior columns left intact. In younger patients with higher energy levels imparted to the spine, a full contact orthosis (such as a thoracolumbosacral orthosis [TLSO]) is recommended. For osteoporotic patients with lower energy trauma, a limited contact orthosis (such as a Cash or Jewett brace) may be appropriate. Increasingly, these injuries are being treated with percutaneous injection of bone cement (polymethyl methacrylate [PMMA]) either with (kyphoplasty) or without (vertebroplasty) balloon reduction of the deformity.

38. How is a burst fracture different from a compression fracture?
A burst fracture includes compression failure of the middle and posterior columns as well. This injury is associated with greater height loss of the anterior column, often with retropulsion of the middle column bone into the canal. A great deal of attention and controversy have been directed to what defines a stable and an unstable burst fracture. Therefore recommendations for treatment of given injuries are often variable. However, the angulation (kyphosis), loss of vertebral height, and canal encroachment as well as the presence or absence of neurologic deficits are evaluated. In general, a neurologically intact patient with little deformity is managed nonoperatively by use of an extension cast or TLSO. Unstable injuries, including those with posterior ligamentous disruption, neurologic deficit, or unacceptable deformities, are treated by surgical decompression and stabilization. This type of surgical procedure may be performed either with a direct anterior decompression and strut graft fusion or with a posterior approach using indirect reduction techniques and screw stabilization.

39. What is a seat-belt injury?
Seat-belt injuries are seen in belted passengers in an MVA without a shoulder harness. A seat-belt injury results from tension failure of the posterior and middle columns. The anterior longitudinal ligament is intact, but there may be compression failure of the anterior column. This injury may occur through bone or soft tissue. If it occurs through bone, it is termed a Chance fracture. Such bony injuries are treated nonoperatively with an extension cast or thoracolumbar spinal orthosis. Close follow-up is required to exclude progressive deformity. If significant soft tissue or ligamentous injury is involved, less predictable healing occurs with closed means, and a posterior stabilization procedure is recommended.
40. How are fracture dislocations different from other types of thoracolumbar traumas? In these injuries, all three columns fail and vertebral translation occurs, causing canal occlusion at the injury site. Therefore fracture-dislocations are associated with a high incidence of neurologic deficits. These injuries may be divided into subtypes based on the direction of translation: flexion-rotation, shear, and flexion-distraction. Almost all of these injuries require operative stabilization.

41. What are some complications associated with the surgical treatment of spinal trauma? Implant displacement, which is most common after posterior instrumentation, is an important consideration in any patient describing increased pain or deformity. Such displacement is often related to poor bone quality, implant placement error, and noncompliance with brace/activity recommendations. Another common problem is postoperative wound infection. Increased drainage, redness, fever, and pain are signs of such an infection.

42. When may a spinal trauma patient be safely mobilized? Mobilization is a critical issue in trauma patients and must be individualized. The benefits of immobilization in shielding the healing spine from excessive external loads are counterbalanced with the drawbacks, including increased muscular stiffness and weakness. In patients with polytrauma or neurologic injury, external bracing is burdensome and interferes with optimal rehabilitation. Stable injuries are mobilized immediately with gentle, passive ROM. In these patients, modalities such as ice, heat, ultrasound, and massage appear helpful in symptomatic relief. A stretching and strengthening program is gradually added as pain levels decrease and motion increases. Unstable spinal column injuries will not tolerate early motion. In general terms, however, an injury with significant instability should be converted to a stable configuration by way of external bracing, surgery, or both.

A rigidly stabilized spine is often mobilized within 2 weeks. In injuries treated with less than rigid fixation or in those patients with poor bone quality or other factors compromising their fixation, 6 to 12 weeks of external orthosis wear is followed by the initiation of gentle, active ROM. Strengthening is instituted upon attainment of full and painless motion in patients for whom x-rays demonstrate no change in position of hardware or vertebral elements.

In patients with unstable injuries treated with nonoperative means, mobilization is started at times predicted by tissue healing. Therefore compression fractures through cancellous bone may tolerate mobilization at 4 weeks. On the other hand, cortical bone injuries (such as dens fractures) and injuries with a significant ligamentous component (burst fractures with severe collapse) will require 12 to 16 weeks of immobilization. Dynamic radiographs (flexion-extension views) are often useful to evaluate healing before aggressive rehabilitation.

43. Name other common postoperative medical problems to which spinal trauma patients are prone. Deep venous thrombosis (DVT), pulmonary embolism, and pressure sores are very serious potential consequences of the immobilization required after major spinal injury. Pneumonia, pneumothorax, and other pulmonary problems are common as well. Autonomic dysreflexia is seen in patients with cervical and upper thoracic spinal cord injuries. In this disorder, bladder overdistention or fecal impaction causes an autonomic nervous system reaction, which leads to severe hypertension. The patient’s presenting symptoms often include a pounding headache, anxiety, profuse head and neck sweating, nasal obstruction, and blurred vision. Treatment begins with immediate placement of a Foley catheter and rectal disimpaction. If the symptoms do not quickly resolve, medications are required.

44. What percentage of patients experience pain relief or functional improvement after kyphoplasty or vertebroplasty? Good to excellent relief of pain is seen almost immediately after both kyphoplasty and vertebroplasty in 80% to 100% of patients. This pain relief persists over time. Vertebroplasty studies often report phone call follow-up of pain levels and have relatively little outcome data, but in studies of kyphoplasty, validated functional outcome instruments have demonstrated clinically and statistically significant improvements, including the SF-35 role physical and physical function subscales, Oswestry scores, and Roland-Morris scores.
45. What is the role of physical therapy in the status of osteoporotic patients after a vertebral compression fracture?

Osteoporotic patients are at risk for additional fractures. In particular, lifting while flexing or lifting overhead increases the risk of fracture. On the other hand, in the absence of weight bearing, bones will continue to deteriorate. Increasingly, a rehabilitation program, including gait and balance training and extensor muscle strengthening, is being recommended in conjunction with a therapist-centered educational program about appropriate lifting techniques and back protection.

46. How can therapy help prevent osteoporotic fractures?

A combination of strength training and weight-bearing exercise can increase bone density in postmenopausal women.

**BIBLIOGRAPHY**


**CHAPTER 60 QUESTIONS**

1. Which incomplete cord syndrome has the best prognosis for recovery?
   a. Central
   b. Anterior
   c. Brown-Séquard
   d. Lateral

2. Which of the following regarding whiplash is false?
   a. It is due to inertial forces on the head.
   b. Men are more commonly involved.
   c. More common in 30–50 year olds.
   d. 8% never return to work.
3. How can therapy help prevent osteoporotic fractures?
   a. Electric stim can increase bone density.
   b. Aerobic exercise can increase bone density.
   c. **Weight-bearing and resistance exercises can increase bone density.**
   d. Aquatic exercise can increase bone density.
1. What are the unique features of the temporomandibular joint (TMJ)?
   The TMJ is divided by a disc into an upper and a lower joint cavity. During mouth opening, rotation of the condylar head takes place in the inferior cavity, and translation occurs in the superior cavity. The TMJ and the disc are both covered with fibrocartilage, which has a superior reparative property to wear and tear. The TMJ, functioning as one of a pair, must perform coordinated movements.

2. What is the incidence of TMJ dysfunction?
   Fifty to seventy percent of the adult population suffers one sign of TMJ dysfunction at some time in their life. Population-based studies have reported 1% to 22% of the general population suffers one symptom of temporomandibular dysfunction (TMD) at some time in their life. Women are affected three times as often as men. Approximately 40% of the population has clicks during daily function.

3. How does TMD manifest clinically?
   Clinical symptoms of TMD include pain in the masseter, temporalis, head, face, and neck area; headaches; dizziness; vertigo; earache or fullness; tinnitus; TMJ noises; toothache; myofascial pain; swallowing difficulty; speech disturbance, etc.

4. What are the causes of TMD?
   Macrotrauma: a blow to the face, auto accident that resulted in neck and jaw injury, prolonged opening of the mouth during an oral/dental procedure, etc.
   Microtrauma: parafunction—e.g., clenching and bruxing; strain of masticatory and cervical muscles as a result of poor posture, etc.

5. What is the anatomic attachment and function of the disc?
   Anteriorly, the disc is attached to the superior belly of the lateral pterygoid muscle. The posterosuperior portion of the disc is attached to the superior stratum, and the posteroinferior portion is attached to the inferior stratum. Medially and laterally, the disc is attached to the medial/lateral poles of the condylar head through the medial and lateral collateral ligaments. The disc protects and lubricates the articulating surfaces. It also accepts force that is exerted upon the TMJ.

6. Describe the innervation of the TMJ.
   The anterior and medial regions of the TMJ are innervated by the deep temporal and masseteric nerves. The posterior and lateral regions of the TMJ are innervated by the auriculotemporal nerve. These three nerves arise from the mandibular division of the trigeminal nerve.

7. What are the kinematic movements of mouth opening?
   During the early phase of mouth opening (11–25 mm), anterior rotation of the condylar head is the primary movement. During the late phase (25 mm to end range) of mouth opening, anterior translation of the condylar head is the primary movement. Most researchers nowadays believe rotation and translation occur simultaneously, with varying dominance during different phases.

8. Describe the functional and normal range of mouth opening.
   The functional range of mouth opening is measured by three fingers' width (or two knuckles' width) of the nondominant hand; the normal range is measured by four fingers' width (or three knuckles' width) of the nondominant hand. For men, the normal range of opening is between 40 and 45 mm; for women, the normal range of opening is between 45 and 50 mm.

9. What is the normal range of motion for lateral excursion, protrusion, and retrusion?
   The normal range of lateral excursion is usually one fourth of the normal opening. For example, if a person has a 48-mm opening, then the lateral excursion is expected to be 12 mm. The normal range of protrusion is approximately 6 to 9 mm, and the retrusion range is approximately 3 mm.
10. Where are the center and axis of rotation of the TMJ?
Many researchers who support the hinge axis theory believe that with the first 20 mm of mouth opening, rotation occurs around a fixed center located in the head of the condyle. Other authors support the theory of the instantaneous center of rotation (ie, the mean location is anterior and inferior to the condylar head, with the axis located outside of the condyle). They think that the mandible undergoes both rotation and translation in varying degrees from the initiation of mouth opening.

11. What are the major elevators of the mandible?
The masseter, temporalis, and medial pterygoid muscles are the three major elevators of the mandible. The superior belly of the lateral pterygoid muscle is active during the closing phase of the mouth, but its function is primarily for stabilization of the disc in relationship to the condylar head.

12. What are the depressors of the mandible?
The depressors of the mandible are the inferior belly of the lateral pterygoid, digastric, mylohyoid, geniohyoid, and stylohyoid muscles.

13. Describe the muscle function and kinematics of lateral deviation.
When the mandible deviates to one side, the muscles involved are the ipsilateral temporalis, the contralateral medial pterygoid, and the contralateral lateral pterygoid. Arthrokinematically, the ipsilateral condyle rotates and spins forward, downward, and medially, and the contralateral condyle translates horizontally toward the ipsilateral side.

14. What is the role of the lateral pterygoid in oral function?
Approximately 30% of the superior belly of the lateral pterygoid muscle attaches to the anteromedial portion of the articular disc. This superior belly is active during mandibular elevation, especially in the last phase of forceful chewing between molars. It helps to stabilize the disc and the condyle in a functional position. Spasm of the superior belly of the lateral pterygoid muscle can result in anterior displacement of the disc because of its anteromedial pull on the disc during contraction.

15. How is pain arising from the retrodiscal pad differentiated from pain arising from muscular contraction?
Using a cotton roll, the patient bites down with the back molars. If pain increases, muscular or ligamentous involvement is indicated. If pain decreases (because of decreased pressure on the retrodisclal pad caused by gapping the TMJ), the retrodisclal pad is involved. This finding can be further confirmed by asking the patient to bite down on the cotton roll with the contralateral molars. If pain increases on the ipsilateral side (from compression on the TMJ), then the retrodisclal pad is affected.

Clenching, bruxing, biting nails, sucking on cheeks, chewing gum, and biting lips are examples of parafunctional habits. These nonfunctioning, repetitive movements can cause microtrauma to the soft tissue and the hard structure. Microtrauma may result in pain, spasm, altered mandibular dynamics, abnormal development, and TMJ dysfunction.

17. What are the three categories of TMD as defined by the American Academy of Orofacial Pain?
The three categories are 1) articular disorders (disc, bony malformation), 2) masticatory muscle disorders (myospasm, myositis, and myofascial pain disorder), and 3) arthritis (osteoarthritis, rheumatoid arthritis, and psoriatic arthritis).

18. How does an anteriorly displaced disc present clinically?
A patient with an anteriorly displaced disc usually has pain and limited opening with deflection to the involved side. In the case of an anteriorly displaced disc with reduction (ADDwR), a louder opening click and a milder closing click (reciprocal clicks) may be heard. In the case of an anteriorly displaced disc without reduction (ADDwoR), the mouth opening is limited to less than 40 mm, and the joint noise is absent.
19. What is an open lock?

An open lock is the inability to close the mouth when the condyle is locked in an open position. This usually happens after wide opening from yawning or a prolonged dental procedure. The most likely cause is an overstretched lateral pterygoid muscle or a posteriorly displaced disc.

20. Explain the significance of opening with a C curve or S curve.

Altered TMJ kinematics is often presented by mouth opening with deviation. A “C” curve usually indicates a capsular pattern, whereas an “S” curve indicates muscle imbalance. However, when joint noises, limited opening, and ipsilateral deflection are also present, disc displacement must be suspected.

21. What is the ideal resting position of the tongue?

With the head and neck in neutral position, the tip of the tongue is placed lightly against the roof of the mouth (the palate), not touching the back of the upper front teeth. Upper and lower lips are kept together, and back molars are kept apart. This position is especially important for patients who clench or grind at night.

22. Describe the connection between TMD and forward-headed posture.

Patients with TMD demonstrate a more forward-headed posture than patients without TMD. Generally, it is believed that approximately 85% of patients with TMD hold a forward-headed posture. The tight suboccipital muscles caused by the habitual forward-headed posture rotate the cranium posteriorly and may pressure the greater occipital nerve, which may result in symptoms in the head, neck, and facial area. A forward-headed posture stretches the infrahyoid and suprahyoid muscles, which in turn pulls the mandible in a direction of retrusion and depression. The change of the condyle position within the fossa will set off a vicious cycle of muscle spasm and disc displacement.

23. How can TMJ problems cause dizziness, headache, and ear symptoms?

The TMJ is innervated by the trigeminal nerve. The neurons from the trigeminal nerve (cranial nerve V) share the same neuron pool as the upper cervical nerves (cervical nerves I, II, and III) and cranial nerves VII, IX, X, and XI. Consequently, all the afferent nerves converge and may affect each other’s innervation. This area, the so-called trigeminocervical nucleus, is considered the principal nociceptive center of the entire head and upper neck. Any pain in the TMJ area can be transmitted through the trigeminocervical nucleus to the head and neck area or can be perceived as pain arising from the head and neck area. Patients with TMJ problems usually demonstrate forward-headed posture and suffer from cervical dysfunction. Tightness of the cervical musculature may compromise vertebrobasilar blood flow, one of the causes of dizziness. On the other hand, disturbances in the cervical column, whether they originate from the muscles, ligaments, or joints, can interfere with tonic neck reflexes and also affect the function of the vestibular nuclei.

The auriculotemporal nerve (a branch of the trigeminal nerve) innervates the posterolateral region of the TMJ. This nerve also sends a few branches to innervate the tympanic membrane, the external auditory meatus, and the lateral surface of the superior auricle. Therefore any symptom that affects the auriculotemporal nerve may cause earache, ear fullness, or tinnitus.

24. What are the differential diagnoses of facial and TMJ pain?

The differential diagnoses include trigeminal neuralgia, migraine headaches, herpes zoster, parotid gland tumor, temporal arteritis, tooth abscess, acoustic neuroma, sinus infection, angina, and cervical dysfunction.

25. Describe current evidence-based physical therapy management for patients with TMD.

- Patient education: posture, body mechanics, tongue resting position, and soft diet
- Modalities: heat/cold, ultrasound, electrical stimulation, and light therapy
- Manual therapy: myofascial release (intraoral and extraoral), joint mobilization, and manipulation
- Home exercise program: TMJ exercise and cervical spine exercise
- Stress management, chronic pain management

26. Discuss the roles of splints.

The repositioning splint is generally used to recapture the anteriorly dislocated disc and/or manage the disc-condyle discoordination. It should be worn continuously throughout the day and night except during oral cleaning or eating. The duration may last from a few weeks to several months, depending on the progress of joint stability. The goal of the repositioning splint is to achieve the
centric occlusion position of the mandible, to relax the masticatory muscles, and to stabilize the disc-condyle complex.

The **resting splint** is preferred when relaxation or balancing of soft tissue is desired. This type of splint can be worn during the day or only at night to offset the soft tissue reaction from nocturnal clenching and/or bruxing.

27. **What imaging modalities are used to diagnose TMD?**

**Plain radiography** of the TMJ includes lateral transcranial, transpharyngeal, and transorbital projections. The lateral transcranial projection is used most often; it images the lateral one third to one half of the condyle and fossa but does not include the condylar neck. The transpharyngeal projection images the lateral and medial portions of the condyle; in combination with the transorbital projection, it images the condylar neck.

**Panoramic radiography** is a modified tomogram used to provide a comprehensive view of the dental and bony structure.

**Arthrograms** are used to identify soft tissue abnormalities (e.g., disc displacement, disc perforation, or retrodiscal inflammation). This technique involves the injection of a contrast medium into the joint space followed by static or dynamic imaging. Arthrography is the most sensitive technique for detecting soft tissue perforation; however, it is invasive and involves high levels of radiation exposure.

**Magnetic resonance imaging (MRI)** provides the most accurate information about the soft tissues of the TMJ. Disc position and condition can be identified with MRI. The use of dynamic MRI can reveal functional information for the joint studied.

**Ultrasonic imaging** has gained popularity in recent years for the study of the musculoskeletal system. It is noninvasive, nonthreatening, less expensive, and easy to operate. The bony structures and the soft tissues of the TMJ can be clearly visualized with ultrasound. However, the disc material is difficult to detect because of its location.

28. **What evidence exists in the literature regarding the efficacy of physical therapy for TMD?**

Wright and colleagues studied the usefulness of posture training for patients with TMD and proved that postural exercises can significantly decrease symptoms.

A critique conducted by Feine et al. on the effect of physical therapy in the management of TMD concluded that TMD patients are helped by reversible, noninvasive therapy, especially a general fitness exercise program.

Nicolakis et al. reported that jaw exercise, manual therapy, postural correction, and relaxation techniques are beneficial in decreasing pain and increasing jaw opening in patients with ADDwR.

Furto et al. reported that manual therapy, a specific condylar remodeling exercise, and iontophoresis produced clinically meaningful improvements in symptoms and disability of 15 TMD patients within 2 weeks.

McNeely et al. and Medlicoff et al. conducted systematic reviews of the effectiveness of physical therapy interventions for TMD patients. Both groups reported that the studied methods (relaxation, biofeedback, manual therapy, electrical stimulation, and exercise) were beneficial but cautioned the inference of the result because of the weakness in research design.

Lisa et al. conducted a systematic review of the management of TMD. They concluded that jaw exercise and postural training are among some of the effective methods for relieving TMJ pain.

**BIBLIOGRAPHY**


**CHAPTER 61 QUESTIONS**

1. A 34-year-old female patient comes into your office complaining of pain in her right TMJ with limited opening. The patient’s symptom started after a lengthy dental procedure the day before. Your quick assessment reveals limited mouth opening at 27 mm, with deflection to the right side. Lateral excursion to the right is 9 mm, and to the left is 5 mm. There is no joint noise audible. What would be your working hypothesis at this time?
   a. Anteriorly displaced disc with reduction of the right TMJ
   b. **Anteriorly displaced disc without reduction of the right TMJ**
   c. Anteriorly displaced disc with reduction of the left TMJ
   d. Anteriorly displaced disc without reduction of the left TMJ

2. Function of the inferior belly of the lateral pterygoid muscle is:
   a. Ipsilateral deviation
   b. **Contralateral deviation**
   c. Retrusion
   d. Elevation (closing of the mouth)

3. Which of the following choices refers to the anatomic connection between the TMJ and the cervical spine?
   a. The auriculotemporal nerve
   b. The masseteric nerve
   c. **The trigeminocervical nucleus**
   d. The deep temporal nerve
FUNCTIONAL ANATOMY OF THE SACROILIAC JOINT

M.E. Lonnemann, PT, DPT, MSc

1. Name the osseous structures of the pelvic ring.
   The ilia, sacrum, coccyx, femora, and pubis are the osseous structures of the pelvic ring.

2. How is the sacroiliac joint (SIJ) classified?
   The SIJ has been classified in several ways. Vleeming has classified the joint as an amphiarthrosis (synarthrodial cartilaginous) joint. However, Gray has classified the joint as a modified synovial joint (diarthrosis). The variation in classification is most likely because there are two aspects of the joint. The main portion of the joint is auricular and is surrounded by a complex capsule lined with cartilage (diarthrosis). There is a second dorsally located fibrous articulation that is extracapsular and is considered a synarthrosis stabilized by the interosseous ligaments.

3. Within which sacral segments does the SIJ form?
   S1, S2, and S3

4. Is the sacrum fully fused at birth?
   No, fusion of the sacral vertebrae begins early in the second decade of life.

5. Which surface of the SIJ is concave?
   The sacral surface is for the most part concave; however, often an intraarticular bony tubercle is present in the anterior and middle aspect of the surface of the sacrum. The iliac part is predominantly convex. Variations do exist.

6. Describe the composition of the articular surfaces of the sacroiliac joint.
   The sacral articular cartilage resembles typical hyaline cartilage, and its thickness ranges from 1 to 3 mm. The iliac cartilage resembles fibrocartilage and is usually <1 mm in thickness.

7. What is the function of the sacroiliac joint?
   The SIJ is the link between the axial skeleton and the lower appendicular skeleton, and thus its main function is to transmit forces from the axial skeleton to the lower limbs and vice versa.


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8. How does the orientation of the SIJ make it difficult to establish a specific axis of motion using conventional planes?
In general, the axes of motion lie in a transverse plane at the level of S2. However, motion and rotational axes at the SIJ have been found to vary considerably because of contour variations in the joint surfaces of both the frontal and the sagittal planes. Motion variations also may result from individual differences in ligamentous laxity.

9. Describe the mechanisms of stability in the SIJ in terms of form and force closure.
Form closure is a concept describing the congruity or interlocking mechanisms of the SIJ based on its osteology. Force closure is described as the mechanism by which the ligaments and muscles achieve stability within the joint.

10. Name and label the ligaments of the SIJ, and explain their function in limiting joint movement.

Interosseous sacroiliac ligament: binds the ilium to the sacrum (not pictured). Long and short posterior sacroiliac ligaments: the long ligaments prevent counterrotation of the ilium (B) whereas the short ligament (A) binds the ilium to the sacrum. Anterior sacroiliac ligament: prevents anterior displacement and diastasis of the joint (F). Sacrospinous (C and G) and sacrotuberous (D) ligaments: prevent nutation of the sacrum by anchoring it to the ischium. Iliolumbar ligament: prevents downward and anterior displacement of the ilium (E).


11. Describe the attachments of the anterior sacroiliac and sacrospinous and sacrotuberous ligaments.
The anterior sacroiliac ligament covers the ventral aspect of the joint and extends from the sacral ala and anterior sacral surface to the anterior surface of the ilium beyond the margins of the joint. It is larger in males than in females. The sacrospinous ligament originates from the inferior lateral angle of the sacrum to the ischial spine of the ilium. The sacrotuberous ligament arises from the posterior superior iliac spine (PSIS), merges with the long posterior sacroiliac ligaments and the lateral margin of the sacrum (where it combines with the sacrospinous ligament), and attaches to the ischial tuberosity.

12. Describe the attachments and function of the interosseous sacroiliac ligaments.
The interosseous sacroiliac ligament is one of the strongest ligaments in the SIJ, with the most extensive volume and bony attachment compared with other SIJ ligaments regardless of gender. It fills the joint spaces of the axial joint/ventral auricular joint, as well as the dorsal, cephalic portion of the synovial joint. It provides multidirectional stability and binds the joint together.
13. Which muscles contribute to the stability of the SIJ?
The muscles that cross the SIJ are designed to create movement of the lumbar spine or hip, as well as contribute to the stability of the SIJ. They are not prime movers of the SIJ. The adjacent muscles, including the quadratus lumborum, multifidus, erector spinae, gluteus minimus, piriformis, iliacus, and latisissimus dorsi, contribute to the strength of the joint capsule and ligaments. Other muscles attaching to the pelvic girdle and contributing to the function of the SIJ include the abdominal muscles: internal and external obliques, rectus abdominis, and transversus abdominis. Several studies have demonstrated that, among other muscles, contraction of the internal oblique and transversus abdominus creates force closure of the pelvis, and the superficial layer of the thoracolumbar fascia attaches to the latisissimus dorsi and gluteus maximus, thereby contributing to compression of the SIJ through its contraction.

14. Describe the innervation of the SIJ.
The posterior portion of the joint receives innervation from the lateral branches of the posterior primary rami of L4–S3. The anterior portion of the joint receives innervation from as high as the L2–S2 segments.

15. What neurologic structures emerging from the sacrum innervate the pelvic region and lower limbs?

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Structures Innervated</th>
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<tbody>
<tr>
<td>Tibial (L4–S3)</td>
<td>Medial hamstrings, adductor magnus, posterior compartment of leg, intrinsicus of foot</td>
</tr>
<tr>
<td>Fibular (L4–S2)</td>
<td>Lateral hamstrings, lateral compartment of leg, EDB of foot</td>
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<tr>
<td>Pudendal (S2–S4)</td>
<td>External urethral and anal sphincters, levator ani, and skin of perineum, penis, clitoris</td>
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<tr>
<td>Superior gluteal (L4–S1)</td>
<td>Gluteus medius, gluteus minimus, tensor fasciae latae</td>
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<tr>
<td>Inferior gluteal (L5–S2)</td>
<td>Gluteus maximus</td>
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<tr>
<td>Nerve to obturator internus and superior gemellus (L5–S2)</td>
<td>Obturator internus, superior gemellus</td>
</tr>
<tr>
<td>Nerve to quadratus femoris and inferior gemellus (L4–S1)</td>
<td>Quadratus femoris and inferior gemellus</td>
</tr>
<tr>
<td>Posterior femoral cutaneous (S1–S3)</td>
<td>Skin on posterior thigh</td>
</tr>
<tr>
<td>Nerve to piriformis (S1–S2)</td>
<td>Piriformis muscle</td>
</tr>
<tr>
<td>Nerves to levator ani, coccygeus, external anal sphincter (S4)</td>
<td>Levator ani, coccygeus, and external anal sphincter; skin between anus and coccyx</td>
</tr>
<tr>
<td>Anococcygeal nerves (S4–C0)</td>
<td>Perianal skin</td>
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16. What are the anatomic differences between the male and female pelvis?
The male pelvis is larger with regard to overall pelvic dimensions (measured from crest to crest). In males the iliac crests also extend higher than the female pelvis. The male pelvis is heavy and thick with larger joint surfaces. The female pelvis is light and thin with small joint surfaces. The muscle attachments in the male pelvis are well defined, whereas the female muscle attachments are rather indistinct. The male sacrum is longer, narrower, and more curved, whereas the female sacrum is short and wide. The pelvic cavity is longer and cone shaped in males, whereas the female pelvic cavity is shorter and cylinder shaped.

17. What are the functional differences between the male and female pelvis?

How do they affect the SIJ?
In males the weight of the body is situated in a direct vertical position above the axis of support of the legs. The body weight in females falls behind the axis of support (upward through the acetabulum) so that the gravity vector tends to create a posterior rotation force on the pelvis. Morphologic changes in the joint surface appear earlier in men and are more extensive with regard to joint surface irregularities. Such changes may be a normal response to greater forces on the SIJs of men compared with women. The primary function of the SIJ in women is to increase the pelvic diameter during labor for vaginal delivery.

18. Describe the influence of hormones on the SIJ.
Relaxin, a hormone secreted by the corpus luteum, is present throughout pregnancy. The role of relaxin is to remodel collagen, thus creating ligamentous laxity in target tissues, including the pubic symphysis, in preparation for delivery. Relaxin is produced during the luteal phase of menstruation, at which
time the endometrium of the uterus prepares for pregnancy (between ovulation and menses). The increased levels of relaxin may provoke symptoms in patients with mobility dysfunctions of the SIJ. Changes in progesterone levels also may affect the laxity of the joint.

19. Describe the amount of potential movement at the SIJ.
Minimal range of motion of the SIJ has been reported in studies with good methodology and reproducibility. Sturesson et al. used roentgen stereophotogrammetry of metal balls inserted into the sacrum and ilium and found 1 to 3 degrees or 1 to 3 mm of motion at the sacroiliac joint. Walheim and Selvik used a similar method at the symphysis pubis and found rotation did not exceed 3 degrees and translation did not exceed 2 mm. Recent data support that small movements or the normal range of movement is maintained in the SIJ through the sixth decade, with a slight reduction of movement in males after the sixth decade.

20. Describe the possible movements of the sacrum and innominate/ilium (based on the osteopathic model)

<table>
<thead>
<tr>
<th>Plane of Motion</th>
<th>Ilial/Innominate Movement</th>
<th>Sacral Movement</th>
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<tbody>
<tr>
<td>Sagittal</td>
<td>Anterior and posterior rotation</td>
<td>Flexion (nutation), extension (countermutation)</td>
</tr>
<tr>
<td>Frontal</td>
<td>Superior and inferior translation</td>
<td>Side-bending</td>
</tr>
<tr>
<td>Transverse</td>
<td>External and internal rotation (outflare and inflare)</td>
<td>Rotation</td>
</tr>
</tbody>
</table>

21. Discuss the theoretic movements of the ilium and sacrum that may occur during trunk forward bending, backward bending, hip flexion, hip extension, and gait.
After about the first 60 degrees of trunk forward bending, the pelvis rotates anteriorly around the hip joints. The sacrum follows the lumbar spine to the extreme of flexion in both standing and sitting positions, when counternutation or backward nodding of the sacrum occurs. During trunk hyperextension of the spine, rotation of the sacrum occurs. With hip flexion, rotation of the ilium occurs in a backward direction, and the opposite occurs with hip extension. Inman studied walking and describes posterior iliac rotation during hip flexion through the swing phase, which is accentuated by heel contact and initial loading. During the loading response, the ipsilateral ilium begins to rotate anteriorly. The sacrum seems to rotate forward about a diagonal axis, creating torsion on the side of loading at midstance.

22. Describe the age-related changes in the SIJ.
Over time the SIJ develops a coarse texture with ridges and depressions that enhance the stability of the joint. During the first 10 years of life, the joint surfaces remain flat, but in the second and third decades they begin to develop uneven articular surfaces. By the third decade, the iliac surface has developed a convex ridge through the center of the joint surface with a corresponding ridge on the sacrum. By the fourth and fifth decades, the joint surfaces become yellowed and roughened with plaque formation and peripheral joint erosions. In all specimens, marked degenerative arthrosis is the rule by the fourth decade. Sacral osteophytes begin to form in the fourth decade at the joint margins. By the sixth and seventh decades, the osteophytes enlarge and begin to interdigitate across the joint surface. The joint surfaces become irregular with deep erosions that sometimes expose the subchondral bone. By the eighth decade, osteophyte interdigitation increases to the extent that some specimens exhibit true bony ankylosis. The joint surfaces demonstrate marked degenerative changes with diminished articular cartilage on both surfaces.

23. Why does the SIJ begin as a mobile joint and progress toward a stable joint?
In the non-weight-bearing infant, the SIJ is not required to provide stability. As the child progresses to weight-bearing movements, the SIJ undergoes a transformation into a stable interlocking joint that serves as a force transmission center from the spine to the lower limbs and vice versa.

24. Explain the standard views for radiographic evaluation of the SIJ, and discuss the anatomic structures that are best visualized in each image.
Standard radiographic views of the SIJ include anteroposterior (AP), axial, and right and left posterior obliques (RPO and LPO, respectively). In the AP view, the articular surfaces present as two radiolucent lines because they are superimposed on each other. The joints are assessed for symmetry and joint margin contour. In the posterior oblique view, the entire margin of the joint space can be visualized.
Assessment from this view includes extent of joint width, location of bony margins, and degenerative or fibrous changes within the joint.

**25. What is the incidence of sacralization in the United States, and how is the presence of a cervical rib associated with sacralization?**

Sacralization is when the transverse processes of the fifth lumbar vertebrae fuse to the sacrum or ilium (unilaterally or bilaterally). It occurs in 6% of adults. Studies tend to suggest a weak association between sacralization and low back pain. In a recent study of 1053 patients, 73% with cervical ribs had sacralization, and 64% with sacralization had cervical ribs. The value of this information is that if a patient is determined to have either a cervical rib or sacralization, the clinician should be aware of the association, which may help with the differential diagnosis of musculoskeletal complaints.

**26. What is lumbarization?**

Lumbarization is when the S1 vertebrae is separated from the sacrum and could be considered a sixth lumbar vertebrae. The incidence ratio of sacralization to lumbarization is 2:1.

**BIBLIOGRAPHY**


**CHAPTER 62 QUESTIONS**

1. Which of the following set of terms best describes the male pelvis compared with the female pelvis?
   a. Larger in width and height, larger joint surfaces, narrow and curved sacrum, and a cone-shaped pelvic cavity
   b. Larger in width and height, smaller joint surfaces, narrow and curved sacrum, and a cone-shaped pelvic cavity
   c. Smaller in width and height, larger joint surfaces, narrow and curved sacrum, and a cylindrical-shaped pelvic cavity
   d. Smaller in width and height, small joint surfaces, short and wide sacrum, and a cone-shaped pelvic cavity

2. The main portion of the SIJ is surrounded by a joint capsule and lined with cartilage. This portion of the joint is considered the __________ joint.
   a. Diarthrodial
   b. Synarthrodial
   c. Synchondrosis
   d. Symphysis

3. Contraction of which of the following muscles would best contribute to forced closure of the pelvic ring?
   a. Hamstrings and gluteus maximus
   b. Gluteus medius and quadratus lumborum
   c. Rectus abdominus and multifidus
   d. Transversus abdominis and internal oblique
1. How are pelvic girdle disorders classified from an impairment-based model?
Lee distinguishes three types of pelvic girdle disorders: 1) hypomobility with or without pain, 2) hypermobility with or without pain, and 3) normal mobility with pain.

2. What are the typical mechanisms of injury of the sacroiliac joint (SIJ)?
Activities that produce posterior torsion stress on the SIJ include heavy lifting, falls on the ischial tuberosity, vertical thrusts on the extended leg (such as a sudden, unexpected step off a curb), and persistent postures (such as standing on one leg, bowling, and kicks that miss the ball or target).

Activities that produce anterior torsion stress include golf swings and horizontal thrusts on the knee with the hip flexed (such as during a motor vehicle accident when the knee is suddenly thrust against the dashboard).

Repetitive strain to the SIJ can result from decreased extensibility of muscles associated with the pelvic girdle. Decreased extensibility of the hip flexor musculature can create a repetitive anterior torsion strain during gait. Decreased extensibility of the hamstrings can produce a repetitive posterior torsion strain.

3. When a patient’s symptoms include sacroiliac dysfunction, are there certain activities that either aggravate or relieve the pain as supported by a base of evidence in physical therapy practice?
Evidence indicates that no aggravating or relieving factors are of value for the diagnosis of SIJ-related pain. Anecdotal evidence has supported walking, unilateral standing, sexual intercourse, climbing or descending stairs, sit-to-stand movements, and getting in and out of a car as activities that aggravate the SIJ. Rolling over in bed also may cause pain by gapping or compressing the involved joint.

4. Do age and gender play a role in the development of SIJ pathology?
Women tend to have smaller and flatter joint surfaces that increase joint mobility. Sacroiliac hypermobility and dysfunction associated with hypermobility is most common in females between the ages of 10 and 40. Joint hypermobility in females may be exacerbated by hormonal changes caused by relaxin. The increase in mobility may lead to hypermobile conditions of the SIJ. The female patient usually presents when age-related changes in degenerative arthrosis are mild. Because the female’s body weight falls behind the axis of support (through the acetabulum), the gravity vector tends to create a posterior rotation force on the pelvis that causes strain on the posterior ligaments of the SIJ. In a recent retrospective review of patients who received dual anesthetic blocks of the SIJs, it was noted that the average age of patients diagnosed with SIJ pathology based on injection was in the mid 50s. This finding may be attributed to the degenerative process and potential for movement impairment in this population. There was no statistically significant difference in the propensity for SIJ pathology in males or females in this age group and population.

5. Describe the pattern of pain referral from the SIJ, as mapped by injection.
The pain referral pattern from the SIJ has been described by Fortin and April as unilateral to the involved side in an area approximately 3 by 10 cm immediately inferior to the posterior superior iliac spine. Slipman found a similar pain pattern using intraarticular injection and reported the following pain patterns: 94% buttocks, 48% posterior thigh, 28% posterior lower leg, 13% foot/ankle, 14% groin, and 2% abdomen.
6. Has limitation in lumbar range of motion been determined to be a predictor of SIJ dysfunction?
No; Schwarzer and Maigne both assessed range of motion in patients with SIJ dysfunction and found no statistical significance for the use of decreased lumbar range of motion as an indicator of SIJ dysfunction.

7. Based on current literature, which appears to be more useful for evaluating the SIJ—assessment of anatomic symmetry or pain provocation?
Assessment of pain provocation is more useful because many asymptomatic patients have minor asymmetry.

8. Which provocation tests have been found to be the most useful in terms of reliability, sensitivity, specificity, and validity?
- Compression
- Distraction
- Thigh thrust
- FABER (fixed abduction external rotation)
- Gaenslen’s
- Resisted hip abduction
- Sacral thrust

The thigh thrust is the most sensitive test (0.88), the distraction test is most specific (0.81), and the compression test has the strongest positive likelihood ratio (2.20). Three or more positive pain provocation tests showed optimal sensitivity (0.85–0.94) and specificity (0.78–0.79) values with high positive likelihood ratios (4.02–4.29).

9. Why is the Patrick’s/FABER test used to assess for SIJ dysfunction?
Patrick’s/Fabers theoretically uses a long lever arm to apply a distraction force to the SIJ. Pain reproduced below the lumbosacral junction overlying the SIJ would be considered a positive test.

10. How could SIJ dysfunction cause acetabular retroversion? And why might this be important to the clinician?
Individuals with SIJ dysfunction oftentimes develop asymmetry of the pelvis with resulting anterior or posterior innominate rotation/tilt, as well as internal or external innominate rotation/tilt. Acetabular retroversion is considered to be the result of a change in orientation of not just the acetabulum but also of the innominate. Acetabular retroversion is associated with rotation of the innominate and this positioning may change the orientation of the acetabulum and increase the propensity for femoracetabular impingement and hip osteoarthritis.

11. Describe the posterior shear or thigh thrust test.
This test is performed with the patient in a supine position. The therapist applies a gentle progressive posterior shearing stress to the SIJ through the femur by contracting the knee and pushing the thigh posterior with the hip flexed. Care must be taken to limit excessive hip adduction. This test assesses the ability of the ilium to translate independently on the sacrum. A painful reaction may be attributable to strain placed on the posterior elements of the joint.

12. Describe the right posterior rotation pelvic torsion provocation test.
Posterior rotation of the right ilium on the sacrum is achieved by flexion of the right hip and knee and simultaneous left hip extension with the patient in the supine position. Overpressure is applied through the right lower extremity to force the right SIJ to its end range. This provocation is sometimes called Gaenslen’s test. A painful reaction may be reproduced by strain on the posterior elements and by joint irritability caused by movement within the joint.

13. Discuss the method and benefits of using injections to diagnose the SIJ as a cause of low back pain.
Diagnostic injections with a local anesthetic and contrast medium can be introduced precisely into the joint via fluoroscopy or computed tomography to assess relief or provocation of pain. A control block eliminates placebo effects. Thus relief of pain gives compelling evidence that the intraarticular SIJ dysfunction is the source. However, it should be noted that pain that arises from the surrounding ligaments or muscles would not be affected by this type of injection. This may give us a reason to question the guided double SIJ injection as the gold standard for validity testing.
14. Describe the osteopathic classifications of sacroiliac dysfunction as presented in clinical practice along with the associated clinical signs.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Bony Landmarks</th>
<th>Leg Length Changes</th>
<th>Lumbar Scoliosis</th>
<th>Muscular/ Ligamentous Changes</th>
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<tr>
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<td>Unilaterally flexed sacrum (left)</td>
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<td>Convex left</td>
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<td>Unilaterally extended sacrum (left)</td>
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<td>Convex right</td>
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<td>Increased psoas and piniformis tone</td>
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<tr>
<td>Bilaterally extended sacrum</td>
<td>Bilateral bases of sacrum prominent</td>
<td>None</td>
<td>None</td>
<td>Tight pelvic diaphragm</td>
</tr>
</tbody>
</table>

PSIS, Posterior superior iliac spine; ASIS, anterior superior iliac spine; ILA, inferior lateral angle.

15. According to the evidence in the current literature, why is it erroneous to consider hypomobility as a clinical syndrome or classification of dysfunction in the SIJ?

The anatomic and biomechanical literature, based on stereophotogrammetric methods, suggests that the SIJ is an inherently stable joint with only approximately two to three degrees of motion. This amount of motion has made it difficult, if not impossible, for clinicians to establish reliable and valid motion assessment techniques in routine clinical examination. Therefore the diagnosis of hypomobility alone is not recommended. The evidence has suggested that excessive mobility—i.e., hypermobility or instability—is more likely to contribute to pain associated with mechanical sacroiliac dysfunction. When the clinician finds asymmetry, a positive provocation test, and differences in passive mobility of the SIJ, the potential diagnosis may better be presented as a “fixated instability.” Theoretically, the joint has become fixated in a nonneutral position, and a discernible difference in passive mobility indicates that it must be excessive to precipitation in the position of fixation. Therefore the joint that appears to be hypomobile is actually an unstable joint that has become displaced or fixated.


Increased passive or active mobility of either the innominate or the sacrum presents with sacroiliac hypermobility dysfunction. Treatment may consist of therapeutic exercises for muscle imbalances, joint manipulation of neighboring hypomobilities in the lumbar spine or hips, patient education about reducing postural and functional stresses through positioning and normal movement for activities of daily
and nightly living, and use of a sacroiliac binder. The use of a sacroiliac binder has been studied in cadavers and found to enhance pelvic stability.

17. What special test is good for determining sacroiliac laxity in postpartum patients?
   - Mens et al. in 1999 found that a positive active straight leg raise (ASLR) test is associated with increased SIJ mobility.
   - Damen et al. found that the ASLR test and the thigh thrust test are good for identifying postpartum patients who have SIJ laxity.

18. What may cause sacroiliac pain when mobility of the SIJ is normal?
   - Mild sprain or strain injury
   - Inflammatory disease
   - Overuse of the adjacent articular or myofascial tissues

19. How can excellent diagnostic accuracy be achieved in the prediction of sacroiliac dysfunction?
   Use of an evaluation to exclude pain of discogenic origin in combination with the use of three provocation tests has been shown to have excellent diagnostic accuracy for sacroiliac dysfunction.

20. What percentage of patients will develop significant SIJ degeneration 5 years after a lumbar fusion?
   Up to 75% of postlumbar fusion patients will develop significant SIJ degeneration.

21. What common medical conditions affect the SIJ?
   - Ankylosing spondylitis (AS) begins as inflammation involving the synovium of SIJs. The ligaments are transformed to bone, beginning at the insertion point, which ends in bony fusion or ankylosis. The incidence varies with ethnic groups: AS is most common in Haida Indians (4.2 per 1000) and Caucasians (1 per 1000). It is more prevalent in males than females by a ratio of 3:1 and is most common in males under the age of 40. Symptoms usually begin in the lumbar spine. Radiologic changes vary from blurring to complete obliteration of the joint margins, resulting in bony fusion of the sacrum to the ilium. AS often appears first with abnormal narrowing of the upper half of the SIJs.
   - Reiter syndrome is precipitated by an infection in the genitourinary or gastrointestinal tract. Although the infection is not found within the joint, the organism causes reactive arthritis, which can cause sacroiliitis. Radiologic changes demonstrate erosions at the insertion points of ligaments.
   - About 15% of patients with inflammatory bowel disease (Crohn’s disease or ulcerative colitis) have sacroiliitis clinically. The radiologic changes resemble those in AS.
   - Psoriatic spondylitis causes bone spur formation and partial bony ankylosis of the SIJs, often asymmetrically. Psoriasis affects 1.2% of the general population; 7% of patients with psoriasis may have arthritis.
   - Other conditions that may affect the SIJ include rheumatoid arthritis, pyogenic infection, tuberculosis, brucellosis, gout, hyperthyroidism, Paget’s disease, diffuse idiopathic skeletal hyperostosis, and osteitis condensans ili.

22. What are the best imaging modalities for diagnosing the cause of SIJ pain?
   No specific imaging studies provide precise findings that are helpful in the diagnosis of SIJ pain. Computed tomography and MRI provide an unobstructed view of the joint and the ability to view the joint margins superiorly and inferiorly for osteophytes. However, they are predominantly used to exclude other causes of sacroiliac pain (tumor, spondyloarthropathies). Bone scans are helpful in determining the presence of stress fractures, infection, inflammation, and tumor.

23. What are the radiologic signs of pubic symphysis instability?
   Instability of the pubic symphysis is suggested by radiographic findings of pubic symphysis separation >10 mm and vertical displacement >2 mm with the single leg stance.

24. Do sacroiliac braces provide pain relief?
   They may provide pain relief. Biomechanical studies of sacroiliac motion while wearing a sacroiliac belt directly superior to the greater trochanter showed an approximately 30% decrease in SIJ motion in cases of peripartum instability. This stabilizing effect could be linked to pain reduction in patients considered to have greater than normal SIJ motion.
25. Do osseous positional changes occur following a high-velocity manipulation to the SIJ?
No. Radiographic stereophotogrammetric analysis before and after manipulation does not demonstrate positional changes of the sacrum and ilium.

26. What is prolotherapy, and is it effective in the treatment of SIJ pain?
Prolotherapy is a form of injection therapy. Sclerosing agents are injected into injured ligaments, which provokes a localized inflammatory reaction. Prolotherapy is proposed to stimulate regrowth of collagen, thus strengthening the ligaments and improving their elasticity and possibly function. Prolotherapy has been found to have superior results to sham injections for chronic nonspecific low back pain; however, its specific application to the SIJ has not been studied.

27. What are some other forms of medical treatments for SIJ pain?
- Nerve stimulators (implanted)—partial pain relief has been reported with selective stimulation of sacral root 3
- Viscosupplementation—partial pain relief has been reported with intraarticular injection of hylan G-F 20
- Radiofrequency neurotomy—64% of 14 patients with SIJ pain who underwent radiofrequency neurotomy demonstrated a >50% pain reduction at a 6-month follow-up visit
- Arthrodesis—a very controversial treatment approach for idiopathic SIJ pain

28. What motor control strategies should a physical therapist assess when considering force closure mechanisms of the SIJ and pelvis?
Alteration in the onset and timing of feed-forward muscular response of the transverse abdominis (TrA) has been identified in patients with SIJ pain.
1. Activation of the transversus abdomenus, pelvic floor musculature
2. Assessment of diaphragmatic breathing
3. Balanced activation between abdominal muscles and trunk extensors
4. Assessment of aberrant movement of active motion of the SIJ/pelvis

BIBLIOGRAPHY


**CHAPTER 63 QUESTIONS**

1. Which of the following tests is most useful in the diagnosis of sacroiliac dysfunction based on its psychometric properties (reliability, sensitivity, specificity, and validity)?
   a. FABER’s, Stork or Gillette’s test, and the standing flexion test
   b. Gaenslen’s, resisted hip abduction, and the supine to sit leg length test
   c. Sacral thrust, sacral shear, and palpation of posterior SIJ ligaments
   **d. Compression, distraction, and thigh thrust**

2. Which of the following diseases/disorders begins as inflammation involving the synovium of the SIJs and causes early onset of bilateral pain in the SIJ region?
   a. Reiter’s syndrome
   **b. Ankylosing spondylitis**
   c. Psoriatic arthritis
   d. Paget’s disease

3. Which is best test to determine SIJ hypermobility or instability?
   a. Compression/distraction provocation test
   **b. Active straight leg raise**
   c. Transversus abdominus muscle activation test
   d. Thigh thrust
1. Describe the articular surfaces of the hip joint.
   The hip joint is created by the acetabulum of the pelvis and the head of the femur. The acetabulum is a cup-shaped structure located laterally on the pelvis and formed by the fusion of the ilium, ischium, and pubis. Only a horseshoe-shaped portion of the acetabulum is covered with articular cartilage and contacts the head of the femur. The acetabular notch lies inferior to this cartilage and is bridged by the acetabular labrum, which also covers the entire periphery of the acetabulum. The acetabular fossa is thus nonarticular and contains a fat pad covered with synovial fluid. The acetabulum faces laterally, anteriorly, and inferiorly.

   The head of the femur is covered completely by articular cartilage except for the fovea or central portion, which serves as the location for the ligamentum teres. The femoral head is circular and attaches to the shaft of the femur by the femoral neck. The femoral head faces medially, superiorly, and anteriorly.

2. How is the hip joint classified?
   The hip joint is a diarthrodial, ball-and-socket joint with three degrees of movement: 1) flexion and extension occur in the sagittal plane around a coronal axis; 2) abduction and adduction occur in the frontal plane around an anteroposterior axis; and 3) internal and external rotation occur on the transverse plane around a longitudinal axis.

3. What is the angle of inclination of the femur?
   It is the angle between 1) the axis of the femoral head and neck and 2) the axis of the femoral shaft in the frontal plane. It begins at approximately 150 degrees in infants and decreases to 125 degrees in adults and 120 degrees in elderly people. The angle is slightly smaller in women than in men because of women’s increased pelvic width. Coxa valga (>150 degrees) is a pathologic increase in the angle of inclination, and coxa vara (<120 degrees) is a pathologic decrease.

4. What is the angle of torsion of the femur?
   It is the angle between the axis of the femoral condyles and the axis of the head and neck of the femur in the transverse plane. The plane of the head and neck is anterior to the plane of the condyles. It is approximately 40 degrees in infants and decreases to approximately 12 to 15 degrees in adults. An increase in the angle of torsion is called anteversion, and a decrease is called retroversion.

5. How is the angle of torsion assessed clinically?
   Femoral anteversion may be assessed using Craig’s test (also called Ryder’s method). The patient is prone with the knee flexed to 90 degrees. The leg is then rotated internally and externally until the greater trochanter is parallel to the table. The amount of anteversion is measured by the angle of the lower leg to the vertical.

6. What gender differences exist in the anatomy of the hip?
   Acetabula are shallower in women than in men. The female pelvis is broader with a greater pubic arch angle. The difference in pelvic geometry creates a reduced tolerance for hip fractures in front-end motor vehicle collisions. The female femur is shorter, lighter, and thinner than the male femur with a smaller femoral head diameter and shorter bicondylar width. This creates a shorter moment arm for the gluteus medius in women and an increase in femoral head pressure. These differences in pelvic and femoral geometry can create a reduced tolerance for hip fractures in female patients.
7. **Describe the joint capsule of the hip.**

The joint capsule is a strong and dense structure that figures prominently in hip joint stability. It attaches proximally to the entire rim of the acetabular labrum and distally to the base of the neck of the femur. The joint capsule covers the head of the femur like a sleeve. It is thickest anterosuperiorly, where the most protection is needed. The posteroinferior attachment is thinner and loose.

8. **Which ligaments contribute to the stability of the hip?**

Two ligaments reinforce the hip anteriorly: 1) the iliofemoral ligament (or Y-shaped ligament of Bigelow), which is the stronger and checks hip hyperextension; and 2) the pubofemoral ligament, which checks hip abduction and extension. The ischiofemoral ligament is located posteriorly; its fibers tighten with hip extension. All of these ligaments are major contributors to stability in an upright standing posture. The ligamentum teres, which passes from the acetabular notch under the transverse acetabular ligament or labrum and attaches to the head of the femur at the fovea, does not add stability to the hip joint.

9. **Describe the arthrokinematics of the hip joint.**

The convex femoral head glides in a direction opposite to the movement on the concave acetabulum in an open-chain condition. In the more common closed-chain condition, the concave acetabulum moves in the same direction as the opposite side of the pelvis.

10. **Describe the osteokinematics of the hip joint.**

Movement of the femur is affected in most directions by the passive tension of two joint muscles. Passive range of motion is as follows:

- **Flexion**—120 to 135 degrees (90 degrees if the knee is extended because of tension in the hamstrings)
- **Extension**—10 to 30 degrees (limited by the rectus femoris if combined with knee flexion)
- **Abduction**—30 to 50 degrees
- **Adduction**—10 to 30 degrees
- **External rotation**—45 to 60 degrees
- **Internal rotation**—30 to 45 degrees

The normal end-feel for all directions of the hip is either tissue approximation or tissue stretch. The movements of the pelvis include anterior and posterior tilting, lateral pelvic tilt, and pelvic rotation.

11. **Name the muscles that cross the hip joint.**

- **Flexors**—iliopsoas, rectus femoris, tensor fascia latae, sartorius, pectineus, adductor brevis, adductor longus, and oblique fibers of adductor magnus
- **Extensors**—gluteus maximus, biceps femoris, semimembranosus, and semitendinosus
- **Abductors**—gluteus medius, gluteus minimus, tensor fascia latae, and upper fibers of gluteus maximus
- **Adductors**—adductor magnus, adductor longus, adductor brevis, pectineus, and gracilis
- **External rotators**—obturator externus, obturator internus, quadratus femoris, piriformis, gemellus superior, gemellus inferior, gluteus maximus, sartorius, and biceps femoris
- **Internal rotators**—gluteus minimus, tensor fascia latae, anterior fibers of gluteus medius, semitendinosus, and semimembranosus

12. **What is inversion of muscle action?**

Muscles that cross a joint with 3 degrees of freedom may have alternate or even opposite (inverted) actions than their classically described actions. The action of the muscle depends on joint position and has important implications for muscle stretching and resistive exercise.

13. **Describe inversion of the flexor component of the adductor muscles.**

All adductors of the hip are also flexors (except the adductor magnus) with the hip in neutral position. With flexion, the femur lies anterior to the origin of the muscle and the adductors become extensors. The adductor longus is a flexor to 70 degrees, the adductor brevis to 50 degrees, and the gracilis to 40 degrees, at which point they become extensors.

14. **Describe inversion of muscle action for the piriformis.**

With the hip in neutral position, the piriformis is primarily an external rotator and a weak flexor and abductor. At 60 degrees of flexion, the piriformis becomes primarily an abductor and medial rotator of the hip.
15. What is the iliocapsularis muscle?
- Origin—anteromedial hip capsule and the inferior border of the anterior inferior iliac spine
- Insertion—distal to the lesser trochanter

The iliocapsularis muscle may tighten the anterior hip capsule to increase stability of the femoral head. The muscle is a landmark during hip surgery in order to expose the anteromedial hip capsule and the psoas tendon interval.

16. What changes occur to the hip musculature following an above-knee amputation?
Amputation changes the geometry of most of the hip muscles because two-joint muscles become one-joint muscles. The cleaved muscles will atrophy 40% to 60% and intact muscles will atrophy up to 30%. If the iliotibial (IT) band is fixed, there is a risk of developing an abduction contracture. With IT band fixation, there is improved hip extension torque by the gluteus maximus to improve propulsion and avoid hip flexion contracture caused by the intact iliopsoas. To avoid an abduction contracture, the adductor magnus is fixated to, and across, the distal femur. When the quadriceps are fixated to the distal femur, the hip should be maximally extended to avoid flexion contracture.

The power generated during the stance phase of gait is reduced 50% in the prosthetic limb. The hip extensors become the primary energy absorbers because of the loss of energy absorption by the knee extensors. During the first 30% to 40% of the stance phase, the hip extensors maintain hip and knee extension to avoid buckling caused by quadriceps and hamstring absence. The intact limb increases hip extension and ankle plantar flexion power in order to clear the prosthetic limb for the swing phase.

17. Are there differences in the strength of hip musculature, with versus without, osteoarthritis (OA) of the hip?
Arokoski et al found a significant reduction in isometric hip abduction (31%) and adduction (25%) strength in males with OA versus without. Hip flexion strength was lower (18%–22%) in males with OA versus without. Hip extension strength was not significantly lower in men with OA versus without, but in those who had bilateral hip OA, the more deteriorated side was 13% to 22% weaker. The cross-sectional area of the hip and thigh musculature did not differ between groups.

18. Describe hip range of motion needed for common daily activities.
- Ascending stairs—40 to 67 degrees of flexion
- Descending stairs—36 degrees of flexion
- Sit to stand—104 degrees of flexion
- Tying shoe—110 degrees of flexion, 33 degrees of external rotation (crossing leg)
- Walking—20 to 40 degrees of flexion

19. Which muscles are active during two-legged erect stance?
None. Stability is maintained by the capsule and ligamentous support.

20. How much force is unloaded from the hip when a cane is used in the opposite hand?
A cane can decrease force loads by 40%. A single contralateral crutch can decrease loads up to 50%.

21. What structures pass through the sciatic notch?
- Vessels—superior gluteal artery and vein, inferior gluteal artery and vein, internal pudendal artery and vein
- Nerves—sciatic, superior gluteal, inferior gluteal, posterior gluteal, nerve to quadratus femoris, nerve to obturator externus
- Muscle—piriformis

22. Describe the blood supply to the femoral head.
- Extracapsular arterial ring—formed posteriorly by a large branch of the medial femoral circumflex artery and anteriorly by the lateral circumflex femoral artery, which are branches of the femoral artery or the profunda femoris artery. The extracapsular ring supplies most of the head and neck of the femur. These arteries surround the neck of the femur and ascend along it, forming rings around the upper neck and subcapital sulcus. The medial circumflex artery branches into the lateral, superior, and inferior epiphyseal arteries, with the lateral epiphyseal artery supplying more than half of the femoral head.
- Ascending cervical branches—formed by the lateral circumflex artery, travel into the joint capsule, and run along the neck of the femur, deep to the synovial lining of the neck. They are at risk with any disruption of the capsule, as may occur in a femoral neck fracture.
- Artery of the ligamentum teres—contributes little, if any, significant supply to the femoral head.
23. Describe the anatomy of the trochanteric bursa.
   A series of three bursae exist: 1) between the gluteus maximus and the gluteus medius tendon; 2) between the gluteus maximus and the greater trochanter; and 3) between the gluteus medius and the greater trochanter. Dunn et al found that multiple bursae could exist and tended to be acquired with age because of excessive friction between the greater trochanter and the insertion of the gluteus maximus at the insertion into the fascia lata.

24. What is the ideal position for hip arthrodesis?
   The ideal position for hip arthrodesis is 25 to 30 degrees of hip flexion in conjunction with neutral abduction and rotation.

25. What is the functional range of motion (ROM) of the hip?
   The functional ROM of the hip is flexion to 90 degrees, abduction to 20 degrees, and internal/external rotation from 0 to 20 degrees.

26. What force acts on the hip joint with active abduction in sidelying?
   Abducting the hip against gravity increases compressive forces at the hip equal to 150% of body weight, which is 450% greater than the force would be by actively abducting in a standing position.

27. Describe the function of the acetabular labrum:
   The fibrocartilage labrum covers the entire border of the acetabulum and increases the coverage of the femoral head by 30%. It functions to improve joint stability by providing a vacuum effect. It also helps absorb shock and transmits stress that would be applied to the cartilage of the femur and acetabulum.

28. What is the maximal loose or open packed position of the hip?
   30 degrees flexion, 30 degrees abduction, and 15 degrees external rotation.

29. What is the maximal close packed position of the hip?
   Unlike other joints, a position does not exist which includes maximal capsuloligamentous tightness and maximal intraarticular pressure during maximal joint surface contact. Intraarticular pressure is greatest with extension and combines with internal rotation or during upright standing. The greatest capsuloligamentous tension occurs during maximal extension combined with either maximal adduction and maximal internal rotation or with maximal abduction and maximal external rotation. Joint surface contact is greatest in maximal flexion combined with maximal abduction and maximal external rotation.

BIBLIOGRAPHY
1. How are muscle strains classified?
   - Grade I—little tissue disruption, low-grade inflammatory response; strength testing produces pain without loss of strength; no loss of range of motion (ROM)
   - Grade II—some disruption of muscle fibers, but not complete; strength and ROM decreased; pain significant
   - Grade III—complete rupture with complete loss of strength of involved muscle; palpable or visible defect may be present

2. How do gluteus medius strains occur?
   The most common cause is the seesaw action of the pelvis during running, although strains also are seen in swimmers. Leg length discrepancies may increase the risk of an abductor strain. Pain is commonly located just proximal to the attachment at the greater trochanter and is reproduced with resisted abduction. It can be confused with greater trochanteric bursitis, which is thought to be painless with resisted abduction, or the two can exist together.

3. What is “bald trochanter”?
   Bald trochanter is the rupture and retraction of the gluteus medius and minimus tendons at their attachment to the greater trochanter as a result of interstitial or deep surface degeneration. It can present as chronic trochanteric bursitis and can be diagnosed with MRI. Treatment involves using a cane in the ipsilateral hand and taking NSAIDs to reduce symptoms. Surgical repair or debridement may be an option.

4. How do groin pulls occur?
   Groin pulls are strains of the hip adductors, most commonly the adductor longus, and occur in sports that require quick acceleration or direction changes. They frequently are seen in ice hockey players, who may be predisposed to groin pulls because of a lack of strengthening (specifically abduction to adduction strength ratio deficits) and stretching of the adductors, previous injury in that area, and lack of experience. A straddle stretch lengthens the muscle bilaterally, but a unilateral stretch may give the athlete better control. Adductor strains also occur in football, rugby, swimming (breast stroke), cricket, bowling, and horseback riding. Most injuries are grades I and II; complete ruptures are rare.

5. What treatment is effective in treating groin pulls?
   Passive physical therapy (massage, stretching, and modalities) has been found to be ineffective in treating groin pulls. However, an 8- to 12-week active strengthening program has proven effective in treating chronic groin strains and allows return to sport. The adductor muscles should be within 80% of the strength of the abductors in order to avoid reinjury. Tyler has developed a program emphasizing eccentric resistive exercise, balance training, core strengthening, and sport-specific movements, which has been supported throughout the literature.

6. When is surgery necessary to treat a groin pull?
   If symptoms persist after 6 months of appropriate physical therapy, and other pathology is ruled out, adductor tenotomy can be performed. However, only 10% of athletes will return to their previous level of competition.

7. What is a “sports hernia”?
   A sports hernia, or athletic pubalgia, is a weakness of the posterior inguinal wall without a clinically palpable hernia. It is characterized by deep groin or lower abdominal pain that is brought on by exertion and relieved by rest, with the presence of palpable tenderness over the pubic ramus, pain with resisted hip
adduction and/or flexion, and pain with abdominal curl-up. Structures that may be at fault include the transverse fascia at the posterior inguinal wall, the rectus abdominus insertion, the conjoined tendon at its distal attachment to the anterior-superior pubis, and/or the external oblique aponeurosis. Athletic pubalgia may be from damage to one or a combination of these structures.

8. How is a sports hernia treated?
Conservative care for athletes and nonathletes would include rest from exerting activities with physical therapy, including manual therapy, trunk stabilization, and dynamic strengthening exercise. If the athlete does not improve with conservative management, surgical repair would be recommended, but not for the nonathlete because of poor surgical outcomes associated with this group.

9. What is the most frequently strained muscle in the body?
The hamstrings have this dubious honor. Injury commonly recurs and usually affects the proximal aspect of the muscle group near the origin at the ischial tuberosity. The mechanism of injury is a rapid, uncontrolled stretch or forceful contraction. A classic example of hamstring injury occurs in hurdlers because maximal hip flexion is accompanied by full knee extension. Proper warm-up and endurance training are important to avoid hamstring strains, which most often occur early or late in a sporting event. Most injuries are grade I or II. True grade III injuries are rare; an avulsion fracture of the ischial tuberosity is more common. Once a strain occurs, proper rehabilitation (improved muscle balance, stretching, proper education about warming up, endurance training, and coordination) is imperative to avoid reinjury. Crosier et al. trained 18 athletes with hamstring strains using specific isokinetic exercises to address their specific strength deficits (quadriceps/hamstring ratios, both concentrically and eccentrically). Of these 18 athletes, 17 improved their isokinetic profiles and returned to sport within 2 months. All 17 remained hamstring injury free at a 1-year follow-up. Although a reduction in strength in the injured hamstring has been found to be a predictor of reinjury, Sherry and Best found that a rehabilitation program needs to include progressive agility and trunk stabilization exercises in order to avoid reinjury. Sports with a high prevalence of hamstring strain include running, sprinting, soccer, football, and rugby.

10. How is hamstring length assessed?
- 90/90-degree straight-leg raise—patient is positioned supine with the hip flexed 90 degrees (either actively or passively). The knee is then actively extended from a starting position of 90-degree flexion toward full extension. The test is positive for hamstring tightness if the angle of knee flexion is >20 degrees.
- Tripod sign—patient sits with knees over the table in 90 degrees of flexion. The examiner passively extends the knee. The test is positive for hamstring tightness if the pelvis is forced into a posterior tilt.
- Hamstring contracture test—patient sits with the tested leg extended while the untested leg is held toward the chest. The patient is instructed to reach the arm ipsilateral to the test leg toward the toes. The test is positive for hamstring tightness if the patient cannot reach the toes while maintaining knee extension.
- Straight-leg raising—patient rests supine while the examiner passively raises the leg with the knee fully extended, and the angle of hip flexion is measured. This test has been found to be highly reliable but does not differentiate between elastic and inelastic posterior hip structures. The medial and lateral hamstrings can be differentiated with a manual muscle test. The semitendinosus and semimembranosus are isolated by positioning the patient in prone with the hip internally rotated and resisted knee flexion. The biceps femoris is isolated by positioning the patient prone with external rotation of the hip and resisted knee flexion. Hamstring tightness should be differentiated from radicular symptoms caused by the sciatic nerve or lumbar spine.

11. Are quadriceps strains common?
No. However, when they occur, they are usually the result of rapid deceleration from a sprint. The rectus femoris is the most commonly affected of the quadriceps muscles because of its two-joint action, but the vastus medialis and vastus lateralis also can be injured. Most damage occurs either in the middle of the thigh or approximately 8 cm from the anterior superior iliac spine for grade I and II strains. Strains are seen in soccer, weight lifting, football, sprinting, and rugby. Tight quadriceps, muscle imbalance between the two extremities, leg length discrepancy, and improper warm-up may be contributing factors.
12. How is rectus femoris length measured?

- Thomas test or rectus femoris contracture test—patient is positioned supine with one knee flexed and held toward the chest. The opposite test leg is positioned so that the lower leg hangs off the edge of the table. If the test knee rests in less than 90 degrees of flexion, the test is considered positive for tightness of the rectus femoris. A positive result is indicated by the inability to rest the leg flat on the table and an increase in lumbar lordosis when the examiner passively extends the knee by pushing the leg into the table. The Thomas test assesses tight hip flexors, which may be present with iliopsoas bursitis. To differentiate between soft tissue and joint restriction, contract-relax maneuvers can be applied at end ROM. If hip extension increases, the hip flexors are the tissue at fault. If the tested leg abducts as the opposite leg is flexed (J sign), tightness in the iliobial band/tensor fasciae latae (ITB/TFL) is indicated. The Thomas test can detect statistical differences in ROM between the two extremities, and applying manual pressure at end range can provide the examiner with valuable information. Information gathered from the Thomas test has not been found to be reflective of dynamic movements of the pelvis during running.

- Ely test—patient is positioned prone. The examiner passively flexes the knee and watches for any hip flexion, which indicates a tight rectus femoris. The examiner should compare results with the other side and watch for reproduced symptoms that may be referred from the femoral nerve.

13. How are the oblique muscles injured?

The external obliques may become strained at their insertion on the iliac crest. Forceful contraction of the abdominals with the trunk laterally flexed is one mechanism of injury (most common in contact sports). The patient has pain with opposite side-bending as well as pain on palpation. Abdominal binders or taping may be necessary to protect the area once the player returns to sport after a period of rest.

14. Describe the treatment for muscle strain.

The length of time for each stage will depend on the severity of the injury.

- Stage 1 (acute phase, first 24–72 hours)—follow basic first-aid protocols of rest, ice, compression, and elevation (RICE). NSAIDs may be administered and crutches may be required for severe strains.
- Stage 2 (reduction of acute symptoms, 2–7 days)—use gentle ROM and isometric exercise with modalities to reduce pain and swelling as needed. Modalities may include ultrasound, hydrotherapy, and muscle stimulation. Gentle friction massage may help avoid adhesion of scarred muscle tissue.
- Stage 3 (pain-free isometrics)—continue with stage 2 treatment as needed for pain, but begin pain-free isotonic and isokinetic exercise. Include stretching and aerobic activity with proper warm-up. Stretching should include static stretches and proprioceptive neuromuscular facilitation (PNF) techniques, such as contract-relax, hold-relax, and contract-relax-contract. Sanders and Nemeth also suggest the use of ballistic stretching, which should follow static stretches and proper warm-up and involves only small movements in the last 10% of the available ROM.
- Stage 4 (ROM 95% of normal, strength 75% of normal)—begin sport-specific exercise with emphasis on endurance and coordination activities. Jogging and running should be progressed gradually.
- Stage 5 (strength 95% of normal)—return to sports with education for maintenance of proper warm-up, stretching, and strengthening program.

15. Describe trochanteric bursitis.

Women are more commonly affected because of the increased breadth of the pelvis. Although trochanteric bursitis occurs most commonly in middle-aged and elderly people, it is also seen in athletes, especially long-distance runners. There are three trochanteric bursae. The first lies between the gluteus maximus and greater trochanter, the second between the gluteus maximus and gluteus medius tendon, and the third between the gluteus medius and greater trochanter.

Onset of disease caused by overuse is gradual, and the patient complains of aching over the trochanter and along the lateral thigh. In runners, a leg length discrepancy may precipitate the condition. Running on banked surfaces may focus more stress on one hip than on the other. Runners who cross midline have an increased adduction angle, which may increase friction at the greater trochanter. Check for excessive wear of the lateral heel in running shoes. Trochanteric bursitis also is seen in
cross-country skiers and ballet dancers. Contact sports such as hockey, football, and soccer may cause bursitis because of direct blows to the lateral hip, which can produce excessive swelling as well as pain.

16. What are the symptoms of trochanteric bursitis?
The patient may complain of a “snapping” at the lateral hip if tightness of the iliotibial band (ITB) is a factor. Pain typically is provoked by ascending stairs and lying on the affected side. Pain also may radiate into the ipsilateral lumbar region. Stretching the gluteus maximus with full hip flexion, adduction, and internal rotation reproduces pain. Resisted testing of abduction, resisted hip extension, and external rotation may be painful. Palpation is positive for tenderness over the posterior aspect of the greater trochanter.

17. How is trochanteric bursitis treated?
Initial treatment consists of rest, ice, and compression wraps, especially in traumatic cases. NSAIDs or local corticosteroid injections may be beneficial. Lying on the affected side should be avoided by changing pillow arrangement. Using pillows between the knees reduces the angle of hip adduction in the side-lying position. Stairs should be avoided. Ultrasound causes an increase in local circulation and may help to resolve the condition. Proper stretching of tightened structures is important; the tensor fasciae latae (TFL), gluteals, and hamstrings may be shortened. Strengthening exercise should correct muscle imbalances across the hip, especially focusing on the gluteals. Cold packs or ice massage help to reduce exercise-induced inflammation.

18. What is Ober’s test?
The patient is positioned in a side-lying position with the tested hip facing upward. The untested leg is flexed at the hip and knee to stabilize the patient. The examiner firmly stabilizes the pelvis at the iliac crest to prevent side-bending of the trunk. The tested hip is extended maximally and adducted. Variations of this test include testing with the knee extended instead of flexed. Stretch on the ITB is increased with the knee extended. Hip internal and external rotation can be added. A positive test reproduces lateral hip pain or restriction in movement. This test is used to assess the length of the ITB/TFL and may be positive in patients with greater trochanteric bursitis. Both Ober’s test and the modified Ober’s test have been found to be a reliable method of testing ITB flexibility but should not be used interchangeably as the modified test will produce significantly more hip adduction range than Ober’s test.

19. How does iliopectineal iliopsoas bursitis develop?
The iliopectineal bursa lies deep to the iliopsoas tendon anterior to the hip joint. Bursitis commonly results from osteoarthritis or rheumatoid arthritis. Other causes include overuse or direct trauma. Overuse can occur with sports such as weight lifting, rowing, uphill running, and competitive track and field. It occurs more commonly in women. An attachment of the bursae to the joint capsule is seen in 15% of cases. Hip joint pathology should be ruled out by checking for a capsular pattern of pain or restriction.

20. Describe the clinical findings in iliopectineal bursitis.
The onset of iliopectineal bursitis is insidious. Pain occurs at the anterior hip and groin with radiation in an L2 or L3 distribution. Lower abdominal pain may be present. The patient may ambulate with a psoatic gait in which the hip is externally rotated, adducted, and flexed during the swing phase. Passive hip flexion with adduction is painful, as is passive hip extension. Strengthening of the hip flexors may be painful and external rotation may be weak when tested with the hip flexed. Palpation elicits tenderness just lateral to the femoral artery at the femoral triangle. The patient may have a palpable snapping at the anterior hip as the involved hip is passively moved from a flexed position into abduction/external rotation and then passively returned to neutral.

21. Describe the treatment for iliopectineal bursitis.
Sanders and Nemeth suggest that ultrasound and interventional current can be beneficial, as is gentle stretching of tightened structures, particularly the iliopsoas. External rotation strengthening has also been proposed, but no studies have verified its efficacy. Local corticosteroid injections may provide relief. Chronic cases may require release of the iliopsoas tendon. Radiographs may be useful to rule out bony pathology.

22. How does ischial tuberosity bursitis present? What is its treatment?
The involved bursa lies between the ischial tuberosity and gluteus maximus. Bursitis usually occurs in people with sedentary occupations or results from a direct fall onto the ischial tuberosity. Pain worsens
with sitting and may refer to the posterior thigh; therefore it is important to rule out lumbar pathology. Palpation over the ischial tuberosity is painful. Hamstring stretching is painful. Hip extension may be reduced in the late stance phase of gait with a shortened stride on the affected side.

NSAIDs and rest are usually successful. The patient should avoid sitting or sit only on well-cushioned surfaces.

23. What is the sign of the buttock?
The patient is positioned in supine while the examiner performs a passive straight-leg raise test. If ROM is limited, the examiner flexes the patient’s knee to see whether hip flexion range increases. If hip flexion increases, the test is negative, but the patient should be examined for sacroiliac, sciatic nerve, or lumbar pathology. A positive test shows no increased hip flexion and indicates pathology of the buttock, which may include ischial tuberosity bursitis. Other pathology should be ruled out, including neoplasm, abscess of the buttock, osteomyelitis, fractured sacrum, and septic bursitis.

24. How are contusions in athletes classified?
- **Grade I** — produces minimal discomfort and should not limit participation in competition
- **Grade II** — more painful and limits ability to perform at extremes of ROM or strength
- **Grade III** — more pain, swelling, and bleeding

25. What is a hip pointer?
A hip pointer is contusion of the lateral hip, which usually results from a blow to the iliac crest. In most cases, the TFL muscle belly is impacted and presents with hematoma; however, the injury may involve tearing of the external oblique at its iliac insertion, periostitis of the iliac crest, or contusion to the greater trochanter. Contact sports such as football, ice hockey, volleyball, soccer, wrestling, lacrosse, and rugby often produce hip pointers from impact with other players. Gymnasts may suffer this injury from impact with equipment. It can also result from a fall with any activity.

26. Describe the clinical findings of a hip pointer.
The injured athlete is immediately disabled by pain. The trunk is flexed forward and toward the side of injury because any side bending or rotation of the trunk is extremely painful. Abrasion or swelling may be present over the iliac crest. Bruising may be immediately present or may become apparent a few days after injury. Pain is caused by any movement involving the muscles that attach to the iliac crest, including the gluteus maximus, gluteus medius, TFL, sartorius, quadratus lumborum, and transverse abdominals. The abdominals may be in spasm.

27. How are hip pointers treated?
Initial treatment is RICE. Crutches may be needed if the patient has pain with ambulation. NSAIDs should not be used before 48 hours after injury because their blood-thinning properties may lead to hematoma. Ice massage is recommended as often as three to four times per day or as pain levels dictate. Gradual stretching keeps the injured area from healing in a contracted position. All exercise should be kept pain free, and pain-relieving modalities such as ultrasound, transcutaneous electrical nerve stimulation, heat, and ice may be used. Strengthening programs should include trunk and leg muscles. The athlete must try to prevent hip pointers in the future by maintenance of a flexibility program and wearing proper protective padding over the iliac crest. Return to sports is allowed in 1 week for grade I injuries; up to 6 weeks may be required for grade II and III injuries.

28. What tests are useful in the diagnosis of hip pointers?
Radiographs help to rule out iliac crest fracture or displaced epiphyseal fracture in athletes who have not reached skeletal maturity.

29. What is the mechanism for a quadriceps contusion? What are the clinical findings?
Usually a direct blow from another player is the cause. In football, contact may be made with a helmet, thigh, or padding. Quadriceps contusion also is seen in rugby, soccer, basketball, and ice hockey. The anterior thigh and lateral thigh are most commonly affected.

Pain occurs with ambulation. The patient is unable to flex and extend the knee fully and may not be able to perform an active straight-leg raise or isometric quadriceps contraction. A hematoma may be palpable.

30. How does treatment for a quadriceps contusion progress?
Initial RICE must be followed strictly for at least 48 hours. Crutches should be used for ambulation. For 48 hours the patient should be nonweight bearing and immobilized in knee flexion to maintain
motion. Then weight bearing should progress once the patient has good quadriceps control and 90-degree pain-free range of motion. Patients should gradually begin passive ROM to avoid contracture. Ice, pulsed ultrasound, and high-voltage galvanic stimulation help to reduce pain and swelling. Patients should begin with isometric exercise and try to progress to straight-leg raises without a quadriceps lag. Massage should be avoided because it may increase hematoma. As patients progress toward pain-free ambulation, crutch use is discontinued, and strengthening should progress gradually as pain allows. Return to sport can begin after full ROM and sport-specific training. There should be less than a 10% difference in strength between the injured and noninjured quadriceps before full return to sport.

31. What causes myositis ossificans?
Myositis ossificans may be a complication of quadriceps contusion and involves development of heterotropic bone in nearby muscle. Surgery or paraplegia also can cause myositis ossificans, or it may result from early treatment of a contusion with massage or heat, premature return to aggressive stretching or strengthening, or premature return to sport. About 7 to 10 days after injury, radiographs may show beginning ossification, which can progress to heterotropic bone in 2 to 3 weeks. Acute contusions should be monitored to watch for thigh and gluteal compartment syndromes.

32. How is myositis ossificans treated?
Early treatment consists only of rest. Weight bearing is reduced with crutches. Once pain and swelling decrease and rehabilitation can begin, initial treatment is geared at gently regaining ROM. Aggressive passive stretching should be avoided for 4 months after injury. Initially no strengthening takes place, but once swelling subsides, gentle isometrics can begin. NSAIDs or corticosteroids may be required to reduce persistent swelling. Once radiographs show that bony growth has subsided, gradual return to activity is progressed. One case study by Wieder showed possible resolution of the bony defect with iontophoresis with acetic acid followed by pulsed ultrasound.

33. Is surgery indicated for myositis ossificans?
Generally no surgery is indicated. If the defect causes significant loss of function, surgery should be performed 9 to 12 months after injury when a bone scan shows no active calcification.

34. What is “snapping hip” syndrome? How is it treated?
Also known as coxa saltans, snapping hip can be internal, external, or intraarticular. The syndrome is characterized by reproduction of a snap or click at the hip with repetitive motion. Most commonly, the cause of external coxa saltans is snapping of the ITB or anterior fibers of gluteus maximus over the greater trochanter. Causes of internal coxa saltans include snapping of the iliofemoral ligaments over the femoral head, the suction phenomenon of the hip joint, and the movement of the iliopsoas tendon over the iliopectineal eminence or lesser trochanter. Intraarticular coxa saltans can be caused by the suction phenomenon of the hip joint, subluxation, a torn acetabular labrum, a loose body, synovial chondromatosis, and osteocartilaginous exostosis. The long head of the biceps tendon snapping over the ischial tuberosity can cause “snapping bottom.” The syndrome is most common in female athletes, such as dancers, runners, gymnasts, and cheerleaders. The clicking in the hip is a greater complaint than pain. Evaluation of which structure is causing the snap or click is made through palpation and while the causative movement is reproduced. Treatment should progress toward alleviating muscle tightness or weakness that may contribute to the disorder. In general, modalities are not required because the condition is usually pain free.

35. Define osteitis pubis.
Osteitis pubis is chronic inflammation of the symphysis pubis. It may occur after operations of the prostate or bladder or result from athletic activity such as soccer, race walking, running, fencing, weight lifting, hockey, swimming, and football. The mechanism of injury is repetitive stress of muscles with attachments at the symphysis pubis, such as the rectus abdominis, gracilis, and adductor longus. Pain in the groin or medial thigh is reproduced with palpation over one side of the symphysis pubis. Abdominal and adductor muscle spasm may accompany pain, and gait may be antalgic with movement adapted to reduce pain.

36. How is osteitis pubis diagnosed and treated?
Radiographs show loss of definition or moth-eaten margins with widening of the symphysis pubis. In chronic cases, the area may appear “moth-eaten.” Bone scans are hot over the pubic symphysis. Treatment consists of rest and administration of NSAIDs with possible use of corticosteroid injections.
37. How does damage occur to the acetabular labrum?
It can occur in a dysplastic hip from changes in the congruence of the joint and abnormal joint stress. It can also occur in nondysplastic hips where labral microtearing, impingement, and cyst formation are precursors to arthritis. Dislocation can result in a labral tear. Anatomic variations in the proximal femur, such as a reduction in anteversion or head-neck offset, can lead to labral tears.

38. How can acetabular labral tears be identified?
- Fitzgerald’s acetabular labral test— if passively moving the hip from flexion, adduction, and external rotation into extension, abduction, and internal rotation reproduces pain, with or without clicking, an anterior labral tear is suspected. If pain is reproduced by moving from extension, abduction, and internal rotation into flexion, adduction, and external rotation, a posterior labral tear is suspected. Fitzgerald found that 54 of 55 hips that tested positive also showed labral tears on MRI or arthrogram.
- Impingement provocation test—patient is supine with the hip flexed to 90 degrees, adducted 25 degrees, and then maximally internally rotated. Pain indicates a possible torn labrum, acetabular rim, or snapping hip syndrome. This test has been found to be able to detect incomplete detaching tears of the posterosuperior portion of the acetabular labrum of dysplastic hips, but it does not correlate well with other arthroscopic findings of dysplastic hips.

39. How are acetabular labral tears treated?
Acetabular tears are treated by reduced weight bearing using crutches and performing range of motion exercises for 4 weeks. If conservative treatment fails, surgery may be an option using open arthrotomy or arthroscopy. Fitzgerald found 13% of patients recovered when treated conservatively. Of those who underwent open arthrotomy or arthroscopic surgery, outcomes were improved if surgery was performed before damage occurred to the femoral head (which created unfavorable outcomes for approximately 12% of subjects).

40. What are the two types of femoral acetabular impingements (FAIs)?
When there is reduced joint clearance between the femur and the acetabulum, FAI results are as follows:
1. Cam impingement is a deformity of the femoral head where an abnormally large radius causes abnormal joint contact, especially with hip flexion combined with adduction and internal rotation.
2. Pincer impingement occurs when the acetabulum covers the femoral head and causes persistent contact of the femoral head into the acetabulum.
Both types of FAIs can lead to labral and chondral damage.

41. How do you test for FAI?
A combination of these tests can be used:
1. FADIR (flexion – adduction – internal rotation) impingement test— hip is placed in 90 degrees of flexion with maximal adduction and internal rotation. This test has been found to be specific for pain provocation in patients with intraarticular, nonarthritic hip joint pain.
2. Patrick/FABER (flexion – abduction – external rotation) test— patient is supine with the heel of the tested limb crossed above the opposite knee while the pelvis is stabilized at the contralateral iliac crest and overpressure is given into abduction and external rotation. Positive finding does not correlate well to a specific hip joint pathology.
3. Scour test— compression is applied to the hip through the femur, over a hip range of motion.
4. Internal rotation at 90 degrees— pain provocation has been associated with bony impingement as a result of FAI.

42. How do you test for ligamentous laxity of the hip joint?
1. Log roll test— patient is positioned supine and the leg is passively rolled into internal rotation and external rotation. Clicking may be indicative of a labral tear, and increased external rotation range of motion may indicate iliofemoral ligament laxity.
2. Long axis distraction— clinician applies a longitudinal distraction force on 30 degrees’ flexion, 30 degrees’ abduction, and 10 to 15 degrees’ external rotation. Excessive motion and apprehension are signs of laxity.

43. Define piriformis syndrome.
Piriformis syndrome is pain in the buttock or posterior thigh and calf caused by inflammation or spasm of the piriformis muscle. Pain is referred in a sciatic distribution because of the close proximity of the piriformis to the sciatic nerve as the two exit the pelvis. Patients complain of pain with walking, ascending stairs, or trunk rotation.
44. How is piriformis syndrome assessed?

1. Frieberg test—patient is positioned supine with the thigh resting against the table while the examiner applies passive internal rotation of the hip.
2. Pace test—patient is positioned in a sitting position while the examiner resists hip abduction.
3. Piriformis test or FAIR test (flexion, adduction, and internal rotation)—patient is positioned in side-lying position with the tested leg facing upward. The test hip is flexed to 60 degrees with the knee flexed. The examiner stabilizes the hip at the iliac crest and passively moves the hip into adduction. A variation of this test is performed in the supine position; with the hip and knee maximally flexed, the examiner moves the hip into full adduction. EMG studies performed in the FAIR position have been found to identify patients who will respond to physical therapy intervention. The FAIR test has been found to have a sensitivity of 0.881 and a specificity of 0.832.
4. Beattie test—patient is positioned side lying as for the piriformis test. With the hip and knee flexed and the knee resting on the examining table, the patient actively externally rotates the hip by lifting the knee off the table and then holds the position.
5. Lee test—patient is positioned in the supine hook-lying position (hip flexed 60 degrees with the foot flat on the table). The examiner resists hip abduction.

A positive result for any of these tests is reproduction of pain symptoms either occurring in the buttock or radiating along the sciatic nerve. Restricted mobility is also a positive finding. Further examination should rule out hip joint and lumbosacral pathology.

45. How is piriformis syndrome treated?

Modalities such as ultrasound or cold pack/ice massage can help to reduce pain and spasm. Fagerson suggests that massage or spray and stretch can help to reduce pain from trigger points in the muscle. Static stretching may be more beneficial than contract-relax if pain is caused by resisted external rotation of the hip. Modifications may be needed in the patient’s base of support in the seated position. Crossing the legs should be avoided, and wallets should be removed from back pockets. Shock-attenuating insoles may help patients who spend a lot of time on their feet, especially on hard surfaces. Correction of leg length discrepancy with a heel lift reduces tension on the piriformis. NSAIDs may be necessary to reduce inflammation. Injection of botulinum toxin A in conjunction with physical therapy has also been found to be of benefit.

46. Define meralgia paresthetica.

Meralgia paresthetica is a nerve entrapment of the superficial branch of the lateral femoral cutaneous nerve as it exits through the femoral canal in the groin or next to the anterior sacroiliac spine (ASIS), where the nerve emerges from the pelvis. Paresthesia is referred along the anterolateral thigh. Common causes include tight-fitting garments, such as a hip-pad girdle or a heavy tool belt, obesity, pregnancy, or direct trauma during contact sports.

47. How is meralgia paresthetica diagnosed and treated?

Tinel’s sign may be positive medial to the ASIS or over the inguinal ligament. Sensory testing should be performed. Meralgia paresthetica is treated with rest. Symptoms typically subside in time; ultrasound and NSAIDs may help a persistent problem. Injection of corticosteroids or surgical nerve release may be required in severe cases.

48. What is hamstring syndrome?

In hamstring syndrome, the sciatic nerve becomes entrapped by adhesions in the proximal hamstrings, which result from repetitive strain. It is seen most commonly in hurdlers and sprinters, and pain may be worse with sitting or stretching or during sport. If conservative measures fail, surgical release of the adhesions may be successful.

49. How does the superior gluteal nerve become entrapped?

As the superior gluteal nerve passes between the greater sciatic notch and piriformis, it may become entrapped by compression of the muscle. Reduced internal rotation of the hip and anterior innominate rotation may be causative factors. Pain occurs in the gluteral area, and tenderness can be reproduced with palpation just lateral to the greater sciatic notch. Treatment is the same as for piriformis syndrome.

50. What outcome measures are validated for orthopedic hip conditions?

The hip outcome score (HOS), the Copenhagen hip and groin outcome score (HAGOS), and the international hip outcome tool (iHOT-33).
1. Describe the Garden classification of femoral neck fractures.
   - Type I—incomplete
   - Type II—complete, nondisplaced
   - Type III—complete, displaced <50%
   - Type IV—complete, displaced >50%

2. Where is the pain from femoral neck fractures typically felt?
   Pain from femoral neck fractures is usually deep in the anterior groin. Pain in the groin with log rolling, in the case of negative x-rays, should be explored with MRI.

3. What are the treatment options for femoral neck fractures?
   In older patients, Garden fractures type I and II may be treated with three percutaneously placed pins. Type III and IV are treated with hemiarthroplasty because of disruption of the femoral head blood supply and high rates of osteonecrosis and nonunion. Patients with preexisting degenerative joint disease may benefit from total hip arthroplasty, although morbidity and mortality are slightly higher. Younger patients (<65) should undergo open reduction and internal fixation (ORIF), if possible, in an attempt to save the femoral head.

4. What is the difference between unipolar and bipolar hemiarthroplasties?
   - Unipolar (Austin-Moore)—only the femoral head is replaced; the native acetabulum is retained. This noncemented prosthesis is used primarily for bedridden and low-demand patients.
   - Bipolar—femoral head is replaced and snaps into a rotating polyethylene shell, which sits in the acetabulum. Bipolar prostheses attempt to reduce acetabulum wear. The superiority of bipolar prostheses has not been proved, although the dislocation rate is lower than with unipolar prostheses.

5. What preventive measures can elderly people take to avoid hip fractures?
   Performing weight-bearing exercises, maintaining adequate calcium intake, maintaining adequate vitamin D intake, decreasing caffeine consumption, cessation of smoking, eliminating household hazards (eg, throw rugs), treating impaired vision, and hormonal implementation can decrease hip fracture risk.

6. Describe the Evans classification of intertrochanteric (IT) hip fractures.
   - Type I—fracture line extends superiorly and laterally from the lesser trochanter
   - Type II—fracture line extends inferiorly and laterally from the lesser trochanter
     Evans further divides the two types into stable and unstable patterns.

7. What are the treatment options for IT fractures?
   IT fractures usually are treated surgically with a dynamic hip screw (lateral sideplate with sliding head screw) or an intramedullary device such as the Gamma nail. Both allow controlled fracture impaction. The Gamma nail may offer more stability for fractures with subtrochanteric extension. The choice of fixation is highly operator dependent.

8. How successful are MRI and bone scans in detecting nondisplaced hip fractures?
   Bone scans detect approximately 80% of fractures within 24 hours of injury. Sensitivity improves to nearly 100% at 3 days. MRI offers immediate, nearly 100% sensitivity in the detection of occult hip fractures.
9. Describe the mortality and morbidity rates associated with hip fractures.

**Mortality Rate**
- Approximately 10% to 30% in the first year after fracture. The mortality risk then returns to the prefracture rate.

**Morbidity Rates**
- Infection—2% to 17%
- Decubitus ulcers—20%
- Nonunion at IT—1% to 2%
- Nonunion at femoral neck—10% to 30%
- Fracture—3% to 4% (for hemiarthroplasty)
- Dislocation—1% to 10% (for hemiarthroplasty)
- Heterotopic ossification—25% to 40% (for hemiarthroplasty)
- Deep venous thrombosis—50% to 60%
- Pulmonary embolism—7%
- Mechanical failure—IT fractures, 12%

In 1990 there were an estimated 1.31 million new hip fractures, 740,000 deaths associated with hip fractures, and 4.48 million patients with disability from hip fracture.

10. Describe the treatment for isolated avulsion fracture of the greater and lesser tuberosities.

These rare fractures usually do well with limited bed rest and progression of weight bearing and ambulation as tolerated. ORIF may be indicated for widely displaced fragments.

11. Define subtrochanteric (ST) femur fractures.

Fractures that occur within 5 cm distal to the lesser trochanter are termed ST femur fractures.

12. What is the recommended treatment for femoral shaft and ST femur fractures?

Fractures in children may be treated with immediate spica casts, traction, external fixation, or flexible nails. Older children and adults usually are treated with a locked intramedullary nail.

13. Should THA be considered in patients with displaced femoral neck fractures?

THA can provide good relief and accelerated rehab in select patients after displaced femoral neck fracture. The dislocation rate can be higher with the posterior approach.

14. What is one predictive measure of outcome after hip fracture?

Grip strength has been correlated with rehab outcomes after hip fracture.

15. What is one of the main predictors of morbidity, length of hospital stay, and complications after hip fracture?

Delay to surgical intervention.

16. What rehabilitation considerations are important after hip fracture?

Capsular trauma is common with a hip fracture despite the lack of frank dislocation. Therefore hip precautions should be used even in patients with ORIF. Hemiarthroplasty allows immediate weight bearing. Although the goal of ORIF is to allow immediate weight bearing to tolerance, weight bearing status should be based on the stability of the fracture pattern and fracture fixation. Nonweight bearing and weight-bearing exercise will increase function after hip fracture.

17. Define the Morel-Lavallee lesion.

The Morel-Lavallee lesion is a closed degloving injury in which the subcutaneous tissue is separated from the underlying fascia. The avascular tissue then undergoes necrosis, resulting in accumulation of liquefied fat and hematoma. This injury is caused by significant blunt trauma that results in acetabular fracture and is at significant risk for infection.

18. What features distinguish a stable pelvis fracture from an unstable one?

Several classification systems attempt to identify which fractures of the pelvis are stable and may be treated nonoperatively and which fractures are unstable and require operative stabilization. Essentially the pelvis is a ring structure. Therefore a single break in the ring usually does not lead to pelvic instability, whereas double breaks (bony or ligamentous) may lead to vertical and/or rotatory instability. The posterior sacroiliac ligamentous complex is the single most important structure for pelvic stability. Fractures that lie entirely outside of the ring (ie, inferior pubic rami fractures) are stable.
19. What is a Malgaigne fracture?
Malgaigne fracture refers to a double vertical fracture of the pelvis, typically a superior and inferior pubic rami fracture associated with an ipsilateral sacroiliac dislocation. The double fracture makes the hemipelvis unstable. Instability can lead to shortening of the hemipelvis and subsequent limb length discrepancy if left untreated.

20. What is the usual mechanism of injury for pelvis fracture?
Low-velocity injuries in older osteoporotic bones often result from lateral compression of the pelvis secondary to a fall. Patients often present with fracture to the superior and/or inferior pubic ramus. High-velocity trauma may result in fractures caused by lateral compression, anteroposterior compression, and vertical shear. These fractures tend to cause significant disruption of the pelvic ring and are therefore more likely to be unstable.

21. Describe the usual mechanism of injury for acetabular fractures.
Fractures of the acetabulum often occur when a direct force is transmitted from the proximal femur. When the hip is flexed (as in an automobile accident), the posterior wall fails. When the hip is extended (as in falls from a height), the anterior wall fails.

22. What are the long-term complications of unstable pelvic ring disruptions?
Chronic low back pain, sacroiliac pain, residual gait abnormalities, and leg length discrepancy are common complaints. Fewer than 30% of patients with >1-cm displacement of the pelvic ring are pain free at 5-year follow-up.

23. Is physical therapy useful after hip fracture?
Physical therapy immediately after surgery is beneficial, based on functional independence measure (FIM) scores at 2 and 6 months post fracture. Home therapy programs, especially those including weight-bearing exercise, have been shown to provide improved strength, walking velocity, and a sense of safety with ambulation. Postinjury levels of function are dependent more upon the age of the patient and their preinjury level of independence than the location of the fracture. Intensive physical therapy has been shown to improve function after hip fracture.

24. Does the rehabilitation site have an effect on recovery of function after hip fracture?
Patients treated in inpatient rehabilitation facilities had higher FIM motor outcome scores and were more likely to reach 95% of their prefracture FIM motor score by week 12 post hospital discharge than those treated in skilled nursing facilities. Also, a significantly greater number of patients were discharged to home from the inpatient rehabilitation facility than the skilled nursing facility.

25. What are the effects of extended outpatient rehabilitation after hip fracture?
Binder et al. compared community-dwelling, frail, elderly patients in supervised physical therapy and exercise training versus home exercise. It was concluded that 6 months of outpatient rehabilitation that included progressive resistance training improved quality of life and reduced disability versus lack of improvement with low-intensity home exercise.

26. What are the differences in rehabilitation between men and women following hip fracture?
Although no differences in the rehabilitation process or outcomes of rehabilitation exist, there is a significant difference in mortality and morbidity. Men are at greater risk of developing a postsurgical complication than women. The risk of increased mortality and morbidity remains elevated for 1 to 2 years post fracture. Men are more susceptible to infections including septicemia and pneumonia than their female counterparts and are twice as likely as women to die within 2 years of hip fracture.

27. Does early mobility after hip fracture influence mortality?
A Finnish study found that patients who could not stand up, sit down, or walk within 2 weeks of hip surgery had the highest mortality rates at a 1-year follow-up. The authors recommended more intensive rehabilitation immediately after surgery. Suetta et al. found that early resistance training markedly reduced hospital length of stay. However, Lauridsen et al. found no significant reduction in time to discharge from rehabilitation with a more intensive program (3.6 hours per week versus 1.9 hours per week); this was probably attributable to a high dropout rate with the more intensive rehabilitation program.
28. Does neuromuscular stimulation to the quadriceps hasten return to mobility after hip fracture?
A study of British women found that neuromuscular stimulation, as part of a home-based rehabilitation program, provided faster return to mobility and a higher percentage of patients returned to preinjury indoor mobility levels by 13 weeks. Electrical stimulation has been found to increase functional muscle performance more than standard rehabilitation alone but did not increase cross-sectional area of the quadriceps as resistance training did. Neuromuscular stimulation of the quadriceps versus placebo produced greater return to recovery of prefracture mobility in the stimulation group.

29. Is there a difference in home physical therapy versus institutional treatment?
Once discharged from the hospital, home-based physical therapy has been shown to yield better ambulation results within five visits than conventional institution-based rehabilitation for 1 month, following fixation of hip fracture.

30. What are the presenting symptoms of a patient with a hip dislocation?
Ninety percent of all hip dislocations are posterior secondary to the mechanism of dislocation and the weak posterior supporting capsule. The posterior hip dislocation can be differentiated clinically because the limb is flexed, adducted, and internally rotated. An anterior dislocation presents with the limb shortened, abducted, and externally rotated. Radiographs should be obtained to evaluate for fracture.

31. What is the postreduction treatment of traumatic hip dislocation?
After closed reduction, thorough neurovascular assessment continues for 24 hours. Patients may be placed in gentle traction for 24 to 48 hours. At that time gentle range of motion may begin. Weight-bearing restrictions continue to be a subject of debate, but in general patients without fracture may slowly begin progressive weight-bearing.

32. What complications are associated with hip dislocation?
- Osteonecrosis—1% to 17%; early reduction decreases the rate
- Degenerative joint disease—33% to 50%
- Sciatic nerve injury—8% to 19%; approximately 50% of patients recover spontaneously
- Femoral head fracture—7% to 68%

BIBLIOGRAPHY


**CHAPTER 66 QUESTIONS**

1. What factor is not important for preventing hip fracture?
   a. Adequate vitamin D
   b. Adequate calcium
   c. Weight-bearing exercise
   d. Aquatic exercise

2. Where is the pain typically felt with a femoral neck fracture?
   a. Lateral hip
   b. Groin
   c. Piriformis
   d. Greater trochanter

3. What is the approximate 1-year mortality rate after hip fracture?
   a. 5%
   b. 20%
   c. 50%
   d. 70%
1. How much force is placed across the hip during routine activities of daily living?
   The force vectors created by contraction of the surrounding hip musculature are the primary determinant of hip joint reactive forces. The double-leg stance has been shown to create hip joint reactive forces of one time the body weight compared with two to three times the body weight for a single-leg stance. Walking produces hip joint reactive forces of two to four times the body weight depending on the pace of gait. Stair-climbing produces forces of three to four times the body weight on the hip joint, in addition to significant torsional forces at the proximal femur. Simply elevating the pelvis to position a bedpan can produce hip joint reactive forces of five to six times body weight as a result of the required hip muscle contractions.

2. What are total hip precautions?
   Instructions given to patients to help minimize the risk of postoperative hip dislocation are termed total hip precautions. The majority of hips that dislocate have a tendency to do so posteriorly. This usually occurs in positions of extreme hip flexion or hip flexion in combination with adduction and/or internal rotation. These hips tend to be stable in positions of extension, abduction, and external rotation. Most patients are instructed not to flex the hip greater than 90 degrees or adduct the leg across midline, especially during the first 6 weeks following surgery, during which time the soft tissues are healing. Patients are instructed not to sleep on the affected hip and to keep pillows between their knees to prevent adduction of the hip.

3. What are the different types of surgical approaches used for hip arthroplasty, and how do they affect rehabilitation?
   The most common surgical approaches performed today are anterolateral, direct lateral, and posterior. The anterolateral approach is performed by developing an interval between the tensor fascia lata and gluteus medius with either partial reflection of the medius or takedown of the greater trochanter to expose the underlying hip joint. After the components are placed, the gluteus medius is repaired or the greater trochanter is reattached. The posterior approach involves splitting of the gluteus maximus with takedown of the deep hip external rotators and conjoint tendon to expose the posterior aspect of the hip joint. After the components are placed, the posterior capsule and conjoint tendon are repaired. The anterolateral approach has been shown to have a lower rate of postoperative hip dislocation, as the posterior hip soft tissues are not violated. However, with this approach, time is needed to allow for the gluteus medius repair or greater trochanter osteotomy to heal, often restricting active hip abduction and full weight bearing. The posterior approach preserves the integrity of the gluteus medius and greater trochanter and allows wide exposure of the hip and proximal femur often needed for revision surgery. Dementia, mental retardation, Parkinson’s disease, stroke, or seizure disorders are relative contraindications to the posterior approach because of the greater potential for postoperative hip dislocation. Implications for rehabilitation include avoidance of active hip abduction exercises following anterolateral and direct lateral approaches for at least 6 weeks and more stringent adherence to total hip precautions following posterior hip approaches because of the potential for hip dislocation. With the direct anterior approach, medial to the tensor fascia latae (TFL), there is little need for any typical total hip precautions given the extremely low dislocation rate.

4. What are typical hip range of motion goals following total hip arthroplasty (THA)?
   Range of motion following THA usually advances rapidly. By the time of hospital discharge, patients should be able to extend to neutral and easily flex the hip to 90 degrees. Most patients will be able to achieve 110 to 120 degrees of hip flexion and will have the needed 160 degrees of combined hip flexion, abduction, and external rotation motion necessary to put on socks and shoes by 6 weeks after surgery.
5. You notice that a patient you are treating following THA has developed increased calf swelling and localized tenderness. What should you do?

An increase in calf swelling, calf pain with dorsiflexion of the ankle, calf tenderness, and/or erythema are all potential signs of deep vein thrombosis (DVT) and should prompt the therapist to contact the physician as soon as possible. These findings warrant the immediate attention of the physician so that appropriate studies may be obtained. The development of DVT following THA is very common despite the use of various types of DVT prophylactic measures (aspirin, warfarin, heparin derivatives, and sequential compression devices). Even with preventive therapy, rates of postoperative DVT following THA range from 10% to 20%. In spite of the high incidence of DVT, the rate of progression to fatal pulmonary embolism in unprotected patients is only 0.34%.

6. What are other typical complications associated with THA?

There are several serious but relatively infrequent complications, including loosening/osteolysis, dislocation, periprosthetic fractures, sciatic nerve injury, heterotopic ossification, and infection. Dislocation following THA is a multifactorial problem with reported rates of occurrence ranging from 1% to 10%. The majority of dislocations occurs within the first month following surgery. The prevalence of dislocation has been related to posterior surgical approaches, smaller prosthetic femoral head size, surgical technique, revision surgery, and patient compliance. Many dislocations can be treated conservatively with bracing and activity modification, particularly in the early postoperative period. Often, recurrent dislocation requires revision surgery. In a Mayo Clinic study of 19,680 hips, it was found that the incidence of dislocation was 1.8% at 1 year and 7% at 5 years, and increased 1% every subsequent 5-year period. The incidence of dislocation also increased after revision surgery to between 9% and 21%. Of the patients who had a dislocation, 16% to 59% had recurrent dislocations. Nerve injuries occur approximately in 1% of primary total hip replacements and 6% of revisions. The rate of nerve injury is higher in females than males. Functional recovery occurs in approximately 80% of patients. Nerve injuries can increase with approximately 1.5 cm of limb lengthening, and if the limb is lengthened 4 cm, significant nerve injury will be seen in 28% of patients. The femoral nerve and the peroneal branch of the sciatic nerve are more likely to recover than are the tibial branch or the entire sciatic nerve. Most patients who recover do so within 7 months, but recovery can continue for 2 to 3 years. Those approached from a direct anterior approach may experience thigh numbness or groin pain.

7. What are the outcomes following THA?

Survivorship analysis in multiple studies has shown acetabular and femoral components lasting 15 to 20 years with acceptable rates of survivorship ranging from 85% to 95%. Pain relief and improved function correlate well with survivorship of components for most patients, with good to excellent results in 85% to 95% of patients at 15 to 20 years. Postoperative limp has been associated with takedown of the greater trochanter and hip abductor muscles. Thigh pain has been associated with uncemented femoral stems. It has been found that the strength of the muscles surrounding the operated hip joint was 84% to 89% of the strength of the uninjured side in men and 79% to 81% of the strength of the uninjured hip in women. It was also found that significant residual muscle weakness persisted in the operated hip for up to 2 years following surgery. This persistent weakness could contribute to higher rates of component loosening. Physical therapy early in THA does restore range of motion, but significant impairments in postural stability remain 1 year after surgery. It is recommended that muscle strengthening exercises be continued for at least 1 year after THA.

8. When can patients with THA resume sexual intercourse?

Out of 254 surgeons surveyed, 67% recommended return to normal sexual activity 1 to 3 months after total hip replacement surgery; 31% of the physicians permitted return to sexual intercourse in 4 weeks or less following surgery. In addition, the surgeons recommended that patients with hip revisions abstain from sexual activity for slightly longer time periods; because of the higher rate of reported instability, time is needed to allow for pericapsular and muscular healing. It was also recommended that extreme hip flexion, adduction, and internal rotation be avoided.

9. Can patients with total hip arthroplasties return to play tennis effectively? Do physicians recommend this?

The average return to tennis was 6.7 months (ranging between 1 and 12 months) when tennis was played approximately three times a week. National rating levels did not drop significantly—from 4.25 before surgery to 4.12 after surgery (with a range of 1–7). Before surgery, all patients had severe pain.
and stiffness while playing; this was decreased to 31% of patients after 1 year, and only 16% reported having pain at the time of the survey (which was a mean of 8 years following surgery). In a study of 28 physicians surveyed at the Mayo Clinic, 3 approved of total hip replacement patients returning to tennis; 9 approved only doubles tennis.

10. Can patients with total hip arthroplasties return to play golf effectively? Do physicians recommend this?
Most golfers returned within 3 to 4 months following surgery, and some returned at 4 weeks after surgery. According to Hip Society surgeons, the recommended average return to golf was 19.5 weeks, with a range of 12 to 52 weeks. On average, patients’ handicaps increased by 1.1 strokes. Patients also noted increased drive length by 3.3 yards; 92% of patients reported no discomfort while playing golf and only 6% noted having pain but stated that it was decreased from preoperative levels. Golfers with a cementless hip prosthesis were recommended to decrease golfing activities for 6 to 8 months if they developed thigh pain while playing. Among doctors in the Hip Society, 69% requested that patients use a cart for the first year after THA. Of Hip Society surgeons, 96% permitted or did not discourage golf after THA, and 68.3% did not discourage patients who had THA revisions from playing golf.

11. Does exercise before THA improve outcomes?
Subjects who exercised before total hip replacement demonstrated progress that was 3 months ahead of that seen in the control group during early rehabilitation. The exercise group had two 1-hour supervised exercise sessions and also performed home exercises two times a week. The exercise group demonstrated greater stride length and gait velocity at 3 weeks after surgery. At 24 weeks postoperatively, in a 6-minute time test the exercise group was able to walk 549.7 meters, as opposed to 485.1 meters for the control group. Gait velocity was also faster in the exercise group at 24 weeks after surgery—1.57 meters per second, compared with 1.36 meters per second in the control group. A gait velocity of 1.22 meters per second is the guideline used by city engineers who set traffic signal crossing times.

12. What is the postoperative weight-bearing status of a THA patient?
Patients with cemented joint replacements can bear weight as tolerated, unless the operative procedure involved a soft tissue repair or internal fixation of bone. Patients with cementless or ingrowth joint replacements are put on partial weight-bearing or toe-touch weight-bearing regimens for 6 weeks to allow for maximum bony ingrowth to take place.

13. What types of patients are candidates for minimally invasive THA? What are the outcomes with this procedure?
Patients who qualify for a minimally invasive total hip replacement have a lower average body mass index, are thinner and healthier, and have fewer medical comorbidities. Patients are typically between 40 and 75 years of age and usually do not have larger, muscular frames. Minimally invasive hip replacements reduce blood loss, transfusion requirements, postoperative pain, and hospital stays. Dislocation rates have been found to be between 2% and 10%, and 35% of those patients do not have reoccurrence. Three times more patients ambulate on day 1 and 50% more patients meet all discharge criteria by day 3 with minimally THA: discharge criteria include the ability to transfer, ambulate with an assistive device, and negotiate stairs independently. The average time for patients to discontinue the use of crutches was 6 days, 9 days to walk independently without an assistive device, 10 days to resume activities of daily living, and 16 days average time to walk a half mile. Patients were able to return to walking with no limp, secondary to insufficiency of the gluteus medius. Average return to driving was 6 days, compared with between 4 and 12 weeks for THA patients.

14. What are the pros and cons of the different types of arthroplasty surfaces: metal-on-metal, ceramic-on-ceramic, and metal-on-polyethylene?
**METAL-ON-METAL**
- Pros—metal-on-metal provides a strong material that resists bending, torsion forces, and fatigue, which allows it to carry a sufficient load. Metal-on-metal has an initial rapid wear period for the first 1 to 2 years, but after this it has a lower and steadier wear. Metal has a 20 to 100 times lower wear rate than conventional polyethylene. Metal surfaces have been found to last over two decades. Wear rates have been found to be 25 to 35 mm per year for the first 3 years and then 5 mm per year thereafter, or 0.6 mm³ of metallic wear debris per year, which is an order of magnitude less than that of metal-on-polyethylene.
• Cons—metal-on-metal does produce metallic debris, which can be cytotoxic, altering the phagocytic activity of macrophages and leading to cell death. Metallosis and its effect on accelerating macrophage response can damage the shell or femoral neck. There are elevated ion levels in the blood and urine, effects of which are unknown. Hypersensitivity responses in the immune system are found in 2 out of 10,000 replacements. There are also possible links to cancer because cobalt and chromium have been found to cause cancer in animals, but more research must be done. The coefficient of friction is approximately two to three times greater than that for polyethylene. Metal-on-metal replacements have a higher cost, are heavier, and are stiffer. Periprosthetic soft tissue reactions can also occur, such as a pseudotumor, metallosis, and cyst formation.

CERAMIC-ON-CERAMIC
• Pros—ceramic-on-ceramic is resistant to chemical and mechanical dissolution. Ceramic is hard, strong, and resistant to oxidation and has high wettability. It has a low coefficient of friction and a scratch-resistant surface. Wear rates are 5 to 10 mm per year.
• Cons—ceramic-on-ceramic is brittle, and there is risk of fracture to the femoral head and acetabular component. Chipping can also occur with impingement to the hip. There are a limited number of femoral head and neck lengths and sizes that are ceramic. There is accelerated wear with higher degrees of abduction of the acetabular component. Ceramics are also high in cost and have increased rates of acetabular component loosening.

METAL-ON-POLYETHYLENE
• Pros—metal-on-polyethylene has low wear rates, costs less, and provides absence of oxidation. There is better adaptability and a forgiving nature of the bearing surface. There is low friction, long-term stability, and low water absorption. Cross-linked polyethylene has been found to have better wear rates than standard polyethylene.
• Cons—metal-on-polyethylene has a tendency to scuff the surface, wearing it away. Other cons with polyethylene are aging, creep, breakage, and abrasion. Polyethylene has a wear rate of 0.1 mm per year.

15. Are hip precautions necessary following THA with an anterior approach?
In a study performed with 2386 patients with 2612 hips replaced with an anterior THA approach, only four dislocations occurred (0.15%). Dislocations occurred between 3 to 12 days postoperatively. Patients were not given any functional restrictions, such as elevated seats, abduction pillows, or driving limits. Of the four dislocations that occurred, two happened when the patient was on the toilet, with one of these patients having previous hip fracture with a modular system. Another dislocation occurred in a patient with developmental hip dysplasia, and the last one was idiopathic. It was found that a no-restriction protocol did not increase the risk of early dislocation following THA with the anterior approach.

16. How does having a THA affect a patient going through airport security?
Eighty-four percent of patients can be expected to trigger an alarm and for wanding to be required with a handheld detector. About 70% can expect to be inconvenienced while traveling.

17. Do high-impact sports affect survivorship after THA?
Yes, there is an increase in risk of mechanical failure for patients after THA. Survivorship at 15 years after THA was about 80% for patients participating in high-impact sports, compared with 93.5% for those who did not.

18. Which type of THA provides better results, cemented or cementless?
Research shows that cemented acetabular components outperformed cementless after a 10-year period with a better survival rate. Cemented femoral fixation outperformed cementless in patients of all ages. Cementless components are continuing to improve but are still outperformed by cemented.

19. Is there an advantage to a minimally invasive THA versus conventional THA?
Minimally invasive has an advantage of decreased pain initially after surgery, earlier discharge to home, and less use of an assisted device. Evaluations at 6 weeks and 3 months post operation revealed no differences between the groups. There is no difference with regard to hip function and complication rates between the two groups.

20. When do patients following THA see the greatest results?
Patients can expect to see the most rapid gain in the first 12 to 15 weeks postoperatively in self-reported and physical performance. Slower recovery occurred in weeks 15 to 20. Patients seem to plateau at 30 to 40 weeks.
21. Is highly cross-linked polyethylene superior to conventional polyethylene?
In two different studies, it was found that cross-linked polyethylene had decreased wear rates compared with conventional polyethylene. One study showed cross-linked had one tenth the wear rate compared with conventional after 5 years. The other study demonstrated cross-linked had a 95% reduced wear rate compared with conventional polyethylene after 5 years.

22. Is direct anterior THA superior to THA done from the posterior approach?
No clear advantage has been consistently proven; however, trends toward reduced pain, reduced use of assistive devices, decreased dislocation rate, and decreased hospital stay are noted with the THA done from an anterior approach.

BIBLIOGRAPHY


CHAPTER 67 QUESTIONS

1. How much combined range of motion of hip flexion, abduction, and external rotation is required to put on socks and shoes following THA?
   a. 160
   b. 120
   c. 90
   d. 180

2. The majority of hip dislocations occurs during what time frame?
   a. 1 to 4 weeks
   b. 5 to 8 weeks
   c. 9 to 12 weeks
   d. After 13 weeks

3. When do most patients following THA return to golf?
   a. 4 months
   b. 6 months
   c. 9 months
   d. 12 months
1. What is a plica?
During embryonic development, the knee is initially divided into three separate compartments by synovial membranes. By the third or fourth month of fetal life, the membranes are resorbed, and the knee becomes a single chamber. If the membranes resorb incompletely, various degrees of separation may persist. These embryonic remnants are known as synovial plicae. Four types of synovial plicae of the knee have been described in the literature. The suprapatellar plica divides the suprapatellar pouch from the remainder of the knee. Rarely, this plica may imitate a suprapatellar bursitis or chondromalacia, and symptoms secondary to these conditions may be present. It courses from the anterior femoral metaphysis or the posterior quadriceps tendon to the medial wall of the joint. The mediopatellar plica is the most frequently cited cause of plica syndrome. It lies on the medial wall of the joint, originating suprapatellarly and coursing obliquely down to insert on the infrapatellar fat pad. This plica, sometimes known as a “shelf,” lies in the frontal plane. The lateral synovial plica is rare and poorly documented. This wider and thicker plica is located along the lateral parapatellar synovium, inserting on the lateral patellar facet. The plica found to be the least symptomatic of all—the infrapatellar plica or ligamentum mucosum—is ironically the most commonly encountered plica. This plica is seldom, if ever, identified as the cause for plica syndrome. This bell-shaped remnant originates in the intercondylar notch, widens as it traverses the anterior joint space, and attaches to the infrapatellar fat pad. The capacity for this plica to block or obscure arthroscopic portal entry sites or interfere with visualization may be its only known significance.

2. Describe the symptoms of an irritated plica.
The exact symptoms will be determined by the location of the irritated plica. The most common symptom location is along the medial (inside) side of the knee. If the plica connects the patella to the femoral condyle, symptoms will mimic patellofemoral syndrome. The plica can refer pain to the medial meniscus and cause patients to describe pain “under the kneecap.” It causes discomfort with prolonged sitting, prompting the term “moviegoer’s sign,” because the knee is less painful in extension. An irritated plica also may cause “pseudolocking” as the knee is extended and may “pop” beneath the patella or “snap” over the medial femoral condyle.

3. Describe patella-trochlear groove contact as the knee moves from full extension to full flexion.
Classic open kinetic chain or nonweight-bearing descriptions of patellofemoral tracking suggest that during the initial 20 degrees of knee flexion, there is no contact between the patella and femur. At 20 to 30 degrees of knee flexion, the distal third of the patella makes contact with the uppermost portion of the femoral condyles, with initial contact occurring between the lateral femoral condyle and the lateral patellar facet. At 45 degrees of knee flexion, the middle third of the patella contacts the femur. At 90 degrees of knee flexion, the proximal portion of the patella makes contact. Finally, at full flexion the odd facets of the patella make contact. In summary, as flexion angle increases, the contact area moves from proximal to distal on the femur and from distal to proximal on the patella. Additionally, femoral rotation creates increased patellofemoral contact pressures on the contralateral patellar facets, and tibial rotation creates increased patellofemoral contact pressures on the ipsilateral patellar facets.

4. Patella baja may result from adhesions caused by disruption of what bursa?
The infrapatellar bursa is located between the undersurface of the distal patella and the anterior proximal tibia. It can be violated during two types of surgeries: 1) distal extensor mechanism realignment when the surgeon medializes the tibial tuberosity and 2) harvesting of the central one third of the patella tendon
for reconstruction of the anterior cruciate ligament (ACL). After the bursa is traumatised, bleeds, and heals, adhesions form.

5. **What portion of the capsular ligament holds the menisci to the tibia?**
The capsular ligament of the knee is often called the coronary ligament. Anatomically the fibers of the capsule run proximal to distal. The capsule originates on the femur and courses first to the outer edge of the meniscus and then to its distal attachment on the tibia. The two distinct ligaments proximal and distal to the menisci are called the meniscofemoral ligament and the meniscotibial ligament, respectively. The meniscotibial portion of the capsule secures the menisci to the tibial plateau. Injury to the meniscofemoral portion leads to a less stable meniscal tear. If the capsule tears completely, swelling may leave the knee joint completely, giving the appearance of a milder knee injury.

6. **Describe the “lateral blow-out” sign of the knee.**
Because the anterior lateral portion of the capsule, just lateral to the patella tendon, is quite thin, Hughston and others refer to it as the “lateral blow-out” sign. When swelling is present in the knee, this area bulges outward, especially when the knee is flexed. Patients often deduce that they have a torn lateral meniscus.

7. **Discuss the role of the posterior oblique (POL) ligament.**
The POL is the predominant ligamentous structure on the posterior medial corner of the knee joint. The POL is located at the posterior one third of the medial capsular ligament, attaching proximally to the adductor tubercle of the femur and distally to the tibia and posterior aspect of the joint capsule. The POL plays a small role in preventing posterior translation of the tibia on the femur because the posterior cruciate ligament (PCL) is so overpowering. The main role of the POL is to control anterior medial rotatory instability and to provide static resistance to valgus loads when the knee moves into full extension. When an athlete makes a side-step cut, the POL contributes to keeping the pivot leg from opening in valgus, possibly acting in synergy with semimembranosus muscle activation. It also helps to prevent excessive tibial external rotation and femoral internal rotation.

8. **What is the importance of the “anterolateral ligament” (ALL)?**
The ALL was originally described by Gray as the “external lateral ligament” in 1858 and as having a fibrous band at the anterolateral human knee by Segond in 1879. It has also been referred to as the capsulo-osseous layer of the iliotibial and the mid third lateral capsular ligament. Claes et al. in 2013 detailed descriptive work regarding its anatomic characteristics. The origin of this structure is from the prominence of the lateral femoral epicondyle, slightly anterior to the origin of the lateral collateral ligament, although some connecting fibers were observed between the origins of each structure. Given its structure and location, this ligament is thought to assist with controlling internal tibial rotation and helping restrain the pivot shift phenomenon. Hughston et al. in 1976 described the same structure as more of a capsular thickening than a ligament, taking these observations to develop lateral extraarticular ACL reconstruction approaches. Because 5% to 15% of patients continue to experience knee instability following ACL reconstruction, it is possible that the ALL may have some role in providing rotational knee stability. Further research is needed to validate its functional significance.

9. **What important function does the arcuate complex provide?**
Each step at heel strike with the knee near full extension exerts tremendous force across the posterior lateral knee. The arcuate complex (posterior one third of lateral supporting structures including the lateral collateral ligament, the arcuate ligament, and the extension of the popliteus) helps to control internal rotation of the femur on the fixed tibia during closed kinetic chain function (or external rotation of the tibia on the femur during open kinetic chain function).

10. **How does the anatomic arrangement of the ACL dictate its function?**
The major functions of the ACL are to 1) stop recurvatum of the knee to control internal rotation of the tibia on the femur during open kinetic chain or nonweight-bearing function (external rotation of the femur on the fixed tibia during closed kinetic chain or weight-bearing function) and 2) stop anterior translation of the tibia on the femur during open kinetic chain or nonweight-bearing function (posterior translation of the femur on the tibia during closed kinetic chain or weight-bearing function). This action stops the pivot-shift phenomenon. Therefore the position of the ACL in extension of the knee elevates it against the intercondylar notch, acting like a “yard arm” to provide strength to the ligament and prevent recurvatum. Internal rotation of the tibia on the femur causes the ACL to tighten. The two main bundles of the ACL are the anterior medial and posterior lateral bundles. The posterior lateral bundle becomes more taut in extension, and the anterior medial bundle becomes more taut in flexion. This arrangement allows the ACL to control the pivot-shift through the complete knee flexion-extension range of motion. Innovative surgical techniques have been developed to reconstruct individual ACL
bundles to improve control of combined internal tibial torque and valgus torque; however, evidence regarding the implications of these techniques on improved patient function is currently lacking.

11. Can anatomic or “footprint” ACL reconstruction provide improved rotational knee control kinematics and clinical outcomes compared with conventional transtibial surgical approaches?  
Anatomic or footprint ACL reconstructions, particularly double bundle reconstructions, more closely restore normal knee kinematics using in vitro biomechanical studies and clinical trials. Therefore one would expect reduced evidence of knee osteoarthritis because normalized knee kinematics better replicate their preinjury state. In a meta-analysis of 19 randomized controlled trials, Xu et al. in 2013 found no difference in subjective patient outcomes between double- and single-bundle ACL reconstructions based on Lysholm, Tegner, and IKDC subjective scores. To date the clinical advantages associated with anatomic and double-bundle ACL reconstruction have been quite limited compared with conventional transtibial tunnel drilling (nonanatomic, single-bundle) techniques. This may be partially because of the limitations in clinical research study designs and measurement methods.

12. What is the function of the PCL?  
The major function of the PCL is to stop posterior translation of the tibia on the femur during open kinetic chain or nonweight-bearing function or anterior translation of the femur on the fixed tibia during closed kinetic chain or weight-bearing function. Its femoral and tibial attachments in the central knee joint enable it to be an ideal passive decelerator of the femur. The PCL is composed of three bundles, which allow some portion of the ligament to be taut throughout the range of motion. When the knee is in full extension, the posterior medial bundle of the PCL is most taut. Even when all of the other ligaments have been resected, the knee maintains some stability to varus and valgus forces when the posterior medial PCL bundle is intact. As the knee moves into flexion, the anterior lateral bundle becomes more taut. When the femur moves into external rotation during closed kinetic chain or weight-bearing function, or when the tibia moves into internal rotation during open kinetic chain function, the PCL becomes tauter.

13. What is the function of the iliotibial band? How does it contribute to the integrity of the knee?  
The iliotibial band (ITB) inserts at Gerdy’s tubercle or the lateral tibial tubercle. In this location it changes its function from extensor to flexor as the knee flexes at approximately 30 degrees. At near full extension, the ITB, through the action of the tensor fascia lata muscle, adds force to extend the knee. Once past 30 degrees, the tendon slips behind the horizontal axis of the knee, providing force for flexion. A portion of the ITB is the iliotibial tract. It has attachments into the linea aspera, which are very strong and help to prevent the pivot shift. Traditionally, surgeons have used it with certain techniques to substitute for an ACL-deficient knee (ITB tenodesis). In combination with the muscles of the pelvic deltidoid, the ITB and its fascial attachments contribute to composite lower extremity postural control during locomotion.

14. How does the ITB affect the pivot-shift test of the knee?  
The ITB plays an integral role in the pivot-shift test. As the knee flexes in the pivot-shift test, the ITB shifts posteriorly. The ACL and the middle one third of the lateral capsular ligament normally prevent the tibia and femur from shifting. However, in their absence, the pull of the ITB allows the shift to occur, with the tibia moving posteriorly and the femur anteriorly.

15. Describe the anatomic reasons for patellar instability.  
A high Q-angle (intersection formed by lines drawn from the anterior superior iliac spine to the center of the patella and from the center of the patella to the tibial tuberosity; normally 13 degrees in males and 18 degrees in females) predisposes the patella to sublux laterally. With the addition of a loose retinaculum, patella alta, and a weak or dysplastic vastus medialis obliquus muscle, the patella can easily sublux in the first 30 degrees of knee flexion. With a flattened lateral femoral condyle, the patellofemoral joint becomes unstable, even though the patella is seated in the trochlear groove.

16. Describe how patella alta can lead to patellar tendinitis.  
One of the roles of the knee extensor mechanism is to keep the femur from sliding forward on the tibia (dynamic back-up to the PCL). When a person decelerates, the knee is flexed and the patella should be in the trochlear groove. If patella alta is present, the patella may not be in the groove, thus increasing stress on the patellar tendon.

17. Describe the anatomy of articular cartilage.  
The superficial layer or tangential zone is composed of densely packed, elongated cells that contain 60% to 80% water. It is the thinnest articular cartilage layer and has the highest collagen content arranged
at right angles to adjacent bundles and parallel to the articular surface. This layer has the greatest ability to resist shear stresses and serves to modulate the passage of large molecules between the synovial fluid and articular cartilage. The superficial layer is the first to show changes with osteoarthritis. Next is the transitional layer with its rounded, randomly oriented chondrocytes (articular cartilage producing cells). The design of this layer reflects the transition from the shearing forces of the superficial layer and the more compressive forces of the deep articular cartilage layers. The radial layer is the largest articular cartilage layer. It is known for vertical columns of cells that anchor the cartilage, distribute loads, and resist compression. The calcified cartilage layer contains the tidemark layer (boundary between calcified and uncalcified cartilage). The tidemark layer is composed of a thin basophilic line of decalcified articular cartilage separating hyaline cartilage from subchondral bone.

18. Describe the arterial blood vessels of the knee.
Branches of the popliteal artery split and form a genicular anastomosis composed of the superior medial and lateral genicular arteries and the inferior medial and lateral genicular arteries. These vessels combine to give the ACL such a plentiful blood supply that a torn ACL results in generous bleeding and hemarthrosis of the knee after injury. The middle geniculate artery supplies the PCL.

19. Do the cruciate ligaments really cross?
From their tibial attachment sites at the anterior (ACL) and posterior (PCL) intercondylar areas, the cruciate ligaments cross before they attach to the lateral and medial femoral condyles, respectively. The cruciate ligaments also twist upon themselves during knee flexion and extension.

20. Describe the alignment of the femur and tibia during weight bearing.
The weight-bearing line or mechanical axis of the femur on the tibia is normally biased slightly toward the medial side of the knee, creating a 170- to 175-degree angle between the longitudinal axis of the femur and tibia, which is opened laterally. If this alignment is altered by degenerative changes, fracture, or genetic conditions, excessive stress is placed on either the medial or the lateral tibiofemoral joint compartment. Tibial varum or femoral valgus (angle greater than 170–175 degrees) leads to increased medial compartment stress, whereas femoral varum or tibial valgus (angle less than 170–175 degrees) leads to increased lateral compartment stress.

21. Are there differences between female and male knee joint anatomy and biomechanics?
No particular anatomic or biomechanic knee joint characteristic is unique to either gender. However, females tend to have a wider pelvis, greater femoral anteversion, more frequent evidence of a coxa varus–genu valgus hip and knee joint alignment with lateral tibial torsion, a greater Q-angle (18 degrees versus 13 degrees), more elastic capsuloligamentous tissues, a narrower femoral notch, and smaller diameter cruciate ligaments.

22. What is the normal amount of tibial torsion, and how does the physical therapist measure it clinically?
Tibial torsion can be measured by having the patient sit with his or her knees flexed to 90 degrees over the edge of an examining table. The therapist then places the thumb of one hand over the prominence of one malleolus and the index finger of the same hand over the prominence of the other malleolus. Looking directly down over the end of the distal thigh, the therapist visualizes the axes of the knee and of the ankle. These lines are not normally parallel but instead form a 12- to 18-degree angle because of lateral tibial rotation.

23. Which meniscus is most commonly injured and why?
Meniscal injuries most commonly occur at the medial meniscus. Although both menisci are prone to injury, the medial meniscus is at greater injury risk for both isolated and combined injury in the young athlete because of its adherence to the medial collateral ligament. In addition to transverse plane rotatory knee joint loads, any direct blows to the lateral aspect of the knee while the foot is planted may lead to injury at both the medial collateral ligament and the medial meniscus. Additionally, as a result of generally greater medial compartment weight-bearing loads during gait, the medial meniscus is more prone to degenerative tears as we age. The lateral meniscus is more often injured in combination with noncontact anterior cruciate ligament injury.

24. What is the function of the popliteus musculotendinous complex?
The popliteus musculotendinous complex functions as a kinesthetic monitor and controller of anterior-posterior lateral meniscus movement—for unlocking and internally rotating the knee joint during flexion initiation and for balance or postural control during single-leg stance. Increased
popliteus activity during tibial internal rotation with concomitant transverse plane femoral and tibial rotation lends support to the theory that it withdraws and protects the lateral meniscus, prevents forward dislocation of the femur on the tibia, and provides an equilibrium adjustment function. Popliteus activation may be most essential during movements performed in midrange knee flexion, when capsuloligamentous structures are unable to function optimally. The anatomic location, biomechanic function, muscle activation, and kinesthesia characteristics of the popliteus musculotendinous complex suggest that it warrants greater attention during the design and implementation of lower extremity injury prevention and functional rehabilitation programs.

BIBLIOGRAPHY


CHAPTER 68 QUESTIONS

1. The most common symptom associated with an irritated plica is ____________.
   a. Lateral retinacular laxity
   b. Mechanical joint locking
   c. Medial patellofemoral joint tenderness
   d. All of the above

2. The arcuate complex of the knee does not include the ____________.
   a. Lateral collateral ligament
   b. Extension from the popliteus tendon
   c. Posterior third of the lateral knee joint capsule
   d. Posterior oblique ligament

3. The posterolateral bundle of the ACL primarily serves to limit ____________.
   a. Tibial internal rotation during extension
   b. Anterior tibial translation during extension
   c. Tibial external rotation during flexion
   d. Posterior tibial translation during flexion
1. **What is the Q-angle?**
   The Q-angle is measured by extending a line through the center of the patella to the anterior superior iliac spine and another line from the tibial tubercle through the center of the patella. The intersection of these two lines is the Q-angle; the normal value for this angle is 13 to 18 degrees. Men tend to have Q-angles closer to 13 degrees, and women usually have Q-angles at the high end of this range. Because the Q-angle is a measure of bony alignment, it can be altered only through bony realignment surgical procedures. Despite the common opinion among clinicians that excessive Q-angle is a contributing factor to patellofemoral (PF) pain, it has not been shown to be a predictive factor in the outcome of patients with PF pain undergoing rehabilitation.

2. **What is the tubercle-sulcus angle?**
   A measurement similar to the Q-angle, the tubercle-sulcus angle is reported to be a more accurate assessment of the quadriceps vector. It is measured with the patient sitting and the knee at 90 degrees of flexion. The tubercle-sulcus angle is formed by a line drawn from the tibial tubercle to the center of the patella, which normally should be perpendicular to the transepicondylar axis.

3. **What may cause an increase in the Q-angle?**
   Excessive femoral anteversion, external tibial torsion, genu valgum, and subtalar hyperpronation can contribute to an increase in the Q-angle. When these conditions are found together, a patient is often said to have malicious or “miserable” malalignment syndrome. However, it should be noted that all of these static measures of angles (in isolation or in aggregate) are not strongly predictive of patellofemoral dysfunction.

4. **What anatomic structures encourage lateral tracking of the patella?**
   Bony factors, such as a dysplastic patella, patella alta, or a shallow intercondylar groove, can contribute to lateral tracking of the patella. Soft tissue structures, such as a tight lateral retinaculum or a tight iliotibial band (which has a fibrous band that extends to the lateral patella), can encourage lateral tracking of the patella.

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5. Define patella alta.
Patella alta refers to a cephalad position of the patella. Usually it is diagnosed by radiography and by determining the ratio between the length of the patellar tendon and the vertical length of the patella (Insall-Salvati ratio). The length of the patellar tendon is determined by measuring the distance between the inferior pole of the patella and the most cephalad part of the tibial tubercle. The normal ratio is 1:1. If the ratio is >1:3, the patient has patella alta. Patients with patella alta are more susceptible to patellar instability because the patella is less able to seat itself in the intercondylar groove.

6. What is the function of the vastus medialis oblique (VMO) muscle?
In their classic cadaver study of quadriceps function, Lieb and Perry reported that the primary function of the VMO is to counter the pull of the vastus lateralis and thus prevent lateral subluxation of the patella. They concluded that the ability of the VMO to contract and maintain patellar alignment throughout the full range of active knee extension enhanced the ability of the vastus lateralis to produce knee extension. Furthermore, when acting without the other quadriceps muscles, the VMO produced no knee extension.

7. How is chondromalacia classified?
The four types of chondromalacia are based on arthroscopic appearance:
- Type I—patellar surface intact; softening, swelling, “blister” formation
- Type II—cracks and fissuring in surface but no large cavities
- Type III—fibrillation; bone may be exposed; “crab-meat” appearance
- Type IV—crater formation; underlying bone involvement

8. How is PF pain classified?
Merchant classified patients according to five different etiologic factors: 1) trauma, 2) PF dysplasia, 3) idiopathic chondromalacia patellae, 4) osteochondritis dissecans, and 5) synovial plicae. These categories were subdivided into 38 subcategories. Others have classified patients with PF pain according to radiologic findings. A simple classification scheme that helps to determine treatment was proposed by Holmes and Clancy. The three major categories are PF instability, PF pain with malalignment, and PF pain without malalignment. In addition, Wilk et al. proposed a classification system that focuses on the underlying anatomic cause and presenting symptoms. The four major “rehabilitation” categories associated with this system require the clinician to recognize instability, tension, friction, and compression disorders and the specific protocols for their appropriate treatment.

Patellofemoral instability includes patients with patellar subluxation or dislocation—either recurrent or a single episode. First-time or infrequent subluxations and dislocations are treated with rehabilitation. Patients who continue to have problems after exhaustive therapy often require surgery.

PF pain without malalignment includes a number of diagnoses, such as osteochondritis dissecans of the patella or femoral trochlea, fat pad syndrome, patellar tendinitis, bipartite patella, prepatellar bursitis, PF osteoarthritis, apophysitis, plica syndrome, and trauma (eg, quadriceps or patellar tendon rupture, patella fracture, contusion). Most patients are treated conservatively with physical therapy, including hip and quadriceps strengthening, lower extremity stretching, and treatment of potential contributing factors.

PF pain with malalignment includes patients with increased Q-angles, tight lateral retinaculum, grossly inadequate medial stabilizers, patella alta or baja, and dysplastic femoral trochlea. Such patients often are treated with surgery only after an exhaustive trial of rehabilitation.
10. Describe the classification scheme of Wilk et al.

<table>
<thead>
<tr>
<th>Category</th>
<th>Affected Anatomic Area</th>
<th>Presenting Symptoms</th>
<th>Examination</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Instability (hypermobile patella)</td>
<td>Ligamentous structures (passive) or insufficient musculature (active)</td>
<td>Patellar instability (subluxation/dislocation)</td>
<td>Integrity of static patellar restraints</td>
<td>Avoid terminal knee extension</td>
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<td></td>
<td></td>
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<td>Medial and lateral patellar glides</td>
<td>Suggest exercise from 90 to 30 degrees</td>
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<td></td>
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<td>Use external support braces (taping, late buttress brace, pain-free ROM)</td>
<td>Open- and closed-chain exercise</td>
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<td>Open- and closed-chain eccentric exercise emphasized</td>
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<td>Plymouthers</td>
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<td>Stretch tight opposing muscles</td>
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<td>Physical agents and electromodalities</td>
<td>Avoid repeated flexion and extension exercises</td>
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<tr>
<td>Friction (soft tissue rubbing)</td>
<td>Friction points under sliding tissues</td>
<td>Pain with repetitive loaded flexion-extension</td>
<td>Observation of activity that replicates pain</td>
<td>Exercise in pain-free ROM; exercise above and below painful ROM</td>
</tr>
<tr>
<td></td>
<td>Commonly involved structures: ITB, plica, fat pad</td>
<td></td>
<td>Palpation of structures associated with common friction syndromes</td>
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<tr>
<td>Compression (articulart and periarticular compression)</td>
<td>Articular surfaces</td>
<td>Osteoarthritis, pain with function under load</td>
<td>Compression testing of PF joint via special clinical tests of functional movements that apply compressive loads to PF joint</td>
<td>Key is to increase quadriceps function to assist in absorbing weight-bearing loads</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td>Radiographic and other imaging studies helpful</td>
<td>Exercise in pain-free ROM in unloaded environment (pool)</td>
</tr>
</tbody>
</table>

ITB, iliotibial band; ROM, range of motion.
11. How can the system of Wilk et al. be applied to common anterior knee pain disorders?

<table>
<thead>
<tr>
<th>General Name/Disorder</th>
<th>Treatment Category</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lateral patellar compression syndrome</td>
<td>Compression</td>
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<tr>
<td>Global patellar pressure syndrome</td>
<td>Compression</td>
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<tr>
<td>Patellar instability</td>
<td>Instability</td>
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<td>Patellar trauma (depends on structure)</td>
<td>Compression or friction</td>
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<tr>
<td>Osteochondritis dissecans</td>
<td>Compression</td>
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<td>Articular defect</td>
<td>Compression or friction</td>
</tr>
<tr>
<td>Suprapatellar plica</td>
<td>Friction</td>
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<tr>
<td>Fat pad irritation</td>
<td>Friction or compression</td>
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<tr>
<td>Medial retinacular pain</td>
<td>Friction</td>
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<tr>
<td>Medial patellofemoral ligament</td>
<td>Friction or instability</td>
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<td>Iliotibial band syndrome</td>
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<td>Bursitis</td>
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<td>Osgood-Schlatter disease (apophysitis)</td>
<td>Tension</td>
</tr>
</tbody>
</table>

12. What is lateral pressure syndrome?
Lateral pressure syndrome, which can result in PF pain, is caused by a tight lateral retinaculum that pulls and tilts the patella laterally, increasing pressure on its lateral facet. Treatment includes stretching of the lateral retinaculum, such as medial glides/tilts and often includes proximal hip musculature through the iliotibial tract to thus "stretch" the distal iliotibial band. McConnell advocates quadriceps strengthening exercises with a medial glide of the patella with patellar taping. If rehabilitation is not successful, a lateral retinacular release often is performed.

Bipartite patellas still have an intact ossification center, most commonly at the superolateral pole. They are present in about 2% of adults and usually are asymptomatic. An anteroposterior radiograph of the bipartite patella may be mistaken for a fracture by the inexperienced eye. Extremely active people may irritate or disrupt this epiphyseal plate, causing PF pain. This area also can become painful after direct trauma to the patella. A bone scan may assist the clinician in diagnosing symptomatic disruption of the bipartite patella.

14. What is Sinding-Larsen-Johansson disease?
Sinding-Larsen-Johansson disease is apophysitis of the distal pole of the patella. Physical therapy intervention would consist of relative rest, temporary heel lift, light stretching of the gastroc/soleus, hamstrings and quadriceps muscle groups, and gentle strengthening that is pain free in nature progressing to functional activities.
15. Can a leg length discrepancy contribute to PF pain?
Few authors describe the precise relationship between PF pain and leg length. However, the common compensations that can result from leg length discrepancy theoretically may contribute to PF pain. Functional shortening of the longer lower extremity may involve excessive subtalar pronation, genu valgus, forefoot abduction, and/or walking with a partially flexed knee. All of these situations can distort PF mechanics.

16. Because articular cartilage is aneural, what tissues around the PF joint cause PF pain?
Normally, healthy articular cartilage absorbs stress across the PF joint. However, when the cartilage is not healthy, stresses are transferred to the subchondral bone, which is highly innervated. Subchondral bone is often thought to be the source of pain arising from the PF joint. Other structures around the PF joint also can cause peripatellar pain, including the infrapatellar fat pad, medial plica, bursa, and distal iliotibial band.

17. Define Hoffa’s disease.
Hoffa’s disease (fat pad syndrome) manifests as pain and swelling of the infrapatellar fat pad, usually from direct trauma to the anterior knee. Tenderness often is present at the anteromedial and anterolateral joint lines and on either side of the patellar tendon. A large fat pad also may become entrapped between the anterior articular surfaces of the knee with forced knee extension.

18. How is Hoffa’s disease treated?
Treatment normally begins with protection of the anterior knee, particularly during activities where repetitive contusion may occur. Local physical agents such as ice or ultrasound also may be used. Quadriceps strengthening should be performed to prevent weakness or atrophy resulting from disuse.

19. Describe the mechanism of pain stemming from the medial plica.
The medial plica is a crescent-shaped, rudimentary synovial fold extending from the quadriceps tendon to around the medial femoral condyle and ending in the fat pad. The medial plica can be injured with a direct blow to the knee or through overuse activities such as repetitive squatting, running, or jumping. Inflammation and edema can lead to stiffening and contracture of the plica. Contracted tissue running repetitively over the medial femoral condyle can cause pain and even erosion of the articular surface of the medial femoral condyle.

20. How is plica syndrome diagnosed?
Patients with plica syndrome have similar complaints as those with PF joint pain. Pain is aggravated by running, squatting, jumping, and prolonged sitting with the knee flexed. The most frequent clinical sign is tenderness located one finger’s breadth medial to the patella. The fold is often palpable, especially when the knee is flexed and the plica is stretched across the medial femoral condyle. Techniques designed to assess the presence of plica syndrome include the stutter test, Hughston’s plica test, and the mediopatellar plica test, but their sensitivity and specificity have not been reported. However, MRI is reported to have a sensitivity and specificity of up to 95% and 72%, respectively.

Housemaid’s knee is the layman’s term for prepatellar bursitis. This injury occurs when the prepatellar bursa is subjected to blunt trauma or repetitive microtrauma over the anterior knee, often found in individuals who work on their knees (carpenters or gardeners). Swelling in the prepatellar bursa occurs almost immediately and varies from slight to severe. Treatment consists of protecting the area from further trauma, applying ice, administering antiinflammatory medications, and performing exercises to maintain range of motion and strength.

22. Describe the mechanism for patellar dislocation.
The typical mechanism is external rotation of the tibia combined with valgus stress to the knee. Frequently this is actually the result of internal rotation of the femur over the tibia with the tibia thus becoming externally rotated and valgus associated with knee positioning. This is often related to strong quadriceps activation. Patellar dislocation also may result from blunt trauma that pushes the patella laterally.

23. What population is more susceptible to patellar dislocations?
Patellar dislocations occur slightly more frequently in women than in men. Patellar dislocations typically affect the adolescent population, with the frequency of their occurrence decreasing with age. Patients with patellar dislocation often experience recurrent episodes, especially adolescent patients.
24. What is the rate of repeat dislocation?
Reports in the literature on the rate of repeat dislocation vary. Repeat dislocation rates among first-time dislocations treated with immobilization are 20% to 43%. The rate depends to a significant degree on the presence of congenital predisposing factors such as PF dysplasia.

25. Can hip weakness contribute to PF pain?
From initial contact to midstance, the hip rotates internally. The external rotators must control this motion eccentrically. If the external rotators are weak, they may not decelerate internal rotation effectively. The result is excessive hip internal rotation, which functionally increases the Q-angle and encourages additional contact pressures between the lateral patellar facet and the lateral portion of the trochlear groove. Powers has proposed as an analogy for this movement the alteration of a train track under the train.

Hip extension weakness also can contribute to PF pain. During a weight-bearing activity such as climbing stairs, the hip and knee extensors work together to elevate the body. People with weak hip extensors may recruit the knee extensors to a greater degree, thus creating greater PF joint reaction force. By itself this reaction force may not cause a problem; in association with malalignment, however, it may contribute to PF pain.

Several researchers have increasingly examined hip weakness as either a result or a cause of patellofemoral pain syndromes. Proximal strengthening is now often a significant part of PFP rehabilitation. Numerous authors have now documented that utilizing a regional interdependence approach and strengthening the hip abductor, hip extensors, and hip external rotators results in improved function and a reduction of pain in patients suffering from PFP. It is unknown if PFP causes hip weakness or if proximal hip weakness contributes to or causes PFP.

26. What criteria are used to assess patellar instability?
1. Static approach—if the examiner can glide the patella laterally >50% of the total patellar width over the edge of the lateral femoral condyle, the patella is said to be unstable.

2. Dynamic technique—examiner observes patellar tracking as the patient moves from approximately 30 degrees of flexion to complete extension. If the patella makes an abrupt lateral movement at terminal extension, it may be considered unstable. This finding also is called a “J” sign because the patella follows the path of an inverted “J.”

27. Are radiologic studies useful?
Routine radiologic studies can show the depth of the intercondylar groove, level of congruence of the PF joint, presence of patella alta or baja, and patellar tilt. When instability is the focus, these tests are helpful as significant structural abnormality may limit the success of conservative measures.

28. What views are best to examine the PF joint?
The Merchant view provides an excellent view of the PF joint. The radiograph is shot with the patient in supine position with the legs over the edge of the examination table and the knees in approximately 45 degrees of flexion. The x-ray beam is aligned parallel to the femoral condyles. From this view, the clinician can see the shape of the articular surface of the patella and femoral condyles, PF joint space, medial and lateral facets, and degree of medial or lateral tilt of the patella.

29. Define the congruence angle.
The congruence angle is measured from a Merchant’s view and provides information about patellar position. A normal congruence angle is ±6 degrees (see figure). Studies have shown the normal congruence angle to be −6 degrees in men and −10 degrees in women. CT scans delineate this better than radiographs, and higher values tend to be associated with patellar subluxation.

30. Is MRI a useful tool to assess patients with PF pain?
With arthroscopy as the gold standard, McCauley et al. found that MRI had a sensitivity of 86%, specificity of 74%, and accuracy of 81%. The accuracy of MRI in identifying patients with chondromalacia patellae is excellent (accuracy of 89%) for identifying stage III or IV chondromalacia and poor for identifying stage I or II chondromalacia patellae.

31. Does strengthening of the quadriceps help patients with PF pain?
Almost all rehabilitation programs for patients with PF pain include some type of quadriceps strengthening exercises. Natri et al. examined 19 factors to determine the best predictors of positive outcome, with quadriceps strength being the single best predictor of outcome. According to Natri et al., the smaller the
difference in quadriceps strength between the affected and the unaffected extremity, the better the resultant outcome. Bennett and Stauber reported that a few weeks of concentric/eccentric quadriceps strength training within a pain-free range of motion obliterated an eccentric strength deficit and provided pain relief in patients with PF pain. Thomee compared 12-week isometric and eccentric quadriceps training programs in the rehabilitation of patients with PF pain and found significant increases in vertical jump height, knee extension torque, and activity level and decreases in pain for both groups. Recent reviews of the literature examining evidence for rehabilitation efficacy in these patients demonstrates very good evidence for the positive effects of pain-free strengthening but somewhat limited support for ancillary interventions.

32. Do all patients need to perform aggressive quadriceps strengthening exercises?
The answer can be found by examining the classification schemes for PF pain. Patients should be treated specifically, depending on the particular problem. In patients with patellar instability, aggressive quadriceps strengthening in the safe parts of the range of motion is a key component of rehabilitation. Patients with global patellar pressure syndrome may have a primary flexibility problem. Although quadriceps strengthening exercises are included in this rehabilitation program, stretching and mobility exercises are the main emphasis.

33. Does electromyographic (EMG) biofeedback strength training help patients with PF pain?
Few studies support the use of biofeedback training in the rehabilitation of patients with PF pain. Early research suggested a statistically significant increase in recruitment of the VMO compared with the vastus lateralis after 3 weeks of biofeedback training. The increase of 6%, however, is unlikely to be clinically meaningful. Ingersoll and Knight found that terminal knee extension exercises without EMG biofeedback resulted in a more lateral patellar position than performing the same exercises with VMO EMG biofeedback. The preponderance of the literature supports EMG biofeedback as an adjunct rather than as a primary focus.

34. What are the advantages of nonweight-bearing exercises for patients with PF pain?
Traditional nonweight-bearing strengthening exercises, such as seated knee extension, offer many advantages to patients with PF pain. Of primary importance, the knee joint and the quadriceps work independently during nonweight-bearing exercises. The only muscle group that can perform knee extension in the nonweight-bearing position is the quadriceps. Other muscle groups cannot substitute for weak or pain-inhibited quadriceps. Thus a maximal strengthening stimulus is provided for the quadriceps. In addition, ROM can be carefully controlled. Strengthening in a limited range can be easily achieved with most equipment. Finally, the amount of resistance also can be easily controlled with nonweight-bearing quadriceps strengthening.
35. What are the disadvantages of nonweight-bearing exercises?
Nonweight-bearing strengthening is often described as nonfunctional. The quadriceps muscles do not work in isolation during normal activities. Strengthening in the nonweight-bearing position does not train the lower extremity muscle groups to work together in synchrony or sequenced recruitment. In addition, in an exercise such as seated knee extension, the quadriceps are working maximally at end-range extension—the position at which the PF joint is most unstable. If the patient has PF instability and/or quadriceps imbalance that directs the patella laterally, the patella may easily track abnormally in complete extension.

36. What are the advantages of weight-bearing exercises for patients with PF pain?
The primary advantage is that the weight-bearing position is the position of function for the knee joint. An exercise such as the lateral step-up allows the quadriceps to train in synchrony with other muscle groups to complete the activity. Although the research supporting this concept is sparse, the law of specificity of training suggests this type of training should lead to the greatest improvement in functional performance. In addition, quadriceps activity is minimal as the knee approaches terminal extension. Therefore minimal quadriceps activity in the least stable position of the PF joint does not encourage lateral tracking of the patella. This advantage is especially important if the patient has patellar hypermobility or muscle imbalance that encourages lateral tracking.

37. What is the main disadvantage of weight-bearing exercises?
In the weight-bearing position, other muscle groups, specifically the hip extensors and soleus muscle, can contribute to knee extension force. Therefore patients with weakness or pain inhibition of the quadriceps may rely on other muscles to perform the knee extension. The result is insufficient stimulus for the quadriceps and minimal strength gains.

38. Are open-chain or closed-chain exercises better for a patient with PF pain?
Clinicians should focus on interventions that enable pain-free actions and target the underlying "cause" with an appropriate protocol (instability, tension, friction, compression). Integration of both open and closed activities appears optimal whenever possible. A recent review of the current evidence emphasizes the utility of an integrated approach that fits the specific presentation of the patient.

39. Can the VMO be strengthened in isolation?
This question is highly controversial. Some studies support the concept of preferential recruitment of the VMO. The VMO is more active than the vastus lateralis during hip adduction. Laprade et al. reported that the VMO is more active than the vastus lateralis with tibial internal rotation. It remains questionable whether the differences are clinically significant. Many studies do not support the concept of selective recruitment of the VMO over the vastus lateralis.

40. Is it better to perform quadriceps strengthening in a specific part of the knee’s range of motion?
The answer may depend on the patient’s specific problem. If lateral tracking or patellar instability is a concern, the patient should avoid strengthening in the last 40 degrees, where the patella is not well seated in the intercondylar groove. If lateral tracking or patellar instability is not a problem, strengthening in the range of 0 to 90 degrees is generally safe. At the other end of the spectrum, extreme amounts of knee flexion (>90 degrees) result in higher PF joint reaction forces and should be avoided.

41. Tightness of which muscles can contribute to PF pain?
Tightness of several musculotendinous groups has been implicated as a contributing factor to PF pain, including the gastrocnemius-soleus group, hamstrings, and iliotibial band. Inflexible plantar flexors may not allow full ankle dorsiflexion, which may result in a compensatory increase in subtalar pronation. This increase may encourage lateral tracking of the patella. Hamstring inflexibility is thought to cause an increase in quadriceps contraction to overcome the passive resistance of the tight hamstrings. The result is an increase in PF joint reaction force and quadriceps fatigue, as well as a decrease in dynamic patellar stabilization. Finally, the distal iliotibial band has fibers that attach to the lateral retinaculum. Tightness of the distal iliotibial band may encourage lateral tracking of the patella. The distal portion of the iliotibial band can be stretched by performing medial glides of the patella with the hip adducted. Patellar taping also provides a prolonged passive stretch to the retinacular tissues.

42. Should physical modalities be a part of the rehabilitation program?
Ice can be an effective modality to decrease pain and inflammation in patients with patellofemoral pain syndrome. In patients with inhibition of the quadriceps resulting from pain or effusion, electrical stimulation may aid in quadriceps muscle reeducation. It should be noted that the modalities are used
to facilitate a second intervention rather than serving as independent treatments in the vast majority of rehabilitation programs.

43. Is patellar taping (McConnell taping) an effective intervention for patients with PF pain syndrome?
Patellar taping is thought to improve “functional” patellar alignment and decrease pain to allow the patient to perform rehabilitation exercises more effectively. Many studies report a decrease in pain or an increase in knee extension moment with patellar taping. Whether the taping actually alters patellar position or alters neuromuscular responses of the lower limb (facilitation, inhibition, and proprioception) is still unknown. Taping has the advantage over bracing because it can be customized to fit the patient’s specific patellar alignment problem. However the reliability of patellar orientation assessment has been poor. The majority of tape use is probably associated with attempting to provide a medial pull (taping lateral to medial) on the “patella.” Past reviews have suggested that patellar taping is effective at reducing patellofemoral pain; however, more recent reviews indicate that the efficacy as a primary intervention is limited.

44. Is kinesio taping an effective intervention for patients with PF pain syndrome?
At this time there is weak evidence to support the use of kinesio taping for patients with PF pain syndrome.

45. Is bracing beneficial for the patient with PF pain?
Early reports suggested decreased PF pain in 93% of patients who used an elastic sleeve brace with a patella cutout and lateral pad. Shellock et al. used MRI to demonstrate centralization of the patella with a patellar realignment brace during active movement. Bracing generally is thought to “more likely” be beneficial in patients with patellar instability than in patients with patellar compression syndromes.

46. How is a patellar tendon strap supposed to alleviate PF pain?
One study reported success in 16 of 17 patients who used an infrapatellar strap. The proposed mechanism for the success of the strap was that it displaced the patella upward and slightly anteriorly. In addition, it was proposed that compression of the patellar tendon altered PF mechanics. Theoretically, elevation of the patella may slightly diminish PF joint reaction force, and compression of the patellar tendon may reduce excessive lateral movement of the tibial tubercle during tibial external rotation. A recent publication indicates that patellar tendon straps significantly reduce the measured strain at the site of a “jumper’s knee lesion” within a patellar tendon. Thus there are well-supported data indicating this device may be a valuable adjunct.

47. What is the relationship between foot mechanics and PF pain?
Multiple studies have demonstrated a relationship between foot posture and PF pain. Powers et al. found that patients with PF pain had an increase in rearfoot varus compared with controls without PF pain. Klingman et al. used radiographic analysis to show that orthotic posting of the subtalar joint in patients with excessive foot pronation results in less lateral displacement of the patella. These data indicate relationships but not necessarily “cause and effects.” Further research is required for definitive implications.

48. Are foot orthotics beneficial for patients with PF pain?
Many clinicians treating patients with PF pain provide anecdotal support for using orthotics. The clinician must treat the patient according to the classification of PF pain. If abnormal foot mechanics are suspected as an etiologic factor, orthotics may play a role in treatment. Eng et al., Lack et al., and Mills et al. showed that patients treated with soft orthotics and exercise had better outcomes than patients treated with an exercise program alone. The use of orthotics appears useful at least in some subsets of PFP patients.

49. When are distal realignment surgical procedures indicated?
Three disorders may require distal realignment procedures: PF instability, PF arthritis, or infrapatellar contracture syndrome. Criteria for considering realignment for each of these categories are outlined next.

**PF INSTABILITY**
- Three-quadrant medial patellar glide
- Tubercle sulcus angle $>0$ degrees
- Patella alta combined with generalized ligamentous laxity and flat trochlear groove
PF ARTHRITIS
• Significant PF chondromalacia or arthritis combined with PF instability indicates the need for anterior and medialized of the tibial tubercle.

INFRAPATELLEAR CONTRACTURE SYNDROME
• If after lateral release and debridement of the fat pad and infrapatellar tissues there is no change in patellar height, a proximal advancement of the tibial tubercle is indicated.

50. What are the long-term results of nonsurgical management of PF disorders?
Patients generally respond well to nonsurgical intervention. The long-term success rate is 75% to 85%. Multiple outcome studies support the importance of strengthening as the primary activity demonstrating efficacy. Thus it is appropriate to institute an individualized rehabilitation program before surgical intervention. If the rehabilitation program fails to provide adequate symptom relief and functional return, then surgery is a viable option.

BIBLIOGRAPHY
CHAPTER 69 QUESTIONS

1. Which of the following is a true statement regarding patellofemoral pain syndrome (PFPS)?
   a. Orthotics are not a useful intervention adjunct.
   b. Kinesio taping is effective at reducing PFPS.
   c. Patellar tendon straps are effective at reducing pain associated with PFPS.
   d. Closed chain exercises are superior to open chain exercises for patients with PFPS.

2. The majority of patellar dislocations occurs:
   a. Medial
   b. Lateral
   c. Superior
   d. Inferior

3. During the examination of a female patient with anterior knee pain, you note “a miserable malalignment presentation” when landing from a jump? Which of the below rehabilitation strategies would be the best regarding this patient?
   a. Hip flexor, adductor, and internal rotator strengthening
   b. Hip extensor, abductor, and external rotator strengthening
   c. Hip flexor, adductor, and external rotator strengthening
   d. VMO strengthening and hip internal rotation strengthening
1. How common are meniscal injuries in the United States?
The prevalence of meniscal tears is 66 per 100,000. The overall male to female incidence is 2.5:1. Peak incidence of meniscal injury in males is between 31 to 40 years of age; for females it is younger, at 11 to 20 years. Currently 850,000 meniscal surgeries are performed each year.

2. Describe the anatomy of the meniscus.
The menisci are wedges of fibrocartilage located on the articular surface of the tibia. The outer portion of the meniscus is thick and convex, whereas the inner portion is thin and concave. The menisci are composed of cells and an extracellular matrix of collagen, proteoglycans, glycoproteins, and elastin. The collagen content is 90% type I collagen, with the remaining 10% consisting of collagen types II, III, V, and VI. The collagen fibers are oriented circumferentially, which helps to transmit compressive loads. Cell types are fibroblastic in the outer third, chondrocytic in the inner third, and fibrochondrocytic in the middle third. The menisci are attached to the tibia at their anterior and posterior horns. The medial meniscus is more C-shaped, whereas the lateral meniscus is more O-shaped.

3. What structures attach to the medial meniscus?
- Joint capsule
- Deep medial collateral ligament (MCL)
- Coronary ligament of patella
- Meniscopatellar fibers from lateral border
- Semimembranosus tendon

4. Is the meniscus avascular?
No. The outer third of the meniscus is supplied by the branches of the geniculate arteries. The anterior and posterior horns are vascular, but the posterolateral corner of the lateral meniscus has no blood supply. The outermost third is called the red-red zone, the middle third the red-white zone, and the inner third the white-white zone. Healing is greatest at the outermost third and decreases with inward progression because of diminished blood supply.

5. List the functions of the meniscus.
- Helps to transmit loads across the tibiofemoral joint by increasing the contact surface area
- Viscoelastic properties add to shock-absorbing capacity
- Serves as secondary restraint to tibiofemoral motion by improving joint fit
- Helps with roll and glide of tibiofemoral arthrokinematics
- May assist in nutrition and lubrication of the joint

6. How important are the menisci in transmitting loads across the knee joint?
The medial and lateral menisci are responsible for carrying 50% to 60% of the compressive load across the knee. At 90 degrees of knee flexion, the percentage of the load borne by the menisci increases to 85%.

7. Do the menisci move with knee joint motion?
Yes. The lateral meniscus is more mobile because of its slacker coronary ligament. It does not attach to the lateral collateral ligament (LCL), whereas the medial meniscus attaches to the deep portion of the MCL. The lateral meniscus translates approximately 11 mm versus 5 mm for the medial meniscus. The menisci move posteriorly with knee flexion and anteriorly with extension. External rotation of the tibia is accompanied by anterior translation of the lateral meniscus and posterior translation of the medial meniscus.

8. What is a discoid meniscus?
It is a congenital deformity in the shape of the meniscus and is more commonly found in the lateral than medial meniscus. The abnormality affects the contact stresses and mobility of the menisci.
The discoid meniscus may cause symptoms of pain, effusion, or snapping. Partial meniscectomy may be required to create a more normal cartilage.

9. What is a meniscal cyst? Where is it likely to occur?
Meniscal cysts are ganglion-like formations secondary to central degeneration of the meniscus. They may occur on either meniscus but are more common on the lateral meniscus at the midportion or posterior one third. The patient may be asymptomatic or complain of a dull ache on the side of the cyst (medial versus lateral). Localized extraarticular swelling may be present and is proportional to the patient’s activity level.

10. What is the most common mechanism of meniscal injury?
The patient describes a turning or twisting maneuver of the leg in weight bearing. Most acute meniscal injuries are associated with ligamentous injury. Additionally, the meniscus may become injured when rising from a squatting position because of excessive compression of the posterior horn in association with an anterior translation of the menisci.

11. Which meniscus is more commonly injured?
Tears of the medial meniscus are more common than tears of the lateral meniscus.

12. What are the signs and symptoms of a meniscal tear?
The patient complains of symptoms such as catching or locking of the knee joint, pain with twisting of the knee, and tenderness along the joint line (77%–86%). In addition, swelling may be present (50% usually 24 hours after injury), especially with activity. Some patients complain of a “giving-way” sensation secondary to instability. A locked knee that will not fully extend usually indicates a large bucket-handle tear.

13. Describe the most common meniscal tears.
Meniscal tears are classified as longitudinal, vertical (transverse), or horizontal. Bucket-handle tears are classified as longitudinal tears that eventually separate and may cause locking of the joint. The parrot-beak tear is a pedunculated tag tear located on the posterior horn.

14. How accurate is magnetic resonance imaging (MRI) in detecting a meniscal tear?
MRI has a fair accuracy rate for detecting medial (88%) and lateral (88%) meniscal tears. According to Gelb et al., MRI has a sensitivity of 82% and a specificity of 87% for an isolated meniscal lesion.

15. Describe the McMurray test.
The McMurray test is the classic manipulative test for meniscal tear. The patient lies supine with the knee in full flexion. The tibia is rotated internally (lateral meniscus) and externally (medial meniscus) while valgus stress is applied and the knee is extended. A positive test is indicated by an audible or palpable “thud” or “click.” The McMurray test has a rather low sensitivity and a high specificity.

16. What other special tests are used to test for a meniscal tear? Do they have high sensitivity or specificity?

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<tr>
<th>Sensitivities and Specificities of Common Meniscal Tear Tests</th>
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<td><strong>CLINICAL TEST/SIGN</strong></td>
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<td>Joint-line tenderness</td>
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<td>Apley test</td>
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<td>Thessaly test</td>
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<td>Steinmann sign</td>
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17. Describe the Steinmann point tenderness test.
The Steinmann test is designed to evaluate meniscal tears. It is performed with the patient sitting and the knee flexed. A tender point along the medial or lateral joint line is located, the knee is either flexed or extended a few degrees, and the tender joint line is palpated again. If joint-line tenderness moves posteriorly as knee flexion increases, or anteriorly as the knee is extended, meniscal injury is indicated rather than capsular ligament pathology.

18. What is the typical management strategy for a meniscal tear?
Management falls into four main categories:
- Nonoperative—degenerative tears in older patients without mechanical symptoms
- Meniscectomy—symptomatic tears that are unable to be repaired and can preserve meniscal function
19. How effective is nonoperative treatment for meniscal tears?
Nonoperative treatment has good outcomes in older patients with degenerative tears without mechanical symptoms. Several studies have shown that a structured physical therapy program consisting of strengthening, flexibility, proprioception, and cycling results in the same favorable outcomes (6-month and 5-year follow-up) compared with those individuals undergoing meniscectomy alone or meniscectomy and physical therapy. Additionally, a recent study found that elderly patients with known osteoarthritis of the knee who underwent arthroscopic lavage or debridement were no better than a placebo surgery group (thus surgery may not be warranted in this population). However, it should be noted that approximately 30% of individuals with degenerative meniscal tears who undergo nonoperative treatment initially may require meniscectomy to achieve adequate pain relief. No current evidence exists related to the effectiveness of a conservative treatment approach for acute meniscal tears in younger populations.

20. What is the most common surgical management of meniscal injury?
Arthroscopic examination followed by partial meniscectomy is the most typical surgical management of meniscal injury. Short-term results suggest 90% satisfaction. Long-term satisfaction has shown that 50% of patients become symptomatic again at 5 years, and they have either modified or given up their sporting activities. Additionally, radiographic degenerative changes rose from 40% to nearly 90% over that same 5-year period.

21. When is a partial meniscectomy indicated?
Partial meniscectomy is indicated for younger or middle-aged patients with symptomatic tears (joint-line catching and pain, effusion, locking, and/or giving way that interferes with daily function) and in those with tears outside of the red-red zone that are not amendable to meniscal repair. Total meniscectomy is no longer considered a treatment option because of the significant increase in contact pressure that results in accelerated articular cartilage damage and pain.

22. What are the predictors of a poor outcome following partial arthroscopic meniscectomy?
- Low preoperative activity level
- >40 years old
- Higher than normal body mass index (BMI)
- Varus or valgus knee deformity
- Preexisting articular cartilage damage
- ACL insufficiency
- Radial tears
- Lateral meniscus tear

23. What is the usual time frame for return to function after partial meniscectomy?
Usually 2 to 6 weeks are required before return to function. Patients ambulate with crutches immediately after surgery and with no restriction in range of motion. Rehabilitation progresses rapidly.

24. When is a meniscal repair indicated?
Indications for repair are peripheral nondegenerative longitudinal tears <3 cm. Short tears of 1 to 2 cm have better success rates, and young patients seem to have the best outcomes.

25. What are the common forms of meniscal repair, and is one better than the other?
Meniscal repair is preferable to partial meniscectomy for salvaging the meniscus and preserving the tibiofemoral joint. There are four basic surgical approaches: 1) open (rarely used in contemporary practice), 2) inside-out suture techniques, 3) outside-in suture techniques, and 4) all-inside techniques. A systematic review examining different repair techniques found no differences in clinical failure rates of inside-out or all-inside techniques. It should be noted that more nerve complications have been noted with all inside-out techniques.

26. What is the clinical success rate following meniscal repair?
When looking at 10-year follow-ups, approximately 75% to 80% of patients undergoing meniscal repair were considered to be a clinical success. Clinical success was defined as low or absent pain and minimal radiographic changes in the tibiofemoral joint. Improved outcomes are seen in the repaired traumatic versus chronic tears (73% vs 42%) and when acute tears are repaired within 3 months of injury versus those repaired after 3 months (91% vs 58%).
27. What are contraindications for meniscal repair?
- Meniscus tears located in the inner-third region
- Chronic degenerative tears
- Longitudinal tears <10 mm in length
- Incomplete radial tears that do not extend into the outer third
- Patients older than 60 years of age
- Patients unwilling to follow postoperative rehabilitation
- BMI >35

28. Does bleeding stimulate the reparative process of a torn meniscus?
Yes. It has been shown that bleeding can stimulate the reparative process within a healing meniscus. It has been shown that patients with meniscal repairs performed in conjunction with ACL reconstruction have up to a 93% healing rate compared with 50% healing rates in meniscal repairs alone. Other interventions used to stimulate healing of the meniscus include an exogenous fibrin clot, which is placed at the site of meniscal injury to form a wound hematoma and trephination (shaving of the meniscus to promote bleeding) at the meniscus. Both of these techniques facilitate healing because of the release of local clotting and growth factors.

29. Can stem cells be used to treat a meniscal tear?
Some evidence has shown meniscus regeneration and decrease in pain following injection of human mesenchymal stem cells in individuals following partial meniscectomy.

30. What are the typical rehabilitation guidelines following meniscal repair?
No consensus exists on a universal rehabilitation protocol. However, general guidelines/milestones are offered herein. Please note time frames may be slightly longer in complex repairs.
- Long leg brace with lockouts—4 to 6 weeks
- Early protected gradual weight bearing (full weight bearing at approximately 5–6 weeks)
- Full ROM—4 to 6 weeks (90 degrees by 2 weeks, 120 degrees by 4 weeks)
- Mini squats—by 3 to 4 weeks
- Hamstring curls (0–90 degrees)—by 5 to 6 weeks
- Leg press (0–70 degrees)—by 5 to 8 weeks
- Stationary cycling—at 7 to 8 weeks
- Running—4 months
- Return to sport—5 months

31. Do surgically repaired menisci appear normal on MRI after 10 years?
A 13-year follow-up study of asymptomatic patients who underwent a previous surgical repair of the meniscus demonstrated abnormal MRI signals even though the meniscus had a stable union. These abnormalities at the site of repair represent edematous scar tissue, not the failure to heal.

32. What is meniscal repair using a bioabsorbable screw or arrow?
This is a relatively new technique that uses an all-inside device. All-inside devices for meniscal repair are attractive because they do not require additional incision or arthroscopic knot tying. An arrow or screw is inserted across the torn meniscus to bring the torn edges together and stabilize the tear. Some of the more recent devices have been designed to allow tensioning of the construct after insertion. This approach is less time consuming and has similar pullout strength to that of sutures used in a standard meniscal repair.

33. What are the outcomes of meniscal repair using a bioabsorbable screw or arrow?
Long-term studies are not available; however, short-term outcomes (12–24 months) are encouraging. In general, Lysholm scores postoperatively range from 80 to 90, and failure occurs in 7% to 10% of the repairs. Surgical complications and infections are minimal. A recent study demonstrated a 28% failure rate with postoperative complications, such as chondral scoring, fixator breakage, and postoperative joint-line irritation.

34. If a meniscal repair fails, can it be repaired a second time?
Yes; a case series of 14 patients who underwent a second repair had a success rate of approximately 72% after a 7-year follow-up.

35. What is a meniscal transplant?
For patients >19 to 50 years of age with severe irreparable meniscal injuries and symptoms, cadaveric meniscal implant is a potential option. Still considered controversial, meniscal transplantation in the
hands of a trained surgeon may be considered before a total joint replacement. Best results are obtained in those individuals who have a stable knee, no signs of advanced tibiofemoral arthritis, absence of varus and valgus deformities, and a BMI <35. A common surgical technique used in meniscal transplantation consists of drilling holes at the anterior and posterior horn attachments of the meniscus and inserting bone plugs that are attached to the cadaveric meniscal transplant and fixing them in place. Additional stabilization is afforded via soft tissue anchoring to the capsule/ surrounding soft tissues or modified bone plug or keyhole techniques.

36. What are the typical rehabilitation guidelines following meniscal transplantation? No consensus exists on a universal rehabilitation protocol. However, general guidelines/milestones are offered herein.

- Long leg brace with lockouts—4 to 6 weeks
- Early protected gradual weight bearing (full weight bearing at approximately 5–6 weeks)
- Full ROM—by 5 to 6 weeks (90 degrees by 2 weeks, 120 degrees by 4 weeks)
- Mini squats—by 5 to 6 weeks
- Hamstring curls (0–90 degrees)/leg press (0–70 degrees)—by 9 to 12 weeks
- Stationary cycling—at 7 to 8 weeks
- Running/sport—7 to 12 months

37. What are the outcomes associated with meniscal transplantation? Success rates (success being defined as a lack of persistent pain and mechanical integrity of the transplant) following meniscal transplantation vary from 12% to 100% (mean 60%). Long-term studies have shown transplantation survival rates of the lateral, medial, and combined transplants as 74% to 76%, 50% to 70%, and 67%.

BIBLIOGRAPHY


CHAPTER 70 QUESTIONS

1. Following meniscal repair, full weight-bearing starts at:
   a. 1 to 2 weeks
   b. 3 to 4 weeks
   c. 5 to 6 weeks
   d. 7 to 12 weeks

2. Which of the following surgeries has the best long-term outcome?
   a. Meniscectomy
   b. Meniscal repair
   c. Meniscal transplant
   d. All of the above have similar outcomes

3. Which of the following populations is best suited for a nonoperative approach for the management of a meniscal tear?
   a. Highly active patients
   b. Patients with ACL insufficiency
   c. Patients with a BMI >35
   d. Older patients with degenerative tears
1. What ligaments of the knee can be disrupted by a hyperextensive force?
   The first step to recurvatum is the anterior cruciate ligament (ACL). As the knee extends, the intercondylar shelf comes in contact with the ACL in midstance, tearing the ligament in a mop-end tear. The result is an isolated ACL tear. The patient shows a positive Lachman’s test. If the anterior lateral capsule is weak, the patient demonstrates positive pivot shift and anterior drawer tests with the tibia in neutral. The diagnosis is anterior lateral rotary instability.

2. Which ligament of the knee is the most likely to be disrupted by a motor vehicle accident in which the tibial tuberosity strikes the dashboard?
   The posterior cruciate ligament (PCL) is the ligament affected by this type of injury. The dashboard drives the tibia posteriorly until the patella and distal end of the femur reach the dashboard and stop the posterior movement. The result is an isolated PCL injury. Clinical testing indicates the loss of “step down” but no other instability. The diagnosis is straight posterior instability.

3. Which ligament is likeliest to be injured by a crossover cut maneuver?
   The ACL is subjected to severe internal rotation stresses. The middle one third of the lateral capsule assists the ACL in controlling internal rotation and varus stress. If the knee pops back into hyperextension during the cut, the potential for ACL injury is high.

4. Which structures of the knee can be injured during a side-step cut maneuver with valgus force?
   A side-step maneuver stresses the medial side of the knee as the lead leg steps to the side, the plant knee flexes, and the femur rotates internally as the tibia rotates externally. Valgus stress is applied across the medial side of the knee joint. The medial collateral ligament (MCL) or tibial collateral ligament resists the valgus force. The middle one third and posterior one third of the MCL provide the first resistance to rotation. If the force continues, the medial meniscus may be torn because of the stress across the meniscofemoral and meniscotibial ligaments. On the lateral side, the lateral meniscus may be impinged and damaged. Further force damages the ACL; if even more force is applied, the patella may dislocate, tearing the raphe of the vastus medialis obliquus (VMO).

5. How might an occult osteochondral lesion be associated with an ACL rupture, and where is it commonly found?
   Johnson et al. found that as many as 80% of all ACL disruption injuries have an associated osteochondral lesion. Lateral compartment chondral lesions associated with ACL ruptures occur as a result of the abnormal anterior and rotary translation of the tibia during common ACL mechanisms of injury. This lesion, or “bone bruise,” is commonly found in one of two locations: the lateral femoral condyle at the sulcus terminalis (anatomic junction between the tibiofemoral articular surface and the patellofemoral articular surface) or the posterolateral tibial plateau.

6. How might an occult osteochondral lesion associated with an ACL rupture affect long-term outcomes?
   Sixty percent of patients with a documented osteochondral lesion at the time of ACL rupture demonstrated persistent MRI evidence of osteochondral defect and symptom sequelae at 5.5 years’ follow-up. A significant percentage of patients who have immediate ACL reconstruction develop degenerative changes in the surgical knee within 5 to 10 years. Osteochondral lesions may be the event that predisposes the knee joint to this postsurgical degenerative osteoarthritis.
7. **What is a Segond fracture?**

A Segond fracture is an avulsion fracture of the anterolateral margin of the lateral tibial plateau, associated with ACL tears. The fracture is distinguished by a “lateral capsular sign” on radiographs and considered pathognomonic for ACL tears. The mechanism of injury is abnormal internal rotation stress of the tibia that causes abnormal tension on the central portion of the lateral capsular ligament, resulting in the avulsion.

8. **Does research consistently support the use of bone-patellar tendon-bone graft versus hamstring tendon graft for ACL reconstruction?**

No. Bone-patellar tendon-bone (BPTB) grafts and double/quadruple hamstring tendon (HT) grafts, using the semitendinosus and gracilis, continue to be the most widely used autologous grafts. Neither graft tissue has consistently been shown to be superior for ACL reconstructions. The incidence of postsurgical instability is not significantly different between BPTP and HT grafts; however, BTBP grafts are more likely to result in reconstructions with normal Lachman and pivot-shift testing, fewer incidences of reinjury postsurgically, and fewer cases of significant postsurgical flexion loss. On the other hand, research demonstrates that HT grafts have fewer incidences of patellofemoral crepitus, less kneeling pain, and fewer incidences of significant residual extension loss.

9. **Which ACL graft is better—allograft or autograft?**

The popular thought is that autografts provide a stronger and stiffer graft for ACL reconstruction. However, each has benefits and limitations. Allografts do not carry with it the donor site morbidity as is typically the case with autografts. Allografts, however, have a greater risk of transmitting infections, have incidences of rejections, and are a bit looser and less stiff than autografts. Allografts also have a history in the literature of demonstrating higher rates of graft rupture postoperatively. In a meta-analysis of over 5000 patients, Kraeutler et al. suggest a threefold increase in rupture rates (12.7% versus 4.3%) in allograft BPTB versus autograft BPTB. The bone–patellar tendon–bone (BPTB) autograft has long been considered the gold standard autograft, but recent literature may suggest that double-looped semitendinosus (DLST) hamstring autografts may now be used equally. BPTB grafts are associated with patellofemoral morbidity including decreased knee extension strength and patellofemoral pain, especially in patients returning to kneeling activities. DLST grafts are associated with hamstring morbidity and decreased knee flexion strength. The quadriceps tendon graft is also a possibility, but it is much less commonly used.

10. **What is the incidence of ACL injury in females versus males, and what are the anatomic, physiologic, and neuromuscular risk factors that could be responsible for a higher percentage of female athletes sustaining ACL tears over male athletes?**

Female athletes are 2.4 to 9.5 times more likely to sustain an ACL injury than male athletes. The following risk factors have been proposed:

**ANATOMIC RISK FACTORS**
- Less muscle mass per total body weight
- Greater joint hyperextension
- Greater joint rotational laxity
- Increased femoral internal rotation (IR), causing valgus stress at the knee
- Increased valgus stress at the knee secondary to increased Q-angle
- Increased femoral anteversion, causing increased valgus positioning at the knee
- Increased foot pronation, causing increased tibial internal rotation and valgus positioning at the knee
- Smaller diameter ACL housed in smaller intercondylar notch, although no consensus as to the role of notch size in ACL injury
- Smaller skeletal size

**PHYSIOLOGIC RISK FACTORS**
- Although there is no consensus in the literature, there have been studies correlating menstruation with ACL tears in women. Estrogen and progesterone receptor sites have been reported in human ACL cells. It has been proposed that levels of these hormones may have deleterious effects on the tensile strength of the ACL.

**NEUROMUSCULAR RISK FACTORS**
- Increased electromechanical delay (elapsed time between neuroactivation of the muscle and actual force generated secondary to increased extensibility of the musculotendinous unit)
• Contraction of quadriceps rather than hamstrings, which are supportive to the ACL, in response to anterior tibial translation
• Tendency for females to land from a jump and perform cutting activities in a more upright position with increased trunk, knee, and hip extension

11. What is the effectiveness, if any, of ACL prevention programs for female athletes?
The literature is scarce and there is little consensus on which regimens are most effective in ACL injury prevention. A recent study by Mandelbaum et al. showed as much as an 88% decrease in incidence of ACL injury at 1-year follow-up and a 74% reduction of ACL injury at 2-year follow-up after sports-specific prevention training for a sample of over 1000 female soccer players.

12. What is the epidemiology of ACL tears in the United States?
Tears of the ACL have been estimated to occur in 1 out of 3500 people in the United States each year. As a result, as many as 100,000 to 200,000 ACL reconstructions are performed annually. The incidence of ACL injury in females is 2.4 to 9.5 times greater than in males. Reinjury of the ACL reconstruction graft is 12% to 15% in males and 25% to 30% in females. About 70% of all ACL injuries are a result of sports participation; 10% to 15% of ACL injuries occur in soccer, 10% to 13% in skiing, 9% to 15% in football, 9% to 10% in baseball, and 8% to 15% in basketball. ACL injury is most common (1 in 1750) in the 15- to 45-year-old population. Sixty-one percent occur in the 15- to 29-year-old age group and 23% in the 30- to 44-year-old age group. About 70% of all ACL injuries are a result of noncontact mechanisms, and 47% of all severe knee ligamentous injuries involve the ACL (single tear).

13. Define anteromedial rotary instability. Which clinical tests are positive for this type of instability?
The classic mechanism of injury for anteromedial rotary instability is the football “clip.” The slightly flexed knee is forced into valgus while the tibia externally rotates. The structures that usually are disrupted are the MCL, posterior oblique ligament, middle third of the capsular ligament, and the ACL. The Lachman test, anterior drawer test, and valgus stress test at 20 to 30 degrees are positive.

14. Define anterolateral rotary instability. Which clinical tests are positive?
The classic mechanism of injury for anterolateral rotary instability is noncontact deceleration on a planted foot. The slightly flexed knee is forced into varus while the tibia internally rotates. The structures that usually are disrupted are the ACL, LCL, iliotibial band (ITB), and possibly the arcuate complex in the posterior lateral corner of the knee. The pivot shift test is positive.

15. Define posterolateral rotary instability. Which clinical tests are positive?
A varus blow from the anterior direction on a slightly flexed knee with the foot planted may result in posterolateral rotary instability (PLRI). The soft tissues involved in a PLRI are the arcuate complex (LCL, posterior oblique ligament, and popliteus tendon). The reverse pivot-shift, the posterior lateral drawer sign, the external rotation recurvatum test, and Loomer’s PLRI test may be positive.

16. Define straight medial knee ligament instability. Which clinical tests are positive?
If the knee receives a valgus blow in extension, the PCL, MCL, and middle one third of the capsular ligaments may be disrupted. In addition, the medial meniscus is pulled apart, and the lateral meniscus is compressed. Laxity noted with valgus stress test in extension indicates disruption to the PCL in addition to the MCL damage.

17. Define straight lateral knee ligament instability. Which clinical tests are positive?
If the knee receives a varus blow in extension, the PCL, LCL, ITB tract fibers, and middle one third of the capsular ligaments may be disrupted. In addition, the lateral meniscus is pulled apart, and the medial meniscus is compressed. Functionally the patient has difficulty on heel strike as the knee shifts laterally. Laxity noted with the varus stress test in extension indicates disruption to the PCL in addition to the LCL damage.

18. Why is there no such thing as posterior medial rotary instability?
In accordance with the logic of the other rotary instabilities, a posterior medial rotary instability would increase internal rotation of the tibia on the femur. Internal rotation of the tibia on the femur is controlled by the ACL and PCL. Therefore if the posterior medial corner is damaged, the cruciate ligaments stop the instability. By definition, posterior medial rotary instability cannot exist. If the PCL is torn, the patient has straight instability.
19. How accurate is a clinical examination for ACL injury?
A well-trained clinician can diagnose an ACL tear with the use of his or her hands and perhaps a knee arthrometer. The Lachman’s test is approximately 95% sensitive, and sensitivity decreases in larger patients. The pivot shift is pathognomonic of ACL rupture, but false-positive rates approach 30%. The prone alternate Lachman’s test may be significantly more sensitive (78%) than the anterior drawer (59%) or the standard Lachman’s test (28%) in patients with large thighs.

20. What is the incidence for reinjury following ACL reconstruction in athletes?
Incidence of a second injury to the ACL 1 year following ACL reconstruction and return to sport has been reported to be up to 15 times greater than in cohorts with no previous ACL injury. At 2 years post ACL reconstruction and return to sport, overall ACL injury rates in the same or contralateral leg are reported to be 6 times greater among athletes who undergo ACL reconstruction than those who have never had ACL injury.

21. Do all ACL tears require surgery?
The ACL and PCL are extrasynovial ligaments. When their sheaths are torn, they are subjected to the strong phagocytic action of synovial fluid. Although they have an excellent blood supply, the ligaments do not heal even when the ends are approximated by surgery. Once a tear of the ACL has occurred, the patient and physician must decide between surgical reconstruction or have the knee remain ACL-deficient and manage conservatively. Approximately 33% of ACL injuries require surgery immediately and another 33% require surgery after reinjury. The remaining ACL tears are managed through conservative, nonsurgical intervention. After sustaining an ACL tear, individuals can be categorized as a “cooper” or “noncooper.” ACL injury can result in tibiofibular laxity and impaired neuromuscular function, which may lead to dynamic instability and dysfunction of the knee joint. Individuals who opt for surgical reconstruction because of these adverse changes are termed “noncopers.” On the other hand, individuals who have ACL-deficient knees who demonstrate no dysfunction or instability and are able to resume preinjury activity with conservative, nonsurgical management are termed “copers.” There is little agreement within the literature as to the gold standard predictive factor(s) that lend the most insight into which individuals will be copers and which will be noncopers. The literature does show, however, that there is a similar number of copers and noncopers who return to preinjury levels and sporting activities.

22. In an open-chain active extension motion, where does maximal stress fall on the ACL?
In open-chain knee extension exercise, anterior translation of the tibia on the femur puts stress on the structures that restrict motion. The force is highest at 20 degrees of knee flexion (beginning at 45 degrees) and diminishes to very little force at full extension, when the quadriceps compresses only the tibia and femur.

23. Do open- and closed-chain exercises put equal amounts of stress on the ACL?
In vitro strain gauge studies indicate that closed-chain squats and open-chain knee extensions put almost equal amounts of strain on the ACL. If resistance is increased in either type of exercise, the strain increases.

24. Can an independent, home-based rehabilitation program be successful for the postsurgical ACL reconstruction patient who is unable to attend skilled physical therapy sessions?
A home-based program can be an effective alternative to supervised rehabilitation. No significant statistical differences in long-term outcomes have been found between groups who participated in home-based rehabilitation and those who participated in physical therapy in a clinical setting (provided there is ample monitoring of outcomes at regular intervals and attention paid to any warning signs by the physician and/or physical therapist). Studies suggest that for an athlete or patient who will be returning to a demanding, active lifestyle, phase III and phase IV of the ACL rehabilitation process, involving more advanced physical demands such as cutting and plyometric loading, had better outcomes when supervised by a skilled clinician. Other similar research suggests that there are higher dropout rates and lower patient satisfaction rates among patient groups performing a home-based program.
25. What criteria are used for the diagnosis of ACL tears with joint arthrometry?
   - Absolute translation >10 mm (at 20 lb)
   - Bilateral differences >3 mm
   If both criteria are met, arthrometry is 99% sensitive for ACL injury.

26. How accurate is magnetic resonance imaging (MRI) in detecting ACL and PCL tears?
   MRI accuracy for detecting ACL tear is 86%, specificity is 89%, and positive predictive value is 90%.
   For PCL tears, MRI accuracy is 98%, specificity is 98%, and positive predictive value is 75%.

27. How strong are the most common ACL grafts?
   Patellar tendon grafts tend to be 1–1.5 times stronger than the native ACL and double-looped hamstring 
tendon grafts about 1.5–2 times stronger. The human ACL has a stiffness of 182 to 292 KN/M, and 
can sustain a maximum load of 1725 to 2160 N. A 10-mm-wide bone-patella tendon bone (BPTB) graft 
demonstrates a stiffness of 424 KN/M and can sustain loads of 2071 to 2977 N. Double-looped 
semitendinosus grafts have a stiffness of 861 to 954 KN/M and can sustain maximum loads of 4304 to 
4590 N.

28. What are some common guidelines for activities after ACL reconstruction?
   - 50% of quadriceps strength for jogging
   - 65% of quadriceps strength for sports agility
   - 80% of quadriceps strength for full return to sports

29. What are the outcomes of ACL repair?
   About 88% to 95% of patients have a stable knee at 5-year follow-up, and 80% to 92% return to full 
previous level of play. Of patients with BPTB grafts, 10% to 40% have some anterior knee pain with 
average quadriceps strength losses of 10%. Patients with hamstring grafts have a lower incidence of 
anterior knee pain (approximately 6%).

30. Describe the grading system for collateral ligament injuries.
   - Grade 1—<5-mm joint-line opening with stress
   - Grade 2—5- to 10-mm joint-line opening with stress
   - Grade 3—>10-mm joint-line opening with stress

31. Compare third-degree injury of the ACL with third-degree injury of the medial 
compartment ligaments of the knee.
   Complete and even partially torn ACLs progress to complete demise over time. Complete rupture of 
the medial compartment ligaments with 1+ or 2+ anterior medial rotary instability heals to almost normal 
stability without surgical intervention.

32. What is the most commonly used graft for a PCL reconstruction?
   Achilles tendon allograft is the most popular graft site for both acute (43%) and chronic (50%) PCL 
reconstructions. Other grafts used to lesser degrees are bone-patellar tendon-bone autografts, 
hamstring tendon autografts, quadriceps tendon autografts, and anterior/posterior tibialis allografts.

33. Describe the general treatment strategy for MCL injuries.
   Grades 1 and 2 are treated nonsurgically with immobilization for 48 hours, followed by gentle ROM 
and progression of exercise as tolerated. Grade 3 injuries are treated similarly, but surgery may be 
indicated if residual instability or stiffness occurs.

34. Testing for medial knee instability in a 10-year-old boy after a valgus injury 
demonstrates a pathologic opening of the medial compartment using a valgus stress 
test. What should be the primary diagnosis?
   In a prepubescent individual, primary diagnostic thought should be an epiphyseal plate injury rather 
than an MCL sprain, as would be suspected in an adult. The reasoning is twofold: 1) the MCL is much 
stronger than the physes in a younger person, making it more prone to failure in a valgus stress 
injury mechanism, and 2) an epiphyseal injury is a much more serious injury in a young person than an 
MCL injury. An epiphyseal injury may require more aggressive medical treatment than an MCL sprain. 
An MCL sprain is often treated conservatively and therefore less of a long-term functional threat if 
initially misdiagnosed.
35. Describe the signs and symptoms of a lateral collateral ligament (LCL) injury.
The most common signs and symptoms of LCL injury are pain, stiffness, swelling, and tenderness along the lateral joint line of the knee. Walking pain typically is worse on heel strike than other parts of the gait cycle. Patients will present with a (+) varus stress test. Patients typically complain of instability, especially in pivoting activity or change of direction, resulting in the knee “giving way.” More severe tears can also cause numbness in the foot along with dorsiflexion and eversion weakness, which can occur if the peroneal nerve is stretched at time of injury or compressed by local edema or tissue post injury.

36. Describe the typical surgical procedures used to repair/reconstruct the lateral knee.
Many grade 1 and 2 LCL injuries can be treated conservatively using rest, antiinflammatories, and bracing to resist varus stress—usually for about 72 hours. Grade 3 LCL tears typically require surgical intervention. If the tear occurs mid tendon, the ends are typically approximated with sutures arthroscopically. In more severe grade 3 injury, where the LCL cannot be repaired, the lateral knee requires reconstruction, which is an open knee procedure and cannot be completed arthroscopically. Tendon autografts are typically harvested from the quadriceps or the hamstrings. The grafts are passed through bone tunnels in the femur and fibula and fixed using screws or posts, or sutures are tied around a post.

37. Describe the role that platelet-rich plasma (PRP) plays in the management of ACL tears.
The efficacy of using PRP in ACL tear management is currently under debate. The use of PRP in ACL reconstruction has been controversial, and the literature has yet to find a protocol that produces consistent results. Although some studies have shown that the addition of PRP to the bone tunnels and graft can reduce time required for maturation of the graft site by up to 50%, others have shown no significant improvement in ACL reconstruction outcomes with PRP versus traditional reconstruction. One possibility currently being explored is using PRP to promote healing of ACL tears, making repair a viable alternative to reconstruction. Synovial fluid in the knee joint has long been known to break down the fibrin-platelet clots needed for spontaneous healing or surgical repair of the ACL (past attempts at repairing ACL tears resulted in a greater than 90% fail rate). Finding a substitute clot through the use of PRP may be the answer to the advanced healing needed for ACL repair. At this point there continue to be many barriers to utilizing PRP for ACL repairs, not the least of which is its liquid consistency. This consistency makes it very difficult to use in anatomic areas such as the knee, without tight natural compartments. Although there is not a consensus on how to administer PRP or its efficacy, there are currently a number of research studies looking into how certain materials and tissues such as thrombin, collagen scaffolding, etc. can be combined with PRP to take full advantage of its healing properties, aiding in reconstruction outcomes and potentially even repair of the ACL.

BIBLIOGRAPHY


**CHAPTER 71 QUESTIONS**

1. Which structures are typically disrupted and what tests are positive in anteromedial rotary instability?
   a. MCL, ACL, posterior oblique ligament, and the middle third of the capsular ligament; Lachman’s test, anterior drawer test, and valgus stress test at 20 to 30 degrees are positive.
   b. ACL, LCL, IT band, and the arcuate complex on the posterolateral corner of the knee disrupted; pivot-shift test is positive.
   c. PCL, MCL, and medial third of the capsular ligament disrupted; valgus stress test in extension is positive.
   d. PCL, LCL, IT band, middle third of the capsular ligaments, and the medial meniscus disrupted; varus stress test in extension is positive.

2. Which structures are typically disrupted and what tests are positive in straight lateral knee instability?
   a. PCL, MCL, and medial third of the capsular ligament disrupted; valgus stress test in extension is positive.
   b. ACL, LCL, IT band, and the arcuate complex on the posterolateral corner of the knee disrupted; pivot-shift test is positive.
   c. ACL, LCL, IT band, and the arcuate complex on the posterolateral corner of the knee disrupted; pivot-shift test is positive.
   d. PCL, LCL, IT band, middle third of the capsular ligaments, and medial meniscus disrupted; varus stress test in extension is positive.

3. Which mechanism of injury would result in straight medial knee instability?
   a. The knee receives a varus blow in extension.
   b. The knee receives a valgus blow in extension.
   c. Noncontact deceleration on a planted foot
   d. The football “clip”: the slightly flexed knee is forced into valgus while the tibia externally rotates.
1. Is the patella typically resurfaced at the time of total knee arthroplasty (TKA)? What are the outcome differences?
Most surgeons advocate resurfacing the patella, especially in the presence of patellar chondromalacia, rheumatoid arthritis, and obesity. The decision of whether or not to resurface the patella has been investigated in several randomized trials. Some studies have shown no difference in subjective performance (ascending or descending stairs) or the incidence of anterior knee pain between resurfaced and nonresurfaced groups with short-term follow-up. Some studies have shown decreased pain and improved extensor mechanism strength in nonresurfaced compared with resurfaced groups. However, several authors have documented persistent anterior knee pain requiring repeat operation for patellar resurfacing following knee arthroplasty.

2. What is the weight-bearing status of most patients following total knee arthroplasty?
Most total knee arthroplasty components are placed using cement fixation. Cement fixation is stable immediately, allowing most patients to bear weight as tolerated on the involved lower extremity. Uncemented components generally rely on bone ingrowth into the component, which usually is present to some degree within 6 weeks following surgery. For this reason, patients with uncemented components usually have a restricted weight-bearing status during this period, most commonly 25% to 50% of full weight bearing.

3. What are the common knee range of motion goals following total knee arthroplasty?
Most patients who are able to achieve 75 degrees of knee flexion at the time of discharge will have at least 90 degrees of knee flexion at 1 year after surgery. The amount of knee flexion needed to perform various activities of daily living has been shown to range from 50 degrees while walking, to 80 to 90 degrees for stair-climbing, to 100 to 110 degrees for activities such as rising from a chair or tying a shoe. Most orthopedists consider 105 to 110 degrees the best long-term goal for knee flexion that will optimize patient function.

4. You notice that a patient you are treating following knee arthroplasty has developed increased calf swelling and localized tenderness. What should you do?
An increase in calf swelling, calf pain with dorsiflexion of the ankle, calf tenderness, and/or erythema are all potential signs of deep vein thrombosis (DVT) and should prompt the therapist to contact the physician as soon as possible. DVT following total knee arthroplasty is very common, despite the use of various types of DVT prophylaxis (aspirin, warfarin, heparin derivatives, and sequential compression devices). Rates of postoperative DVT, despite preventive therapy, range from 10% to 57% of patients following total knee arthroplasty, but the incidence of fatal pulmonary embolism in unprotected patients is only 0.19%. The reliability of physical examination findings for the detection of a DVT is notoriously inaccurate.

5. What is the difference between a posterior cruciate substituting and a posterior cruciate retaining knee replacement? How do they affect rehabilitation?
Posterior cruciate substituting systems require removal of both cruciate ligaments at the time of surgery. Knee stability is obtained through a design that allows the tibial intercondylar eminence to articulate within the femoral intercondylar box during knee flexion, thus preventing posterior translation of the tibia in relation to the femur. Posterior cruciate retaining designs spare the posterior cruciate ligament, allowing the ligament to retain its functional purpose. Clinical trials have demonstrated excellent results with both design types. Posterior cruciate retaining devices have the theoretical advantage of maintaining the proprioceptive function of the ligament. Additionally, posterior femoral rollback facilitated by the posterior cruciate ligament during knee flexion potentially allows greater knee flexion range of motion and improves the mechanical advantage of the quadriceps mechanism. One study has shown improved knee kinematics while ascending stairs in patients with posterior cruciate retaining knee replacements versus those with substituting designs.
Proponents of posterior cruciate substituting designs site greater ease of surgery, greater ability to correct
deforimities, and, most importantly, potentially decreased polyethylene wear rates as advantages of these
designs. Rehabilitation protocols are generally identical for both design types. Because of the rare reports of
posterior knee dislocation in cruciate-substituted knees, some studies advocate avoidance of resistant
hamstring strengthening in positions of extreme knee flexion.

6. What complications are associated with total knee arthroplasty?
There are many complications that are relatively uncommon, including peroneal nerve palsy (0.5%),
vascular injury (0.03%–0.2%), infection (1%–5%), periprosthetic fracture, extensor mechanism
dysfunction, wound healing complications, and arthrofibrosis. Peroneal nerve palsy is a serious
complication that may have permanent consequences upon ankle strength and control. The prevalence of
peroneal nerve palsy after total knee arthroplasty has been reported to be around 0.5%. The development
of nerve palsy has been associated with several risk factors, including preoperative valgus knee
alignment, preoperative knee flexion contracture, and epidural anesthesia for postoperative pain control.
In many instances, nerve function will return if diagnosed early and treated accordingly. The extensor
mechanism is the most common source of continued postoperative knee pain and can be related to patella
fracture, patellar tracking problems, parapatellar soft tissue impingement, and failure of patellar
components. Arthrofibrosis relates to scar tissue formation in and around the knee, resulting in restriction
of range of motion. This is treated with manipulation, aggressive physical therapy, and arthroscopic
release of scar tissue. Infection is a serious complication following total knee arthroplasty, with reported
rates of 1% to 5% depending on the patient population. Risk factors include revision surgery, delays in
wound healing, skin ulcers, rheumatoid arthritis, and, in some studies, urinary tract infections and
diabetes mellitus. Early infections can sometimes be treated with debridement and antibiotics, with later
infections often requiring removal of components.

7. What are the outcomes of total knee arthroplasty?
Outcomes following total knee arthroplasty are excellent in appropriately selected patients. Most clinical
studies following total knee arthroplasty report survival rates between 80% and 95% for the tibial
and femoral components at 10 to 15 years’ follow-up. Approximately 10% of patients will have pain
local to the patellofemoral joint. Up to 94% survival rates of tibial and femoral components at 18 to
20 years have been reported following cemented posterior stabilized total knee arthroplasty, with overall
survival rates of 90% when patellar revisions were included. Lower rates of success have been
demonstrated in certain well-defined patient populations. It is found that the greatest amount of
improvement is seen within the first 3 to 6 months after surgery, with more gradual improvements
occurring up to 2 years after surgery. Walking speeds for patients with total knee arthroplasties were
found to be 13% and 18% slower at normal and fast speeds, compared with subjects without knee
pathology. Stair climbing was compromised by 43% to 51% with patients following total knee
replacement compared with other subjects. Men with total knee arthroplasty were 37% to 39% weaker
and women were 28% to 29% weaker in their knee extensors compared with healthy individuals.

8. What are the indications for unicompartmental knee arthroplasty (UKA)?
- Greater than 60 years old
- Arthritis limited to one compartment of the knee
- Patient who is not overweight or a heavy-demand laborer
- Knee ROM >90 degrees with less than a 5-degree flexion contracture
- Angular deformity <10 degrees
- A functional ACL

9. What are the outcomes of UKA?
UKA may be converted to TKA with somewhat less difficulty than a proximal tibial osteotomy.
Augmentation with metal wedges is required in approximately 20% of the cases, and pain relief and
function are similar to those for primary TKA. Unicompartmental knee replacements are less invasive,
preserve the bone stock, are more cost effective, and have faster recovery times. Survivorship at 10 years
after surgery for patients <55 years old has been reported to be 87.5% to 96%, and for patients
≥60 years old survivorship rates are 94% to 98%. Average range of motion is 114 to 125 degrees after
UKA. Patients have shown a loss of torque of approximately 30% in extension and flexion at 60 to 180
degrees/sec of isokinetic testing compared with individuals without knee pathology. Patients with UKAs
showed no significant difference with regard to proprioceptive testing compared with normal controls.
Unicompartmental knee replacements also preserve normal knee kinematics, which are significantly changed in total knee replacements. A common cause of failure that would lead to revision is progression of arthritis at the patellofemoral joint and the contralateral compartment. Progression of arthritis to the lateral compartment can result from slight overcorrection into valgus, and better results are found when the knee is slightly undercorrected. It has been found that neutral to slight valgus is the optimal alignment for unicompartmental knee arthroplasty for anterior medial osteoarthritis. Overcorrection can shift the mechanical axis to the unreplaced compartment.

10. What are the indications for proximal tibial osteotomy?
- Less than 60 years old
- Arthritis limited to one compartment of the knee
- Patient who is not overweight or a heavy-demand laborer
- Knee ROM >90 degrees
- Varus angle deformity of 10 to 15 degrees
- Flexion contracture <15 degrees
- Enough strength to successfully use walker or crutches

11. What are the outcomes of proximal tibial osteotomy?
Typically there is approximately a 73% survivorship at 10 to 14 years, thus significantly delaying TKA for the appropriately chosen patient. Good to excellent results are slightly lower after conversion to TKA (63%) versus primary TKA (88%).

12. Can a patient kneel after total knee arthroplasty?
Two studies found that 64% and 82% of subjects tested were able to kneel after at least 6 months following surgery with minimal to no pain (rated 0–4 out of 10). Range of motion was a mean of 114 degrees in patients who could kneel comfortably and 110 degrees for subjects who could not kneel comfortably. Forces through the femoral component are similar in kneeling compared with those of walking and standing. Most patients avoid kneeling secondary to third-party advice in order to protect the prosthesis. Scar pain and back-related problems were also factors in limiting kneeling ability.

13. Can patients with total knee arthroplasty return to playing tennis? Do doctors recommend this?
Subjects returned to playing tennis approximately 5.9 months after surgery (range between 1 and 10 months). The average national tennis player rating level before surgery was 4.35 compared with 4.26 after surgery (range 1–7). Subjects noted that court speed deficiencies disappeared as quickly as 6 months after surgery (range between 1 and 12 months). Two subjects in the study needed revision, but they were also playing five times a week; the rest of the subjects were playing approximately three times a week. In a survey of surgeons, 21% believed that patients could return to playing singles tennis; 45% allowed doubles tennis only.

14. Can patients with total knee arthroplasty return to playing golf? Do doctors recommend this?
Surgeons’ recommendation for return to golf was approximately 3 months and to begin slowly. One study allowed for patients to start chipping and putting as soon as they demonstrated good balance and were no longer using assistive devices. A total of 77% of physicians have suggested that patients use a golf cart. Approximately 84% to 90% of patients reported having no discomfort while playing golf, and 34.9% had mild pain after golfing. Patients did note that pain was increased if their replacement was on their lead knee. In two different studies, 93% and 92% of surgeons did not discourage patients from golfing.

15. Does following an exercise program before total knee replacement surgery improve outcome?
Studies have found no significant difference between groups who followed an exercise program before surgery and a control group that did not exercise. It was found that preoperative levels of pain and function are the best predictors of pain and function at 6 months following surgery. These studies did have poor patient compliance, however. Implementing a strengthening program before surgery did improve patients’ general confidence, increased knee strength, augmented thigh muscle strength, and improved endurance after surgery. Patients with poor preoperative functional levels did not attain the same level of function at 2 years following surgery.
16. What is a rotating platform total knee arthroplasty?
A mobile-bearing rotating platform total knee replacement consists of a dual-surface articulation between a polyethylene insert and a metallic femoral tibial tray. The tibial tray has a conical cavity that articulates with the central cone of the one-piece polyethylene insert. This design increases articulation conformity, decreases polyethylene wear, and minimizes the shear stress at the tibial tray-bone cement interface. Rotating platforms provide unlimited axial rotation but with limited anterior, posterior, medial, and lateral translation of the femoral polyethylene insert. This was designed to approximate the kinematics of a natural knee. Dislocation rates were found to range between 0.5% and 4.65% and usually occurred in the early stage of total knee replacement, between 6 days and 2 years, although late dislocation has also been known to occur. Dislocation rates are higher in those patients with a preoperative valgus deformity and greater age at surgery. Average range of motion has been determined to be between 107 and 115 degrees of knee flexion. Survival rates have varied from 89.5% at 12 years, to 92.1% at 15 years, to 97.7% at 20 years. There has been no superiority found from a mobile-bearing rotating platform total knee replacement compared with a fixed total knee arthroplasty.

17. What are preoperative predictors for return to work following TKA?
The median time for patients to return to work was 9 weeks and can be as little as 4.5 weeks depending on the patient’s urgency to return. Factors that influenced a faster return to work included patients who were self-employed, have higher physical and mental functional scores, and a handicap-accessible workplace. Females returned faster than males. Slower return factors included having less preoperative pain, physically demanding job, and receiving worker’s compensation. Motivation is the greatest factor.

18. Is there an advantage of having a mini-invasive or computer-navigated total knee arthroplasty versus conventional TKA?
There is no statistical difference between mini-invasive versus conventional after 1 year for range of motion, Knee Society total, function scores, visual analog pain scores, and activities of daily living. There was no difference between computer-navigated and conventional TKA at 10-year follow-up for clinical function, alignment, and component survival.

19. What are indications for manipulation under anesthesia for a total knee arthroplasty? What are expected gains? When is it performed?
Knee manipulation is used primarily to increase knee flexion of less than 90 degrees. Patients with greater preoperative pain are more likely to have a manipulation. On average, knee flexion gains were found to be 22 to 35 degrees, and extension was 4 degrees. Knee range of motion after manipulation is similar to preoperative range of motion levels. Manipulation can be performed up to 12 weeks after surgery but was found to be most effective before 8 weeks.

20. What characteristics are commonly found in patients with knee flexion contractures following TKA? Are there surgical procedures to help prevent this?
Patients with flexion contractures following TKA typically have preoperative contractures, are older, and are more likely to be male. Flexion contractures greater than 5 degrees are correlated with poorer outcomes. Surgical techniques used to address preoperative flexion contractures include bone resection, posterior capsule release, ligamentous release, and removal of posterior osteophytes.

21. What are the risks and benefits of simultaneous TKA?
Patients receiving simultaneous TKA have a higher risk for cardiac complications, pulmonary complications, and mortality. Males have a higher complication rate than females. Other risk factors included advanced age (>65), presence of coronary artery disease, pulmonary hypertension, and congestive heart failure when considering simultaneous TKA. Simultaneous TKA did demonstrate a decreased risk for infection and mechanical failure. Patients would also have decreased time with rehabilitation.

22. Does preoperative osteoarthritis affect pain and dissatisfaction after TKA?
Patients who have less pain and decreased osteoarthritis before surgery actually had a higher percentage of pain postoperatively. Patients with less than a grade 3 or 4 may be at greater risk for pain and dissatisfaction after TKA.
23. Does obesity affect outcomes after TKA?
Obese patients showed some lower functional scores versus nonobese patients but still have substantial relief of symptoms after 9 years. There was no difference in implant survival rate for obese patients. Patients with a BMI of less than 35 only had a mild decrease in complication rates versus those with a BMI greater than 35.

24. Can TKA be done in an outpatient setting?
Kolisek demonstrated no significant differences in TKA done in an inpatient or outpatient setting, in appropriately selected patients.

BIBLIOGRAPHY


**CHAPTER 72 QUESTIONS**

1. How much knee flexion (degrees) is needed to ambulate stairs following TKA?
   - a. 60 to 70
   - b. 70 to 80
   - c. 80 to 90
   - d. 90 to 100

2. How long following TKA is manipulation most effective?
   - a. Before 8 weeks
   - b. 10 weeks
   - c. 12 weeks
   - d. 16 weeks

3. When do patients typically return to work following TKA?
   - a. 6 weeks
   - b. 9 weeks
   - c. 12 weeks
   - d. 16 weeks
**CHAPTER 73**

**KNEE FRACTURES AND DISLOCATIONS**

*R. “C.” Hall, DPT, ATC*

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**PATELLAR FRACTURES**

1. List, in order of frequency of occurrence, the five types of patellar fractures.
   - Transverse
   - Comminuted or stellate
   - Vertical
   - Osteochondral
   - Polar (apical or basal)

2. List the two major mechanisms of injury that result in patellar fractures.
   - Direct trauma (blow or fall) to the patella with significant articular cartilage damage
   - Indirect force (jumping) resulting in a displaced or transverse fracture

3. When is nonsurgical treatment indicated for a patellar fracture?
   - Minimal displacement (<2–3 mm)
   - Intact extensor mechanism
   - Minimal articular step-off (1–2 mm)

4. Describe the course of conservative treatment for patellar fractures.
   - Aspiration of hematoma and full extension in a long-leg cylinder cast or brace for 3 to 6 weeks
   - Quadriceps set and straight-leg raises with return to weight bearing as tolerated
   - Gradual progression of active knee flexion and strengthening after cast removal
   - Progression of closed-chain exercises (eg, biking) at 6 weeks, with a goal of return to full range of motion (ROM) and strength at 12 weeks

5. What are the common sequelae of patellar fractures?
   - The typical sequelae of patellar fractures is the following: patellofemoral arthritis, instability, decreased knee ROM, quadriceps weakness, and difficulty with stairs, downhill walking, and kneeling.

6. How is a bipartite patellar differentiated from a fracture?
   - On radiographs a bipartite patellar shows well-rounded, smooth margins and usually has one fragment in the superolateral position. Bipartite patellars occur in 0.05% to 2% of the population and are bilateral in 43% of the cases.

7. Describe the outcomes for nonoperative treatment of nondisplaced patellar fractures.
   - Most patients have full ROM and return to normal quadriceps strength without patellofemoral problems. Complications, such as nonunion or patellofemoral problems, occur in <2% of cases. Patient satisfaction has been reported as high as 95% or greater.

8. What are the outcomes for open reduction and internal fixation (ORIF) of patellar fractures?
   - Good to excellent results (return to full function within 6–9 months) are reported in 70% to 80% of all cases. Fair to poor results are reported in 20% to 30% of cases, and loss of the extensor mechanism is reported in 20% to 49% of cases. In one study, late displacement occurred in 7.4% of cases. Refracture has been reported in 5%. Prolonged immobilization (>8 weeks) increases the likelihood of poor results.

9. By what mechanism does the tension-banding technique stabilize patellar fractures?
   - The wires are placed in such a fashion that with knee flexion (increased quadriceps tension) the tension in the wires increases to intensify compression of the fragments and facilitate fracture healing.
10. How does rehabilitation differ between patients with nondisplaced fractures and patients with severely comminuted fractures?
Nondisplaced fractures are treated with knee immobilization in full extension, early weight bearing as tolerated, and isometric quadriceps exercises with a gradual increase in active assisted ROM at 4 to 6 weeks. Severely comminuted fractures with ORIF are treated like nondisplaced fractures but require partial weight bearing for the first 6 weeks and a gradual increase in active assisted ROM at 3 to 6 weeks (with demonstration of stable fixation). All other patients with ORIF may begin active assisted ROM at 1 to 2 weeks.

11. What are the outcomes for patellectomy?
Good to excellent outcomes have been reported in 22% to 85% of cases and fair to poor outcomes in 14% to 60%. Loss of quadriceps strength has been reported at around 50% decrease in peak torque. As a result, rehabilitation and return to function may be prolonged up to 6 to 8 months or longer.

12. At what age does a quadriceps tendon rupture typically occur? How do patients present?
Eighty percent of quadriceps tendon ruptures occur in patients older than 40 years. The mechanism of injury is forced knee flexion with maximal quadriceps contraction. Presentation includes intense pain, inability to walk, swelling, palpable defect, and hemarthrosis. Patients usually seek immediate medical attention.

13. How is a quadriceps tendon rupture treated? What is the expected outcome?
Repair is often primary anastomosis, with the knee immobilized in full extension for a minimum of 6 weeks, followed by 6 months of rehabilitation for full recovery. Acute repairs usually result in good recovery of ROM and strength sufficient for activities of daily living. A 20% decrease in quadriceps strength was reported in 50% of patients in one case series. Late repairs are at risk for significant extension deficit.

14. At what age does a patellar tendon rupture typically occur? How do patients present?
Patellar tendon ruptures most commonly occur in people younger than 40 years of age with a history of patellar tendonitis or steroid injections. Other pathogenesis includes long-standing tendinopathy, mucoid degeneration, and tendolipomatosis. Tendon ruptures are associated with high-energy trauma. Presentation is similar to that of a quadriceps tendon rupture, with a palpable defect and superiorly displaced patellar.

15. What is the incidence of repeat patellar tendon rupture following surgical repair?
The rerupture rate of patellar tendon rupture repairs is reported at less than 10%.

16. How are patellar tendon ruptures repaired? What is the expected outcome?
Ligament is sutured to bone, and the knee is immobilized in full extension for 6 to 8 weeks with <50% weight bearing. Earlier repairs have better outcomes than late repairs. Complications include decreased knee flexion and patellar baja.

DISTAL FEMORAL FRACTURES

17. What is the typical direction of displacement for a supracondylar distal femoral fracture? Why?
The distal fragment is flexed by the gastrocnemius, causing posterior displacement and angulation. The pull of the quadriceps and hamstrings causes the femur to shorten.

18. How are closed supracondylar fractures treated after reduction?
A cast brace is used for 6 to 8 weeks. If displaced and not reducible, supracondylar fractures may require ORIF. Skeletal traction is used less often.

19. What are the primary goals of operative treatment of distal femoral fractures?
- Anatomic reduction of joint surfaces
- Rigid fixation
- Restoration of limb length
- Early knee motion
20. What injuries are commonly associated with distal femoral fractures?
- Ipsilateral hip fracture or dislocation
- Peroneal nerve injury
- Vascular injury
- Damage to the quadriceps apparatus

21. Describe the age distribution of distal femoral fractures.
The age distribution is bimodal: 1) young males have a higher incidence of high-energy trauma and intraarticular damage, and 2) elderly women have a higher incidence of low-energy trauma with fractures secondary to osteopenia.

22. What are the indications and contraindications for operative and nonoperative treatment of distal femoral fractures?
- **Operative indications**—absolute: displaced intraarticular fractures, open fractures, neurovascular injury, ipsilateral lower extremity fractures, and pathologic fractures; relative: isolated extraarticular fractures and severe osteoporosis
- **Operative contraindications**—preexisting infection, marked obesity, comorbid conditions, poor bone quality, and systemic infections
- **Nonoperative indications**—nondisplaced or incomplete fractures, impacted stable fractures in elderly osteopenic patients, significant underlying medical disease (cardiac, pulmonary, neurologic), advanced osteoporosis, selected gunshot wounds, and nonambulatory patients

23. Why is fat embolism such a concern with femoral fractures?
The pathogenesis of fat embolism is a subject of conjecture and controversy. Most investigators agree that the bone marrow is the source of the fat. Fat embolism is associated more often with intramedullary instrumentation of the femur than with fracture. Fat embolism typically occurs in high-energy tibial or femoral fractures among patients between the ages of 20 and 40. Embolism is also common among elderly patients (60–80 years old) with low-energy hip fractures.

24. What are the outcomes for low profile minimally invasive plating for distal femoral fractures?
ROM averages 1 to 109 degrees, 93% heal without bone grafting, nearly all maintain fixation, malreduction occurs in approximately 6%, and infection occurs in 3% of patients.

25. How do distal femoral fractures present in children? What is the incidence of distal femoral fractures in children? What are the common mechanisms of injury?
Presenting symptoms for distal femoral fractures in children include inability to bear weight, maintaining the knee in flexion, gross deformity, and occasionally neurovascular compromise. Salter-Harris type II fractures are the most common category of distal femoral fractures in children (54%), with physeal fractures accounting for 1% to 6% of all physeal injuries in children.

- **Nondisplaced fractures** are treated with a long-leg cast or hip spica cast for 4 to 6 weeks.
- **Displaced Salter-Harris type I and II fractures** are treated with closed reduction with traction and gentle manipulation, followed by immobilization with or without percutaneous pinning. The position of immobilization depends on the direction of the displacement.
- **Displaced Salter-Harris type III and IV fractures** are treated with open anatomic reduction.

27. What are the indications for ORIF of distal femoral fractures in children?
Irreducible Salter-Harris type II fractures, unstable reductions, and Salter-Harris type III and IV fractures are all candidates for ORIF.

28. What complications are associated with distal femoral fractures in children?
- **Acute**—peroneal nerve palsy (3%) from traction or attempts at reduction, recurrent displacement, and popliteal artery injuries (<2%) associated with hyperextension injuries
- **Late**—angulation deformity (19%–24%), leg length discrepancy (24%–30%), knee stiffness (16%), avascular necrosis (rare), and nonunion (rare)
PROXIMAL TIBIAL FRACTURES

29. What are the general types of proximal tibial fractures?
   - Extraarticular—tibial spine, tibial tubercle, and subcondylar
   - Articular—condylar, bicondylar, and comminuted
   - Intraarticular—epiphyseal

30. What kinds of condylar fractures are often seen in the elderly?
    Insufficiency fractures of the medial tibial condyle are often found in the elderly. Varus deformity on examination usually indicates a depression or split-depression fracture (more common).

31. What injuries are associated with condylar fractures?
    Meniscal injuries occur in up to 50% of all condylar fractures and ligamentous injuries in 30%. Peroneal nerve neurapraxia and popliteal artery injury are also associated injuries.

32. Which tibial condyle is fractured more frequently? Why?
    The lateral condyle is fractured 70% to 80% more often because of weaker trabeculation, valgus orientation of the knee, and valgus-directed external forces.

33. Describe conservative treatment of nondisplaced condylar fractures.
   - Early passive exercise to maintain mobility and strength without weight bearing; weight bearing delayed until the fracture heals (6–12 weeks)
   - Nonweight bearing cast immobilization (long-leg foot-groin cast, 5 degrees of flexion) for 3 to 6 weeks, followed by 2 to 4 weeks of nonweight bearing rehabilitation, with progressive weight bearing from 9 to 16 weeks
   - Traction with passive exercise for 6 weeks, followed by nonweight bearing at about 12 weeks; return to full weight bearing when tissue healing is evident
   - Cast bracing with initial nonweight bearing and progressive weight bearing for up to 12 weeks; full weight bearing when tissue healing is evident

34. Describe the outcomes of low-profile minimally invasive plating for proximal tibia fractures.
    Approximately 91% heal without major complication, some malalignment occurs in 10%, need for hardware removal occurs in 5%, and infection is seen in 4%. Mean final ROM is approximately 1 to 122 degrees, with full weight bearing allowed an average of 12.6 weeks postoperatively.

35. Traumatic avulsions of the tibial tubercle are seen most often in what age group? Describe the mechanism and rate of injury for proximal tibial physeal fractures in children.
    Traumatic avulsions of the tibial tubercle are most often seen in young patients. Injury results from a strong quadriceps contraction with a slight degree of knee flexion. The sustained or sudden force disrupts either the tibial apophysis or the proximal tibial epiphysis.
    Proximal tibial physeal fractures account for 3% of all physeal injuries. Hyperextension forces the metaphysis posteriorly. Salter-Harris type II fractures are most common (35%).

36. How are proximal tibial physeal fractures in children treated?
   - Type I and II are treated with closed reduction followed by immobilization.
   - Type III and IV are treated with closed reduction, percutaneous pinning, and immobilization with an above-the-knee cast in 10 to 20 degrees of flexion for 6 to 8 weeks.

37. What complications are associated with proximal tibial physeal fractures?
    Vascular compromise occurs in 5% to 7% of cases, angular deformity in 28% of cases, and leg length discrepancy in 19% of cases.
38. Describe the weight bearing progression for the various fractures about the knee.

<table>
<thead>
<tr>
<th>Fracture</th>
<th>Surgical Fixation</th>
<th>Nonoperative Treatment</th>
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<tbody>
<tr>
<td></td>
<td>NWB (weeks)</td>
<td>FWB (weeks)</td>
</tr>
<tr>
<td>Patella</td>
<td>—</td>
<td>Immediate 6</td>
</tr>
<tr>
<td>Distal femur</td>
<td>—</td>
<td>Immediate 12</td>
</tr>
<tr>
<td>Proximal tibia</td>
<td>2–4 weeks 9 weeks</td>
<td>16*</td>
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NWB, nonweight bearing; PWB, partial weight bearing; FWB, full weight bearing.

*Based on signs of healing.
†Minimally displaced fractures.
‡After traction.

KNEE DISLOCATIONS

39. What is the frequency of vascular and nerve injury following knee dislocation?

One large retrospective study of 267 patients who had knee dislocations in the United States reported associated knee vascular injury at 3.3%. Another larger meta-analysis found a higher rate of vascular injury at 18%. This same study reported knee dislocation with an associated nerve injury of 25%.


Disruption of a popliteal artery presents with absent or decreased distal pulses and signs of ischemia. The artery must be repaired within 8 hours of injury to avoid limb amputation. If timing allows, a vascular surgeon may elect to perform an arteriogram to rule out an intimal tear. In clinically ischemic legs, however, the surgeon may proceed directly to open exploration and repair.

41. Should repair of ligament tears be acute or delayed in knee dislocations?

This issue is somewhat controversial. Some authors advocate acute repair of arterial structures with delayed repair of ligamentous structures to allow better healing of vascular repairs. Others advocate acute repair of both vascular and ligamentous structures with limited early motion, depending on the extent of the vascular repair. The literature slightly favors acute ligamentous repair, because early motion results in fewer postoperative complications.

42. Does the use of a hinged external fixator provide for better outcomes in knee dislocations?

Although the use of a hinged external fixator does significantly reduce ligament reconstruction failure in knee dislocations, overall it does not appear to enhance the chances of return to full function in patients with such devastating injuries.

PATELLAR DISLOCATIONS AND SUBLUXATIONS

43. What are the anatomic characteristics of typical patients with patellar dislocations?

<table>
<thead>
<tr>
<th>Anatomic Characteristic</th>
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<tbody>
<tr>
<td>Genu valgum</td>
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<tr>
<td>Shallow lateral femoral condyle</td>
</tr>
<tr>
<td>Elongated patellar tendon</td>
</tr>
<tr>
<td>Deficient vastus medialis</td>
</tr>
<tr>
<td>Lateral insertion of patellar tendon</td>
</tr>
<tr>
<td>Shallow patellar groove</td>
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<tr>
<td>Deformed patellar</td>
</tr>
<tr>
<td>Pes planus</td>
</tr>
<tr>
<td>Increased Q-angle</td>
</tr>
<tr>
<td>Ligamentous laxity</td>
</tr>
</tbody>
</table>
44. What type of fracture is frequently associated with acute patellar dislocations?
Osteochondral fractures of the medial facet of the patellar have been reported in up to 66% of all patellar dislocations. Osteochondral fractures of the lateral femoral condyle are also common.

45. What are the two main mechanisms of patellar dislocation and subluxation?
- Direct trauma or blow to the patellar with the knee in slight flexion
- Powerful quadriceps contraction combined with slight flexion and external rotation of the tibia on the femur

46. Describe the typical conservative course of treatment for a first-time patellar dislocation.
The course of treatment for first-time patellar dislocation is as follows: in the absence of osteochondral fracture, 6 weeks of brace immobilization in full extension; progressive weight bearing as tolerated; early quadriceps isometrics, with straight leg raising (SLR) as pain allows; passive pain-free ROM progressing to full active ROM and aggressive closed-chain strengthening at 6 weeks.

47. What factors contribute to recurrent instability after acute patellar dislocation?
Anatomic factors include trochlear dysplasia, patellar alta, injury to the medial patellofemoral ligament, connective tissue disorders, overall limb alignment, and poor muscle tone. Additionally, first-time dislocations have lower recurrence rates, and individuals experiencing two or more dislocations have a 50% higher risk of reinjury.

48. What are the indications for surgery with a patellar dislocation?
- First-time dislocation with significant osteochondral fracture
- First-time dislocation with inadequate or unstable reduction
- Recurrent dislocation not responding to nonoperative treatment
- Disruption of the medial patellofemoral ligament on magnetic resonance imaging

49. What are the indications and contraindications for lateral retinacular release?
- Indications—intractable patellofemoral pain with lateral tilt, lateral compression syndrome, persistent subluxations, patellar dislocations
- Contraindications—patellofemoral pain without lateral tilt, advanced patellofemoral arthrosis, lateral hypermobile patellar, normal tracking patellar, patellar subluxation and dislocation with significant extensor mechanism malalignment

50. How effective is reconstruction of the medial patellofemoral ligament (MPFL) for the treatment of instability?
MPFL reconstruction is somewhat technically demanding, with complication rates being reported around 26%. Proper patient selection has been reported to enhance success. Patients with severe trochlea dysplasia, femoral anteversion, and obesity lend to poor outcomes. Factors for success include accurate graft placement and tensioning to avoid overloading the medial patellar facet or and medial patellar subluxation.

51. What is the typical progression of rehabilitation following a lateral retinacular release?

<table>
<thead>
<tr>
<th>Weeks 1–2</th>
<th>Weeks 3–5</th>
<th>6 Weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight bear as tolerated</td>
<td>Full weight bearing</td>
<td>Sport-specific activities</td>
</tr>
<tr>
<td>Early AROM 1–115 degrees</td>
<td>Full AROM</td>
<td>Functional/isometric eval</td>
</tr>
<tr>
<td>Multitangle isometrics</td>
<td>Closed-chain exercises</td>
<td>Brace for activity</td>
</tr>
<tr>
<td>Patella mobilization</td>
<td>Aerobic reconditioning</td>
<td>Agility drills</td>
</tr>
<tr>
<td>Control pain/swelling</td>
<td>Isometric strength eval</td>
<td></td>
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</tbody>
</table>

52. When is a tibial tubercle osteotomy indicated, and what kind of outcomes can be expected?
Patients who have instability such as an increased tibial tubercle to trochlea groove distance of 20 mm or greater, patellar alta, or damage to distal lateral articular cartilage may benefit from this procedure. Long-term follow-up has reported that 80% of patients get good to excellent results.

53. What is the average recurrence rate after lateral retinacular release for recurrent patellar dislocation?
Published studies show a recurrence rate of as much as 5%, with a range of 14 to 48 months of follow-up.
54. What degree of tubercle-sulcus angle (Q-angle at 90 degrees) indicates potential patellar instability?
A tubercle-sulcus angle <10 degrees indicates potential patellar instability.

55. What radiographic view is used to assess patellar malalignment?
The Mercer-Merchant patellar view at 45-degree knee flexion angle is used to assess patellar malalignment.

56. What are the outcomes of medial retinacular repair with lateral retinacular release for acute patellar dislocation?
Good to excellent results have been reported in 81% to 91% of cases, with <2% redislocation rates.

BIBLIOGRAPHY

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CHAPTER 73 QUESTIONS

1. A 45-year-old man presents as nonambulatory stating that he slipped and hyperflexed his knee while jumping off his tractor. Examination reveals a large swollen knee, inability to actively extend it, and a palpable defect superior to the patellar. Which injury is most likely?
   a. Patella tendon rupture
   b. Bipartate patellar
   c. Quadriceps tendon rupture
   d. Tibial tubercle avulsion

2. A 45-year-old man presents as nonambulatory stating that he slipped and hyperflexed his knee while jumping off his tractor. Examination reveals a large swollen knee, inability to actively extend it, and a palpable defect superior to the patellar. Which injury is most likely?
   a. Patella tendon rupture
   b. Bipartate patellar
   c. Quadriceps tendon rupture
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3. A 45-year-old man presents as nonambulatory stating that he slipped and hyperflexed his knee while jumping off his tractor. Examination reveals a large swollen knee, inability to actively extend it, and a palpable defect superior to the patellar. Which injury is most likely?
   a. Patella tendon rupture
   b. Bipartate patellar
   c. Quadriceps tendon rupture
   d. Tibial tubercle avulsion
2. Which type of weight bearing status best describes treatment for a nonoperative minimally displaced proximal tibial fracture?
   a. Immediate partial weight bearing for 2 weeks
   b. 4 to 6 weeks nonweight bearing progressing to full weight bearing depending on healing
   c. Immediate partial weight bearing for 12 weeks
   d. Full weight bearing as soon as tolerated

3. Which of the following is true with regard to patellar dislocations?
   a. Patellar dislocations usually occur with the knee in full extension.
   b. First time patellar dislocations require 6 weeks of nonweight bearing.
   c. Medial patellofemoral ligament reconstruction has excellent outcomes.
   d. A shallow lateral femoral condyle is a predisposing factor to dislocation
1. In what order are sensory fibers normally lost after nerve injury?
   - Two-point discrimination
   - Light touch
   - Pinprick

2. Are motor fibers or sensory fibers the first to show electrophysiologically measurable signs of entrapment?
   Compression and subsequent ischemia affect large fibers more than small fibers, and fibers situated peripherally in the fascicle are more susceptible than centrally located fibers. Some researchers have documented that sensory nerve function is affected before motor function, whereas others have observed the opposite. Because sensory decrements are observed by the patient before subtle motor changes, the most prominent clinical sign is usually sensory. The largest fibers, irrespective of type, are affected first; both motor and sensory function need to be evaluated as part of the physical examination.

3. What constitutes compression of a peripheral nerve?
   An abridged list of factors that determine the effect of compressive force on peripheral nerves includes:
   - Manner in which the compressive force is applied
   - Type of underlying surface
   - Whether the nerve passes through or is contained within an unyielding compartment
   - Location and size of individual fibers and neural connective tissue
   - Magnitude and duration of the compressive trauma
   Nerves with increased amounts of connective tissue tend to be more resistant to compressive trauma, and nerves exposed to either long-lasting or high-magnitude compression demonstrate a greater degree of dysfunction. Epineurial blood flow is reduced at 20 mm Hg. Axonal transport is decreased at 30 mm Hg. Paresthesias occur at 30 to 40 mm Hg, and complete axonal blockade may occur at 50 mm Hg. Ischemia and motor blockade occur at pressures >60 mm Hg.

4. Describe the negative effects of compression on nerve function.
   - Endoneurial stasis of circulation caused by retrograde effects from the epineurial venule circulation
   - Anoxia of the endothelial cells of the endoneurial capillaries
   - Loss of integrity of the endothelial capillary tight junctions secondary to anoxia, resulting in increased vascular permeability
   - Leakage of fluid and proteins from the endoneurial capillaries into the endoneurial space, resulting in edema
   - Increased endoneurial fluid pressure
   - Antegrade and retrograde axonal transport systems also may be affected; low pressure compressions are the most common type with entrapment injuries

5. What nerve entrapments are found in the lower extremity?
   - Lateral cutaneous nerve of the thigh mononeuropathy (meralgia paresthetica)—lateral cutaneous nerve of the thigh (lateral femoral cutaneous nerve) is compressed medial to the anterior superior iliac spine as it passes under the inguinal ligament
   - Femoral nerve entrapment—femoral nerve is a mixed nerve that can be entrapped in the anterior abdominal wall as it passes under the inguinal ligament or in the femoral triangle
   - Obturator nerve entrapment—obturator nerve, although not usually compromised, is a mixed nerve that can become entrapped in the obturator foramen and as it passes through the obturator externus
   - Saphenous nerve entrapment—saphenous nerve is a cutaneous nerve that can become entrapped in the distal thigh as it passes through the adductor canal; it is the distal extension of the femoral nerve
• Piriformis syndrome—sciatic nerve can become entrapped as it passes through the piriformis muscle
• Common fibular (peroneal) neuropathy—both the superficial and the deep fibular nerve branches can become compressed as they pass around the fibular head
• Fibular neuropathy (superficial branch)—superficial fibular nerve can be compressed as it passes through the deep fascia of the anterolateral leg to become subcutaneous
• Fibular neuropathy (deep branch)—compression can affect the deep fibular nerve in the anterior compartment
• Ski boot syndrome (anterior tarsal tunnel syndrome)—deep fibular (peroneal) nerve can become entrapped at the ankle, most commonly as a result of tight-fitting shoes
• Tarsal tunnel syndrome—medial and/or lateral plantar nerve can be compressed at the ankle
• Sural nerve compression—purely cutaneous sural nerve can be compressed as it passes through the deep investing fascia of the leg or by an extrinsic source such as tight boots

6. How does lateral cutaneous nerve of the thigh mononeuropathy (meralgia paresthetica) present clinically? Describe its pathogenesis.
Presenting symptoms typically include altered or absent sensation over the lateral aspect of the mid thigh. Other sensory symptoms may include burning pain, dull ache, itching, and tingling over the cutaneous nerve field supplied by the lateral cutaneous nerve of the thigh (lateral femoral cutaneous nerve). The nerve is purely cutaneous and becomes superficial to supply the skin of the lateral thigh about 10 cm distal to the inguinal ligament. No loss in motor function should occur with isolated involvement of this nerve, apart from possible guarding secondary to pain with hip extension, which may increase symptoms.

7. Describe the cause and prognosis of lateral cutaneous nerve of the thigh mononeuropathy (meralgia paresthetica).
The cause may be tight clothing (tight underwear or tight jeans), pendulous abdomen, or rapid increase in weight. A variant in the normal path of exit from the pelvis also may increase the likelihood of entrapment. With the recent changes in the anterior approach to hip arthroplasty, there have been a number of studies reporting damage to the lateral cutaneous nerve of the thigh. The damage occurs either from direct injury to the nerve or as a result of traction of the nerve during the surgical procedure. Although rare, injury to the contralateral lateral cutaneous nerve of the thigh during total hip arthroplasty has been reported, due to the obesity of the patient and the surgical positioning. The prognosis is good in the vast majority of patients when the predisposing cause has been identified and removed, and the majority of paresthesias associated with surgery also resolve within a year. The peak incidence occurs during middle age, when progressive weight gains are also frequently observed. The incidence is equivalent on both right and left sides; symptoms may occur intermittently over a period of years, either unilaterally or bilaterally. Other forms of treatment include injection of an anesthetic agent with or without a corticosteroid in the area of suspected involvement.

8. What causes femoral nerve entrapment?
The femoral nerve can become compressed anywhere along its course by such diverse factors as tumors, psoas abscesses, lymph node enlargement, hematoma, or penetrating trauma. The nerve also can be compressed at the inguinal ligament or stretched when it is subjected to excessive hip abduction and external rotation (eg, during vaginal deliveries). With the move to the muscle-sparing approach provided by anterior hip arthroplasty, there have been a number of studies reporting damage to the femoral nerve. The observed damage may be due to the femoral nerve block used to deliver anesthesia, traction of the nerve during the surgical procedure, or direct injury to the nerve. The femoral nerve can also be damaged by tourniquet-related injury following knee surgery. Weakness in knee extension and possibly hip flexion, because of the involvement of the rectus femoris, may be noted. Sensation may be affected on the medial aspect of the knee and the anterior aspect of the thigh, which are supplied by the saphenous branch of the femoral nerve and the anterior cutaneous nerve of the thigh, respectively.

9. How does an obturator nerve entrapment present?
Obturator nerve entrapments are rare. When they occur, they usually are associated with acute trauma attributable to an event such as childbirth, pelvic trauma, or surgery. The adductor muscles supplied by the obturator nerve may be weakened, and sensation may or may not be decreased in the middle portion of the medial thigh. Problems noted by the patient include pain in the region of the inguinal ligament, instability of the lower extremity during gait, and atrophy of the adductor muscles.
10. What clinical manifestations are associated with entrapment of the saphenous nerve?

Before passing through the adductor hiatus, the saphenous nerve pierces the tough connective tissue layer between the sartorius and gracilis muscles to supply the skin of the anteromedial knee, medial leg, and medial side of the foot as distally as the metatarsal phalangeal joint. In some cases, the nerve also may pass through the sartorius muscle. The possible site of entrapment at this location is the point where the nerve passes through the thick connective tissue of the investing fascia and undergoes a sharp angulation. It is also possible to have a second site of entrapment as the infrapatellar branch of the saphenous nerve passes through the sartorius tendon. In this case symptoms are restricted to the infrapatellar region.

The most common complaint is knee pain, which may or may not be associated with sensory changes in the distribution of the saphenous nerve. Vigorous palpation at the point where the nerve pierces the subsartorial canal may reproduce the patient’s symptoms. Treatments range from injection of an anesthetic with or without corticosteroid to surgical decompression.

11. List four sites of potential fibular (peroneal) nerve entrapment.

- In the popliteal space behind the knee
- At the fibular head
- In the anterior compartment of the leg (as the deep fibular nerve)
- In the lateral compartment of the leg (as the superficial fibular nerve)

The common fibular nerve is the most commonly injured nerve in the lower extremity. Patients may present with clinical findings of weakness of the ankle dorsiflexors and toe extensors (e.g., footdrop), which may also be a consequence of a lumbar (L5) radiculopathy. The clinical and electrophysiologic evidence distinguishing between a common fibular nerve (peroneal nerve) mononeuropathy and an L5 radiculopathy are weakness and denervation in one or more of the proximal muscles of the lower extremity that have L5 innervation (e.g., tensor fascia lata, gluteus medius, semitendinosus, and short head of the biceps femoris). Additionally, a number of studies have shown that this nerve is susceptible to dysfunction from other causes, such as prolonged ice pack application, cysts, ankle sprains, cancer and the associated weight loss that accompanies it, and direct trauma. Some treatments, such as a short leg walking cast that distributes inappropriate pressure to the common fibular nerve just distal to the knee, can also result in a common fibular neuropathy.

12. Describe the clinical presentation of compression of the superficial sensory fibular nerve.

Approximately at the junction between the middle and distal third of the leg, the purely cutaneous continuation of the superficial sensory fibular nerve passes through the deep fascia to become subcutaneous. At this site, the fascia may be tough or restrictive, creating a potential point of entrapment. The terminal extensions of the superficial fibular nerve are the medial and lateral cutaneous branches, which supply the distal two thirds of the anterolateral leg and the dorsum of the foot, apart from the web space between the great and second toes. Symptoms are present along the distribution supplied by the nerve—over the distal leg and dorsum of the foot. Common injuries, such as an inversion sprain of the ankle, may stress this nerve at the point where it passes through the fascial opening.


Once the nerve has left the region of the fibular head and entered the anterior compartment, it is relatively protected and rarely entrapped, apart from problems associated with the anterior compartment. Again, distinguishing between a deep fibular nerve mononeuropathy and an L5 radiculopathy is paramount for both the clinical and the electrophysiological examinations. Anatomically, a compartment is created with the tibia medially, the fibula laterally, the interosseous membrane posteriorly, and a tough fascial layer anteriorly. Insults that involve this compartment can affect deep fibular nerve or anterior tibial artery function or muscle tissue directly. Examples range from anterior tibialis strain (shin splints: a mild form of anterior compartment syndrome) to muscle inflammation secondary to prolonged exercise, direct trauma to the leg, snake bites, or arterial bleeding. Significant increases in pressure are treated with fasciotomy—an incision of the anterior fascia of the leg.

14. Describe the tarsal tunnel.

The tarsal tunnel can be anatomically described as an anterior tarsal tunnel and a posterior tarsal tunnel (PTT). The more common and traditional use of the term tarsal tunnel syndrome relates to the nerve and vascular structures that may be compromised in the PTT. The anterior tarsal tunnel (ATT) is
located anterior to the talotibial and talonavicular joints where the deep fibular (deep peroneal) nerve and dorsal pedis artery pass beneath the inferior extensor retinaculum of the ankle. The PTT or tibiotalocalcaneal tunnel is posterior to the medial malleolus of the tibia. The tibial nerve usually branches into its four divisions—medial plantar, lateral plantar, medial calcaneal, and inferior calcaneal nerves—within the confines of the PTT. In the PTT, the posterior tibial artery usually branches into the medial plantar, lateral plantar, and medial calcaneal arteries.

15. What is anterior tarsal tunnel syndrome?
Anterior tarsal tunnel syndrome (ATTS), also known as ski boot syndrome, is caused by compression of the deep fibular nerve (DFN) as it passes deep to the inferior extensor retinaculum. ATTS is also seen in runners and soccer players who wear tight-fitting shoes, compressing the nerve in the region of the anterior ankle. After the deep fibular nerve passes through the ATT, it will provide motor function to the extensor digitorum brevis (EDB) and extensor hallucis brevis (EHB) and sensation to the web space between the great and second toes. The most common presentation involves only the sensory component; numbness and tingling are identified in the web space between the great and second toes. However, both motor and sensory fibers may be involved, in which case weakness may be identified in the EDB and EHB. Electrophysiologic testing, including sensory and motor nerve conduction studies of the DFN and needle EMG studies of the EDB and EHB, can be used to identify involvement of the distal aspect of the deep fibular nerve. Clinically, this nerve is sometimes compromised after repeated ankle sprains.

16. What is PTT syndrome?
The PTT is the region where the muscular, vascular, and nerve structures of the posterior compartment of the leg continue into the foot, passing between the medial malleolus and calcaneus in a tunnel created by the flexor retinaculum. In 90% of individuals, the tibial nerve splits into the medial plantar, lateral plantar, medial calcaneal, and inferior calcaneal nerves while still within the PTT. The medial calcaneal branch of the tibial nerve may bifurcate in this region, but its origin is highly variable and may occur proximal to, within, or distal to the PTT. Thus entrapment of the nervous structures in this region may affect the medial plantar nerve, lateral plantar nerve, medial calcaneal branch, or inferior calcaneal branch, or any combination of these nerves. Symptoms involving the plantar nerves include pain, burning, and paresthesias, often in the distribution of one or both plantar nerves. Recent studies have demonstrated effective techniques that facilitate the electrophysiologic examination of this region, including the medial plantar nerve, lateral plantar nerve, and the medial calcaneal nerve. The focus of these procedures has been to improve the consistency of the measurements and electrophysiologic signals.

17. Is tarsal tunnel syndrome a common problem? What branch of the plantar nerve is preferentially involved?
The diagnosis of posterior tarsal tunnel syndrome (PTTS) is not particularly easy because there are no hallmarks of the disorder. Atul et al. looked at the usefulness of EMG and NCS in the evaluation of PTTS and determined that sensory NCS may be useful in the identification of PTTS. However, EMG and motor nerve conduction studies are of limited benefit. An area of potential controversy is the actual extent of the PTT. Some clinicians refer only to the region under the flexor retinaculum as the PTT. Others refer to the entire region from the flexor retinaculum proximally to the metatarsophalangeal joint distally as the PTT. Both methods of describing the PTT are accepted although electrophysiologic testing of these structures may specifically demonstrate involvement of the individual plantar or calcaneal nerves. Sensory nerve conduction is the most sensitive electrodagnostic examination for possible compromise of the medial plantar nerve, lateral plantar nerve, or calcaneal nerve in the region of the ankle or foot.

The medial plantar nerve may be involved more frequently than the lateral plantar nerve, although the overall incidence of plantar neuropathies is relatively low. Steinitz et al. reported an incidence rate of 0.58% (51) after 8727 electromyographic examinations, similar to the 0.5% incidence found by Oh. Because repetitive pronation or foot hypermobility may stress the medial plantar nerve in activities such as jogging or jumping, the constellation of symptoms associated with medial plantar neuropathy has been called “jogger’s foot.” Determination of the extent of nerve involvement is an electrodagnostic challenge that requires detailed examination and meticulous technique.

18. What causes entrapment of the sural nerve?
In general, passage through the fascia of the leg is not a common site of entrapment; thus sural nerve compressions are relatively rare. When an entrapment occurs, it usually is associated with factors
such as a ganglion cyst, tight combat boots, or stretch injury. An important clinical point is that the sural nerve is often evaluated when generalized polyneuropathy is suspected. A decrease in nerve conduction velocity in this nerve as well as other major nerves of the leg (eg, tibial and deep peroneal) suggests polyneuropathy.

19. How sensitive and specific is electrophysiologic testing?
Most of the work investigating sensitivity and specificity in electrophysiologic testing has been conducted in the upper extremities, with values of greater than 85% sensitivity and 95% specificity reported for entrapments of the median nerve at the wrist. Other researchers have found, for the median nerve, the composite electrophysiologic measures for sensitivity range between 49% and 84%, with specificity values of 95% or higher reported. Assuming that these values can be applied to lower extremity entrapments, they indicate that although the electrophysiologic tests are quite good, they are not perfect. False-positive electrophysiologic findings have been reported, and the clinician using the information derived from these examinations must also consider the patient’s clinical presentation and clinical evaluation.

20. Are there regions of the lower extremity that have a tendency to generate electrophysiologic false positives?
Yes. Recent research identified that 21% of patients have abnormal needle electromyographic (EMG) results that suggested denervation, when examining the abductor hallucis intrinsic muscle of the foot. These abnormal electrophysiologic findings additionally appeared to increase with age, being most noticeable in individuals over the age of 60. A second muscle examined, the fibularis (peroneus) tertius, also demonstrated positive findings in normal subjects over the age of 60 but at a much lower rate of 9%. The reasons for these findings and the anecdotal reports of spontaneous abnormal EMG in the foot are unclear, but the findings may be associated with the fact that we ambulate on some of the intrinsic muscles of the foot or that these muscles and their vascular and nerve supplies are restricted by structures such as shoes.

Other studies have demonstrated a much more conservative prevalence of only 2% when examining foot intrinsics in normal subjects. Regardless of the cause, false positives are possible and relatively common with EMG of the small muscles of the foot.

21. What neurologic conditions should be considered in patients with bilateral lower limb numbness, tingling, and pain?
- Spinal stenosis
- Spinal cord tumor
- Radiculopathy
- Mononeuropathy multiplex
- Peripheral neuropathy

22. What are the various causes of lower limb peripheral neuropathy?
- Metabolic and endocrine disorders: diabetes, metabolic liver, and thyroid disease
- Small vessel disease: caused by diabetes and vasculitis
- Autoimmune diseases: Sjögren’s, lupus, and rheumatoid arthritis; acute conditions like Guillain-Barré; chronic conditions like chronic inflammatory demyelinating peripheral neuropathy (CIDP)
- Kidney disease
- Cancer: can trigger global immune responses; radiation treatment can also lead to neuropathic conditions
- Infections (viruses/bacteria): HIV, Lyme disease, diphtheria, leprosy, or West Nile virus
- Medication toxicity: chemotherapy (30%–40% of all patient receiving chemotherapy develop neuropathy); medications used to fight infections, heart and blood pressure abnormalities, and anticonvulsants
- Environmental toxins: lead, mercury, arsenic, and insecticides
- Heavy alcohol consumption
- Inherited peripheral neuropathies: Charcot-Marie-Tooth, Dejerine-Sottas, and Friedrich’s ataxia

23. How does a sciatic nerve injury present?
The most common presentation of a sciatic nerve injury is footdrop. It can often be confused with L5 radiculopathy and fibular nerve injury. In severe sciatic nerve injuries, patients will also exhibit (in addition to weak ankle dorsiflexion) weak ankle plantar flexion and knee flexion, decreased ankle jerk reflex, and sensory loss of the lateral leg and dorsal and plantar aspects of the foot.
24. What are the most common causes of sciatic nerve injury?
The most common causes of sciatic nerve injury are hip trauma and surgery. Sciatic nerve injury can occur up to 3% of the time following total hip replacement. The next most common injuries are external compression and penetrating injuries (i.e., gunshot, knife, injections). Least common are tumors in adults; however, in the pediatric population a tumor is the most common cause of sciatic neuropathy.

25. What are common nerve conduction and electromyography findings in patients with sciatic nerve injury?
NCS findings:
The sural and superficial fibular sensory nerves will have reduced amplitudes or, in severe injuries, absent responses. Because sciatic nerve injuries most often involve the fibular portion versus the tibial portion of the sciatic nerve, the fibular motor nerve amplitude will be significantly reduced, and the tibial nerve and tibial H-reflex will demonstrate normal or near normal values. Fibular F-waves will be abnormal.

EMG findings:
Sampling of the lumbar paraspinals (lumbar dorsal rami), gluteus maximus (inferior gluteal nerve), tensor fascia latae (superior gluteal nerve), quadriceps (femoral nerve) will test normal because they are not innervated by the sciatic nerve. Muscles showing denervation will primarily be fibular innervated muscles (94%–100% of patients), such as the biceps femoris short head and pre Tibial and lateral lower leg compartment muscles. Tibial innervated muscles (medial hamstring muscles, posterior and medial leg compartment muscles) can and will be involved in severe sciatic nerve injuries (74%–84% of patients).

26. What is the prognosis for patients who have sustained a sciatic nerve injury?
The prognosis is dependent on the severity of the injury (not location). However, most individuals have a good outcome 3 years following injury, whereas 30% of individuals sustaining sciatic nerve injury have near normal function 1 year post injury. The best outcomes occur in those patients with a common fibular nerve conduction response that is obtainable from the extensor digitorum brevis muscle and the absence of paralysis of the pretibial and/or posterior compartment muscles.

BIBLIOGRAPHY
CHAPTER 74 QUESTIONS

1. In a patient with footdrop (ie, weakness of ankle dorsiflexors and toe extensors), the clinical and electrophysiologic evidence distinguishing between a common fibular nerve (peroneal nerve) mononeuropathy and an L5 radiculopathy is:
   a. Weakness and denervation in the tibialis anterior, EDL, and EHL
   b. Weakness and denervation in the EDB and EHB
   c. Weakness and denervation in the tensor fascia lata and tibialis anterior
   d. Loss of sensation over the dorsum of the foot

2. A patient presents with numbness and tingling on the dorsal aspect of his foot between the first and second toe and 2/5 MMT of the extensor digitorum brevis muscle. No other weakness or loss of sensation is noted in the involved lower limb. What is the MOST likely lower limb nerve entrapment?
   a. L5 radiculopathy
   b. Fibular nerve injury at the fibular head
   c. Deep fibular nerve injury at the ankle
   d. Tarsal tunnel syndrome

3. Following a routine arthroscopic knee surgery, a patient reports loss of sensation along the medial aspect of the lower limb. There is no motor weakness of the involved lower limb and reflexes are intact. What is the most likely nerve injury?
   a. Femoral nerve
   b. Lateral cutaneous nerve of the thigh
   c. Obturator nerve
   d. Saphenous nerve
1. What are the major anatomic divisions of the bones of the foot?

The rear foot (tarsus) consists of the talus and calcaneus. The midfoot (lesser tarsus) consists of the navicular (or scaphoid), cuboid, and 3 cuneiforms (medial, intermediate, and lateral). Distal to the midfoot are the metatarsals and phalanges. The foot also may be divided into medial and lateral columns. The medial column is composed of the talus, navicular, 3 cuneiforms, and metatarsals 1 to 3, along with their respective phalanges. The lateral column consists of the calcaneus, cuboid, and metatarsals 4 to 5 along with their respective phalanges.

2. What are the four muscular layers, from superficial to deep, from the plantar aspect of the foot?

**FIRST LAYER (3 MUSCLES)**
- Abductor hallucis
- Flexor digitorum brevis
- Abductor digiti minimi

**SECOND LAYER (2 MUSCLES)**
- Quadratus plantae
- Lumbricals

Note anatomic location: the tendons of the flexor hallucis longus and the flexor digitorum longus, which are considered extrinsic foot muscles located in the leg, pass through this layer.

**THIRD LAYER (3 MUSCLES)**
- Flexor hallucis brevis
- Adductor hallucis
- Flexor digiti minimi brevis

**FOURTH LAYER (2 MUSCLES)**
- Plantar interossei (3 muscles; PAD = Plantar ADduct)
- Dorsal interossei (4 muscles; DAB = Dorsal ABduct)

Note anatomic location: the tendons of the peroneus longus and tibialis posterior, which are considered extrinsic foot muscles located in the leg, pass through this layer.

3. Describe the axis of movement and range of motion (ROM) of the foot and ankle.

<table>
<thead>
<tr>
<th>Talocrural</th>
<th>Subtalar</th>
<th>Midtarsal</th>
<th>Tarsometatarsal</th>
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<tbody>
<tr>
<td><strong>ROM</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PF: 0–50 degrees</td>
<td>IN: 0–35 degrees</td>
<td>IN: 0–20 degrees</td>
<td>PF: 0–15 degrees</td>
</tr>
<tr>
<td>DF: 0–20 degrees</td>
<td>EV: 0–15 degrees</td>
<td>EV: 0–10 degrees</td>
<td>DF: 0–3 degrees</td>
</tr>
<tr>
<td><strong>Axis</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>80 degrees from vertical reference (10 degrees, up from horizontal), 84 degrees from longitudinal reference of foot</td>
<td>40–45 degrees superior from horizontal reference, 15–18 degrees medially from longitudinal reference (sagittal plane)</td>
<td>15 degrees superior from horizontal reference, 9 degrees to midline for IN/EV</td>
<td>Similar to subtalar joint</td>
</tr>
</tbody>
</table>

PF, Plantar flexion; DF, dorsiflexion; IN, inversion; EV, eversion; ROM, range of motion.
4. **How much talocrural ROM is typically required for normal gait?**

   Although compensations may occur in the lower limb, pelvis, or lumbar spine to accommodate for a restricted talocrural joint, approximately 6 to 10 degrees of dorsiflexion and 20 to 30 degrees of plantar flexion are required for normal gait.

5. **How much subtalar ROM is required for normal gait?**

   Although compensations may occur in the lower limb, pelvis, or lumbar spine to accommodate for a restricted subtalar joint, a total of 4 to 6 degrees of inversion/eversion is generally required for normal gait.

6. **What is the correct terminology to use when referring to or describing foot and ankle motion?**

   It is correct to use the suffix “-us” or “-ed” when describing or referring to a static position (eg, supinatus or pronated) and “-ion” and “-ing” when describing or referring to motion or a movement (eg, supination or pronating).

7. **Define pronation and supination in relation to the rear foot.**

   Pronation and supination are the triplane motions in the subtalar joint, the so-called universal joint of the lower extremity. In weight bearing, pronation occurs at initial contact through the loading response during gait. Internal rotation of the lower leg produces talar adduction and plantar flexion relative to the calcaneus, and the calcaneus everts and abducts. This process typically occurs during the first 25% of the stance phase of gait, as the foot adapts to the ground.

   Supination during gait occurs from the start of the midstance phase of gait (foot flat) through terminal stance. This process occurs as the lower leg starts to rotate externally, leading to talar abduction (dorsiflexion relative to the calcaneus), and the calcaneus inverts and adducts. In the nonweight bearing or swing phase, the talus is relatively fixed in the ankle mortise, and supination/pronation occurs through the subtalar joint by movement of the calcaneus and foot around the subtalar joint axis of motion. In supination, the calcaneus and foot move through a combination of inversion, adduction, and plantar flexion in relation to the fixed talus. In pronation, the calcaneus moves through eversion, abduction, and dorsiflexion relative to the fixed talus.

8. **Explain the windlass mechanism of the foot.**

   The windlass mechanism of the foot refers to the seemingly simple maneuver of dorsiflexion of the toes of the foot, most specifically related to passive hallux extension that elevates the medial longitudinal arch through hindfoot supination when the calcaneus inverts. The plantar fascia and intrinsic foot musculature are supinators around the subtalar joint axis of motion. Hence, dorsiflexion of the digits produce supination, which creates the medial longitudinal arch of the foot through midtarsal joint motion.

9. **What are the common arches of the normal foot?**

   There are four primary arches of the normal foot supported by myoligamentous structures. The two longitudinal arches are the medial longitudinal arch (MLA) and the lateral longitudinal arch (LLA). The calcaneus is common to both longitudinal arches. The MLA is formed by the medial columnar structures of the calcaneus, talus, navicular, 3 cuneiforms, and metatarsals 1, 2, and 3. The MLA is primarily involved in weight bearing. The LLA is formed by the calcaneus, cuboid, and metatarsals 4 and 5. There are two transverse arches: the proximal transverse arch, formed by the bony structures of the navicular, 3 cuneiforms, and cuboid, and the distal transverse arch, formed by the heads of the 5 metatarsals.

10. **What are the main noncontractile or passive supports of the longitudinal arches?**

    The noncontractile passive supports of the longitudinal arches are the 1) plantar aponeurosis, 2) long plantar ligament, 3) short plantar ligament, and 4) plantar calcaneonavicular ligament, also commonly known as the “spring” ligament.

11. **What are the main dynamic support structures of the longitudinal arches of the foot?**

    The dynamic support structures of the foot include the intrinsic muscles (4 layers) and the muscles and tendons of the fibularis longus, posterior tibialis, flexor hallucis longus, and flexor digitorum.

12. **What is pes planus?**

    Pes planus (flatfoot) describes a foot that exhibits no longitudinal arch and an ankle that is everted (valgus). It can be classified as rigid or flexible. A rigid flatfoot is often associated with a tarsal coalition or a vertical talus, and a flexible flatfoot is considered a normal variant. Pes planus is normal in children.
up to 6 to 7 years of age. A rigid flatfoot is always flat but a flexible flatfoot appears normal when nonweight bearing but becomes flat when standing. If a flexible flatfoot is asymptomatic, no treatment is warranted, but if it is symptomatic, then arch supports are often incorporated. If the deformity is rigid, then the underlying cause (eg, tarsal coalition, vertical talus) must be addressed.

13. What is pes cavus?
Pes cavus refers to a high arch foot. This can be a benign condition that merely describes a foot type exhibiting an abnormally high arch or can be related to muscle imbalances in the immature foot, although it is important to rule out the possibility of underlying neuromuscular disease (such as Charcot-Marie-Tooth disease). The presentation is typically an 8- to 10-year-old child who complains of ankle pain, habitually toe-walks, and exhibits tight Achilles tendon and limited ankle dorsiflexion. A clinical workup may be needed that includes radiographs, EMG/NCS, and MRI of the spine to rule out occult neuromuscular disease. Treatment may include bracing/ankle-foot orthoses (AFOs) or osteotomies and tendon transfers in more severe cases.

14. What is the ideal position for ankle fusion (eg, arthrodesis)?
The ideal position for ankle arthrodesis is neutral dorsiflexion (or slight plantar flexion if heeled shoes are preferred, eg, by women), slight valgus (0–5 degrees), and external rotation of approximately 5–10 degrees.

15. What percentage of weight does the fibula bear?
The fibula supports approximately 12% to 17% of the axial load.

16. What is Fick’s angle?
Normally when an individual stands, the posture of the foot assumes a slight toe-out position, and this angle, approximately 12–18 degrees in the adult (5 degrees in children), is sometimes referred to as Fick’s angle.

17. Describe the function of the deltoid ligament.
The deltoid or medial collateral ligament of the rear foot consists of a superficial and a deep ligament complex. The superficial deltoid ligament consists of the ligament attachment to the distal tibia (medial malleolus) with insertions onto the navicular, sustentaculum tali, and talus. The majority of these ligament fibers are vertically oriented and therefore prevent or limit excessive rear-foot eversion in the frontal plane. The deep deltoid ligament consists of relatively transversely oriented fibers deep to the superficial band from the medial malleolus anteriorly and posteriorly along the medial body of the talus. Thus it resists excessive transverse plane rotation (abduction) of the talus. The deltoid ligament may be sprained under excessive loading of the ankle and rear foot in eversion or may avulse a portion of the medial malleolus as part of an ankle fracture (four components: tibionavicular, tibiocalcaneal, anterior tibiotalar, and posterior tibiotalar).

18. What are the lateral collateral ligaments of the ankle and rear foot?
The lateral collateral complex of rear-foot and ankle ligaments consists of the anterior and posterior talofibular ligaments and the calcaneofibular ligament. The anterior talofibular ligament and calcaneofibular ligaments are most commonly sprained in inversion ankle injuries. The horizontally oriented anterior talofibular ligament and the more vertically oriented calcaneofibular ligaments provide reciprocal stability to the rear foot. In a plantar-flexed position of the ankle, the anterior talofibular ligament (flat, fan-shaped capsular ligament) is the primary stabilizer to rear-foot inversion. In a dorsiflexed position, the cordlike calcaneofibular ligament is the stabilizer to rear-foot inversion.

The Lisfranc ligament is the plantar tarsometatarsal ligament, spanning the medial cuneiform to the base of the second metatarsal. In fractures and dislocations of the Lisfranc joint, this ligament commonly avulses a fragment of bone from the plantar medial base of the second metatarsal.

20. What is the spring ligament?
The spring ligament is another name for the calcaneonavicular ligament, which extends from the plantar aspect of the sustentaculum tali (on the calcaneus) to the navicular. It provides support to the plantar head of the talus and talonavicular joint and is a primary static stabilizer, reinforcing the medial longitudinal arch.
21. What is the bifurcate ligament?
The bifurcate ligament is Y-shaped and originates from the anterior floor of the sinus tarsi and anterior process of the calcaneus. It extends and divides distally into two distinct bands that attach to the cuboid laterally and navicular medially. This ligament provides important stability to the rear foot.

22. Define Chopart and Lisfranc joints.
The Chopart joint is the midtarsal joint, which consists of the talonavicular and calcaneocuboid joints. The Lisfranc joint is the tarsometatarsal joint, which consists of the 3 cuneiforms and metatarsals 4 and 5.

23. How does the weight bearing surface of the ankle change after syndesmotic injury of the ankle?
Mortise widening, resulting in a 1-mm lateral shift of the talus, decreases the weight bearing surface of the talus by 40%, a 3-mm shift by >60%, and a 5-mm shift by approximately 80%. Increased contact pressures may lead to early degenerative joint disease.

24. Why is the anterior talus subject to impingement?
The anterior portion of the talus is 2.5 mm wider than the posterior talus. With dorsiflexion, the space available in the anterior mortise is decreased. This space can be further compromised by osteophytes, scar tissue, or overly compressed open reduction and internal fixation (ORIF) to the syndesmosis after ankle fracture. The compression/distraction of the ankle joint (talocrural joint) that occurs with normal walking may be important for normal lubrication of the joint.

25. What is the sinus tarsi?
The sinus tarsi is a funnel-shaped opening in the rear foot between the talus and calcaneus. It is widest anterolaterally and narrows as it passes posteromedially between the talus and calcaneus, separating the anterior and middle facets of the subtalar joint from the posterior facet. The narrow posteromedial section of this space often is called the tarsal canal. Through this area pass the interosseous talocalcaneal ligament and the major blood supply to the body of the talus (the anastomosis between the artery of the tarsal canal and the artery of the tarsal sinus).

26. What are the contents of the tarsal tunnel?
From superficial to deep and anterior to posterior, the contents of the tarsal tunnel can be remembered by the mnemonic “Tom, Dick, And Very Nervous Harry”:

- **Tom** = posterior Tibial tendon
- **Dick** = flexor Digitorum longus
- **And Very Nervous** = posterior tibial Artery, Vein, and Nerve
- **Harry** = flexor Hallucis longus

27. Describe the structure of the tarsal tunnel.
The tarsal tunnel is bounded by the distal tibia (medial malleolus) anteriorly and the Achilles tendon posteriorly; it is roofed by the flexor retinaculum (laciniate ligament). The flexor retinaculum divides into fibrous (septae) bands that separate the contents of the tarsal tunnel into individual compartments.

28. List the five nerves that cross into and supply the motor and sensory fibers to the foot.
1. Sural nerve—sensory (posterolaterally)
2. Superficial peroneal nerve—motor and sensory (anterolaterally)
3. Deep peroneal nerve—motor and sensory (anteriorly, traveling with the dorsalis pedis artery)
4. Saphenous nerve—sensory (anteromedially, as the long continuation of the femoral nerve distally)
5. Posterior tibial nerve—motor and sensory (posteromedially, dividing to supply the foot distally as the medial and lateral plantar nerves)

29. Define porta pedis.
The porta pedis is the anatomic opening into the plantar aspect of the foot beneath the belly of the abductor hallucis muscle. Through this opening pass the medial and lateral plantar nerves and arteries/veins distally from the tarsal tunnel into the foot. The porta pedis is a potential site for compression of the plantar nerves and may also be a cause of heel pain.

30. What structure is referred to as “freshman’s nerve”?
The plantaris muscle tendon, which often appears like a nerve to new dissectors of the human cadaver, is referred to as “freshman’s nerve.” However, its location, flat, firm appearance, and
consistency reveal that it is a tendon. It travels deep to the gastrocnemius and superficially to the soleus to lie medially to the Achilles tendon, where it attaches onto the medial aspect of the posterior calcaneal tuberosity.

31. What is meant by an accessory bone of the foot?
An accessory bone is a small ossicle or extra bone that separates from the normal bone (most commonly caused by fracture or a secondary ossification center). Accessory bones are more frequently found in the foot than anywhere else in the body. The most common are the os trigonum (from the posterior talus), the os tibiale externum (from the navicular tuberosity), the bipartite medial cuneiform (superior/inferior), the os vesalianum pedis (tuberosity of the base of the fifth metatarsal), the os sustentaculum (sustentaculum tali), and the os supranaviculare (dorsum of talonavicular joint).

32. Describe the function of the sesamoids.
The sesamoids are located beneath the head of the first metatarsal. The two functions of the sesamoids are 1) to transfer loads through the soft tissues to the metatarsal head and 2) to increase the lever arm of the flexor hallucis brevis to aid in push-off.

33. What is the master knot of Henry?
The master knot of Henry is a fibrous band on the plantar aspect of the foot adjoining the flexor digitorum longus and flexor hallucis longus tendons in the second layer of the intrinsic foot muscles.

34. What is the effect of an increasing hallux valgus on plantar flexion force at push-off?
A hallux valgus angle of 40 degrees decreases push-off strength of the great toe by 78%. Adding a 30-degree pronation deformity decreases the plantar flexion strength to 5% of normal.

35. What is Toygar’s triangle?
On the lateral radiograph of the foot and ankle, Toygar’s triangle is the hypodense radiographic triangle bordered by the more radiodense Achilles tendon posteriorly, the superior border of the calcaneus at its base, and the posterior border of the mid to distal tibia. When the triangle is not apparent on the lateral radiograph, the usual cause is accumulation of fluid along the tarsal tunnel, which may suggest inflammation from ankle, subtalar joint, or retrocalcaneal bursitis. The triangle may be obliterated completely by hematoma or swelling around an Achilles tendon rupture.

36. What are the normal forces (relative to body weight) acting on the ankle joint during functional activities, such as walking, running and jumping?
Compressive forces during normal walking are 1 to 1.2 times body weight, running 2 times body weight, and jumping (from a height of about 24 inches) 4 to 5 times body weight.

37. How many muscles attach to the talus?
None. One of the unique features of the talus is that there are no musculotendinous attachments to it.

38. What is metatarsus adductus?
This refers to one of the most common pediatric foot disorders and describes the position of the forefoot in varus and adduction. It is often associated with intrauterine position and clinically presents with a “kidney-bean” appearance, depicting the nature of the deformity and an in-toeing gait. Most will resolve with normal development, minor shoe modifications, or serial casting. Rarely is surgical intervention (eg, midfoot osteotomy) required.

39. What is the function of the interossei and lumbral muscles?
The dorsal interossei (DAB, four muscles) are abductors of the toes, while the plantar interossei (PAD, three muscles) perform adduction of the toes. The combined primary actions of the lumbricals and interossei are plantar flexion of the metatarsophalangeal joints and extension at the proximal interphalangeal and distal interphalangeal joints. These intrinsic muscles provide stability, support, and integrity to the arches of the foot.

40. Describe the functional anatomy of the anterior talofibular ligament.
Bone is less dense at the fibular attachment, but the enthesis fibrocartilage is more prominent. Fibrocartilage is present at the site where the ligament wraps around the lateral talar articular margin in the plantar-flexed and inverted foot, likely as a result of compression in this region. Avulsion fractures are less common at the talar end because the bone in this area is denser and stress is dissipated away from the talar enthesis by the fibrocartilaginous character of the ligament near the talus.
41. What is the relationship between metatarsal length and midfoot arthrosis?

Patients with midfoot arthrosis have a significantly higher ratio of second metatarsal to first metatarsal length compared with controls. Studies have shown the functional length of the second metatarsal was 18.6% greater than the first metatarsal in the arthrosis group, compared with 4.1% for the control group.

**BIBLIOGRAPHY**


**CHAPTER 75 QUESTIONS**

1. Which of the following ankle ligaments of the lateral complex is most likely to be injured with an inversion mechanism with the foot in a plantar-flexed position?
   a. Anterior talofibular ligament
   b. Posterior talofibular ligament
   c. Calcaneofibular ligament
   d. Anterior inferior tibiofibular ligament

2. Which of the following ligaments supports both the medial longitudinal arch and the plantar aspect of the talonavicular joint?
   a. Long plantar ligament
   b. Short plantar ligament
   c. Bifurcate ligament
   d. Calcaneonavicular ligament

3. A rigid flatfoot is often an indication of which of the following underlying pathologies?
   a. Tarsal coalition
   b. Lisfranc sprain
   c. Charcot-Marie-Tooth
   d. Os trigonum
1. What is the difference between Achilles tendinitis, tendinosis, and tendinopathy?
   Terminology used to describe disorders of the Achilles tendon is very confusing. Although “itis” suggests inflammation, inflammatory cells are often absent and degeneration of the tendon (tendinosis) is present. True tendonitis is an inflammatory process of the tendon. This can be caused by overuse activities or be related to a specific disease process, such as rheumatic diseases. The signs of inflammation are pain on palpation, swelling, warmth, and pain on active contraction of the muscle-tendon complex. Passive stretching may also induce pain. The clinical practice guidelines of the American Physical Therapy Association (APTA) have established that tendonitis or tendinosis is misleading and should be replaced with “tendinopathy,” unless histologic evidence has proven otherwise.

   In Achilles tendinopathy the patient may report mild pain and stiffness with his or her desired level of activity, and it may progress to limiting activity considerably. There may be an appearance of thickening of the tendon, but this is not swelling related to the inflammation. Most patients will report a failure of NSAIDs to provide relief. MRI findings may show signal changes inside the tendon. In addition, high-resolution ultrasound identifies this pathology more clearly. The tendon may have hyperechoic areas, indicating disorganization of the collagen fibers. There have been multiple studies confirming the phenomenon of neovascularization—the attempt of the tendon to heal by bringing blood vessels to the damaged areas. It is speculated that nerve fibers, which accompany these new blood vessels, are the source of long-term pain experienced in these patients.

2. What is the difference between midsubstance tendinopathy and insertional tendinopathy, and what are the treatment implications?
   There is a clear distinction between proximal tendinopathy, also called midsubstance or Zone 1, and distal insertional Achilles tendinopathy, and the treatment will be different. Midsubstance tendinopathy occurs 4 to 6 cm proximal to the Achilles insertion, and the Alfredson eccentric protocol has the strongest evidence for efficacy. Distal tendinopathy or insertional tendinopathy is located where the tendon inserts into the calcaneous. Often, calcification is found in the tendon. Treatment for insertional tendinopathy has included modification of shoe wear, heel lifts, orthosis, antiinflammatory medication, rest, night splints, gastroc and soleus stretches, and iontophoresis and ice. A recent randomized controlled study confirmed that the addition of eccentric training for patients with insertional pain did not lead to further improvements.

   Haglund’s “triad,” which occurs at the site of the calcaneous and includes Haglund’s deformity (pump bump) retrocalcaneal bursitis and insertional Achilles tendinopathy, also should be considered.

3. What are the risk factors for developing Achilles tendinopathy?
   Intrinsic risk factors associated with Achilles tendinopathy include abnormal ankle dorsiflexion range of motion, abnormal subtalar joint range of motion, decreased ankle plantar flexion strength, increased foot pronation, and abnormal tendon structure. Obesity, hypertension, hyperlipidemia, and diabetes are medical conditions associated with Achilles tendinopathy. Clinicians also should consider training errors, environmental factors, and faulty equipment as extrinsic risk factors associated with Achilles tendinopathy.

4. What should be considered when evaluating Achilles tendinopathy?
   Palpation of the length of the tendon from origin to insertion and surrounding areas for tenderness and pain is most important. Dorsiflexion range of motion, subtalar joint range of motion, plantar flexion strength and endurance, static arch height, and forefoot alignment should be assessed. It is also the responsibility of the physical therapist to assess the function of the entire lower extremity to identify whether there are other impairment findings in the lower extremity (hip/knee weakness; flexibility issues) that may have contributed to the cause of the tendon dysfunction.
5. What is the best treatment for midsubstance Achilles tendinopathy?
The initial intervention is rest and adequate unloading of the tendon. The patient must understand that tendons “heal” very slowly, and this process will take months to accomplish. Frequently, rigid heel lifts of ½ inch either in shoes or attached to the outer sole and clinically have been observed to reduce symptoms. Consensus of current literature demonstrates the strongest evidence for midsubstance tendinopathy is an eccentric training program. Alfredson initially introduced the eccentric program with studies confirming that with a program of 12 weeks of eccentric loading, patients improved significantly in function and had reduced pain. In addition, Ohberg, using high-definition ultrasound, confirmed improvements in the structure of the tendon after this eccentric type of loading program.

To supplement eccentric strengthening, manual therapy to increase ankle and subtalar range of motion (ROM) is indicated. Taping techniques either to offload the tendon or to limit ankle ROM and/or taping the arch have all been clinically beneficial.

Studies have also shown moderate support for the use of low-level laser therapy to decrease pain and stiffness in patients and iontophoresis with dexamethasone to decrease pain and improve function in patients with Achilles tendinopathy.

6. What is the eccentric protocol for Achilles tendinopathy?
The typical heel drop exercise is done with the patient standing on a block or a step. The patient is instructed to go up as high as he or she can on both feet and then come down slowly on the affected side, only to drop the heel below the step.

This original protocol developed by Dr. Alfredson recommends the completion of 3 sets of 15, twice a day for 12 weeks, with the leg both flexed and extended (a total of 180 daily heel drops). Pain is actually expected with the exercise and should be expected after exercise. Progression of the exercise should be when the exercise is pain free and increased repetitions or a load can be added.

Recently, Stevens and Tan reported that a “6-week, as tolerated” program of eccentric exercise was as effective and less uncomfortable than the Alfredson protocol for midportion Achilles tendinopathy. Patients are asked to perform as many heel drops as they could tolerate. This program may be appropriate for patients who have trouble executing the Alfredson protocol.

7. What is the differential diagnosis for those with heel and posterior lower leg pain?
- Acute Achilles tendon rupture
- Partial tear of the Achilles tendon
- Retrocalcaneal bursitis
- Haglund’s deformity—a protuberance of bone originating on the calcaneus at the tendocalcaneal junction that can cause friction between the Achilles tendon and the shoe
- Posterior ankle impingement by osseous abnormalities including os trigonum syndrome
- Irritation or neuroma of the sural nerve
- Accessory soleus muscle
- Achilles tendon ossification
- Systemic inflammatory disease
- Insertional Achilles tendinopathy
- S1 radiculopathy

8. What are the common rupture sites of the Achilles tendon complex?
The most common site is a complete midsubstance rupture of the Achilles tendon. This area, 2 cm to 6 cm proximal to the insertion site, is most susceptible to injury because it is hypovascular. The second most common site is the musculotendinous interface, followed by the rare avulsion of the tendon from the bone. An incomplete rupture of the Achilles tendon also can evolve from chronic tendinosis.

9. Describe the typical patient with Achilles tendon rupture.
The typical patient is male between 30 and 50 years of age who engages in a physical activity or sport. Predisposing factors include advanced age, weekend athletes, history of tendonitis or tendinosis, and loss of flexibility in the Achilles tendon.

10. How is an Achilles tendon rupture diagnosed?
According to the American Academy of Orthopedic Surgeons, the following tests are recommended to arrive at the diagnosis of Achilles tendon rupture:
- Thompson’s test—The patient is positioned prone with the foot hanging off the table. The examiner squeezes the widest girth of the calf and observes for plantar flexion to occur. If no plantar flexion occurs, the test is positive for rupture.
• Decreased plantar flexion strength—The patient most likely will be unable to perform a heel rise while standing.
• Presence of palpable gap, defect, or lack of contour of the Achilles tendon.
• Increased passive ankle dorsiflexion.

* The evidence surrounding the use of MRI, ultrasound, and radiographs in the diagnosis of Achilles tendon rupture is inconclusive.

11. Is operative treatment necessary for acute Achilles tendon ruptures?
Although surgical repair is more common, it is not necessary and controversy continues with regard to the optimal treatment for acute Achilles tendon ruptures. Treatment can be classified into operative (open or minimally invasive/percutaneous) and nonoperative. Postoperative support can be divided into cast immobilization and functional bracing. Weight-bearing status also varies, depending on the surgeon.

Primary repair seems to be the “gold standard” of care; however, surgically repaired tendons rarely recover functionality similar to the previous state. Most patients with an Achilles tendon rupture seldom achieve full function at 2 years after surgery; moreover, results after the first year are quite variable. Poor results have been attributed to overstetching the tendon or alterations in the cellular organization within the tendon that occur at the time of injury and during early healing stages. In the future, tissue engineering, such as human growth hormones, may lead to improved management of these injuries.

12. What is a recommended nonoperative and operative rehabilitation protocol after Achilles tendon rupture?
There is no clear consensus regarding the optimal rehabilitation protocol for this injury. Traditionally, patients have been placed in a cast with the ankle in plantar flexion for the first 6 to 8 weeks nonweight-bearing. However, more recent literature advocates for accelerated early range of motion exercises and weight-bearing protocol for both operative and nonoperative management. Additionally, outcomes appear very similar for these two groups.

- 0–2 weeks: NWB in plantar-flexion-posterior splint
- 2–4 weeks: CAM boot with 2-inch heel lift, PWB with crutches, and active ROM (plantar and dorsiflexion) to neutral.
- 4–6 weeks: WBAT in CAM boot; continue active ROM to neutral
- 6–8 weeks: remove heel lift; dorsiflexion stretching, hydrotherapy, and graduated resistance exercises
- 8–12 weeks: wean off boot, continue active ROM, strength, proprioception, and prn crutches
- 12 weeks: sport-specific training

This accelerated protocol focuses on early movement of the ankle between neutral and plantar flexion and while in a brace for 6 weeks. Prospective studies and randomized controlled trials have shown that, compared with cast immobilization, the use of early postoperative ROM and weight bearing actually showed significant improvement in health-related quality of life in the early postoperative period, posed no additional risks, and demonstrated a trend toward a reduction in lost work days and an earlier return to sports.

13. What is the goal for functional outcome after Achilles tendon rupture with appropriate rehabilitation?
A systematic review of the literature performed by the American Academy of Orthopedic Surgeons (AAOS) in 2009 found that patients who participate in sports are able to return to jogging as early as 3 to 4 months, with the majority returning at 6 to 8 months.

14. What are the most important considerations with rehabilitation of the Achilles tendon rupture?
The goal is to prevent overlengthening of the Achilles tendon. Protection of the ankle is very important, and heel lifts can be used to reduce stress on the repair while the patient progresses in weight-bearing status. Early overstetching the tendon with calf stretches can be detrimental to the length-tension relationship and have permanent consequences on recovery. We advocate no stretching of the Achilles tendon during the first 4 months after repair.

15. Describe symptoms for tarsal tunnel syndrome.
Pain in the area of the tarsal tunnel or into the foot is the most commonly reported symptom. Some patients also may complain of paresthesia in the foot.
16. What factors may contribute to tarsal tunnel syndrome?

Mechanical factors (such as foot pronation) may cause compression of the tibial nerve and its branches in this location. Trauma to the lower leg—from fracture, sprain, or other soft tissue injury—may also lead to increased swelling. The abnormal swelling of the lower leg may cause compression on the nerve in this closed space. In addition, more proximal pathology may be associated with tarsal tunnel syndrome. A thorough review of systems is essential in identification of tarsal tunnel syndrome. Rheumatic disease may also cause swelling around the nerve or a peripheral neuropathy can present with similar symptoms; both should be ruled out. For example, patients with other nerve lesions, such as a lumbar spine pathology, may also have concomitant symptoms in the area of the tarsal tunnel. In closer proximity to the tarsal tunnel is the soleus hiatus, where the tibial nerve can be compressed because it is surrounded by a fibromuscular tunnel.

17. What is posterior tibialis tendon dysfunction (PTTD)?

PTTD is a progressive degeneration of the posterior tibial tendon and is the most common cause of painful and debilitating acquired flatfoot deformity in adults. The dysfunction is often progressive and may result in collapse of the plantar arch associated with tendon rupture. Many practitioners have currently labeled this sequela as adult acquired flatfoot disorders (AAF) because not only is the posterior tendon involved, but many of the structures of the foot are affected. Before rupture, the tendon undergoes attenuation and degeneration, resulting in frequent episodes of debilitating pain.

18. What are the different stages of PTTD?

Johnson and Strom originally described the progressive clinical stages of posterior tibial tendon dysfunction. In stage I the patient has mild swelling and medial ankle pain but no deformity. There is mild weakness and the length of the tendon is normal; however, degeneration is present. A patient may be able to perform a single heel rise, but this movement is painful. Stage II is progressive flattening of the arch, with an abducted midfoot. In this stage the tendon is ruptured or functionally incompetent. The foot is still flexible and correctable, but the patient is unable to perform a single heel rise because the tendon is functionally incompetent. In stage III all of the signs of stage II occur; however, the hindfoot deformity becomes fixed. Myerson added stage IV for those patients who progressed to valgus tilt of the talus in the ankle mortise, leading to lateral tibiotalar degeneration. Stages I and II are most amenable to conservative treatment, while III and IV often require surgery.

19. What causes PTTD?

Patients with excessive pronation are likely predisposed to the condition because pronation changes the alignment of the foot and, over time, can lead to adult acquired flatfoot deformity. When the foot is excessively pronated, the balance between the tibialis posterior tendon and the fibularis longus is lost. The pronated foot then changes the alignment of the gastroc/soleus tendon relative to the axis of rotation, and then, instead of being an invertor of the foot, the Achilles tendon is lateral to the axis and pulls the foot into further eversion. Overload of the posterior tibial tendon then occurs, causing the other supporting structures of the foot, such as the spring ligament, and plantar aponeurosis to take over and the deformity progresses. Another predisposition is a critical area of hypovascularity in the tendon posterior and distal to the medial malleolus. In addition, medical factors linked with PTTD include hypertension, obesity, diabetes, steroid exposure, and inflammatory arthritides.

20. What should be considered when evaluating PTTD?

- Observation—arch height and foot posture including the “too many toes sign” (as seen from behind, the toes are seen laterally indicating abduction of the forefront)
- Location of symptoms—medial ankle pain and swelling behind the medial malleolus is the hallmark. However, in very later stages, patients may have pain laterally because of impingement of the fibula or lateral talar process by the anterior process of the calcaneus.
- Palpation—tenderness in the same area as well as more proximally in the muscle and possible pain in other arch-supporting structures of the foot, such as the plantar fascia, because of overload of these tissues
- Ability to perform a pain-free heel rise—the calcaneus should be in an inverted position and full range of motion. Make sure to compare to the uninvolved side first. Observe for common compensations of knee flexion or pushing with the upper extremities. Repeated heel rise for endurance should also be tested.
- Manual muscle test of the tibialis posterior in plantarflexion with inversion
- ROM and mobility of the ankle, subtalar, and first MTP as well as lower extremity strength and endurance, especially the gluteal muscles
21. What is the best evidence-based treatment for PTTD?
Initially, immobilization and rest of the tendon are necessary to prevent excessive pronation and to decrease demand on the posterior tibialis. Techniques include taping to support the arch, custom-made foot orthotics, a custom-made ankle-foot orthosis, or even complete immobilization with a cast or walking boot. The Richie Brace®, an AFO with a custom-contoured footplate, is a lightweight, low-profile design that is suited to control the abnormal pronation forces and is recommended for support and comfort. Calf stretching with both the knee straight and flexed should be implemented. After immobilization, progressive strengthening in the pain-free range of the posterior tibialis as well as strengthening of the foot intrinsics is beneficial. For patients with stage I and II PTTD, strengthening both eccentrically and concentrically have proven to be effective without harmful effects. Kulig and colleagues have clearly demonstrated that the best exercise to selectively and effectively train the tibialis posterior is resisted foot adduction with the foot in contact with the floor, in a windshield-wiper type of motion. The use of an arch support or orthoses during this exercise will recruit the tibialis posterior more effectively. In addition, studies have confirmed that woman with PTTD have impaired ability to perform single-leg stance and diminished hip strength and endurance bilaterally, so strengthening weak proximal muscles would be indicated.

22. What causes peroneal tendon subluxation?
Both the longus and brevis tendons are at risk for subluxation or dislocation from the fibular retromalleolar sulcus. The most frequent cause is a skiing injury, but subluxation has been reported in several other sports (e.g., soccer, football, basketball, tennis, and gymnastics). The most commonly described mechanism is sudden, forceful passive dorsiflexion of the everted foot with sudden, strong reflex contraction of the peroneal muscles. The injury also has been described with forced inversion, which also causes sudden contraction of the peroneals.

23. How is peroneal tendon subluxation diagnosed?
An acute subluxating peroneal tendon frequently is misdiagnosed as an ankle sprain. The patient usually describes a traumatic injury with lateral swelling and ecchymosis, which often are associated with popping or snapping sounds. Often patients with a subacute condition also have sprained the lateral collateral ligaments. Most patients complain of pain behind the fibula and above the joint line, which differentiates it from the pain of a lateral ankle sprain. The patient’s presenting symptoms usually include swelling and tenderness posterior to the lateral malleolus. Provocative tests should be done but may not be helpful in the acute setting. Dislocation of the peroneal tendon is evident during a stress test of inversion. Testing is done by resisting eversion with a dorsiflexed ankle.

24. Summarize the differential diagnosis for heel pain.

**POSTERIOR HEEL PAIN**
- Retrocalcaneal bursitis
- Haglund’s deformity (“pump bump”)
- Hagland’s triad
- Achilles tendonitis or tendinosis
- Calcification within the Achilles tendon
- Referred pain from a soleus muscle trigger point
- Radiculopathy of S1

**PLANTAR HEEL PAIN**
- Inflammation or microtrauma of the plantar fascia
- Entrapment neuropathy of the tibial nerve or branches
- Fat pad atrophy
- Heel spur
- Stress fracture
- Tarsal tunnel syndrome
- Systemic problems (Reiter syndrome, rheumatoid arthritis, and gout; more common bilaterally)
- Radiculopathy of S1

25. What is plantar fasciitis?
Although the term “fasciitis” denotes inflammation, like Achilles tendon problems, cells associated with inflammation are typically not present. Plantar fasciitis is one of the most common foot-related disorders seen in the outpatient setting. The most common location of pain is at the origin of the plantar fascia at the medial plantar tubercle of the calcaneus. Pain at the midportion of the plantar fascia can occur but is less common.
26. Besides the plantar fascia, what other structures can be involved with this syndrome? Pain may arise from one or more of the following structures: subcalcaneal bursa, fat pad, tendinous insertion of the intrinsic muscles, long plantar ligament, medial calcaneal branch of the tibial nerve, or nerve to abductor digiti minimi. True plantar fasciitis is characterized by progressive pain with weight bearing as well as pain with the first few steps upon rising from a sitting position.

27. What tests and measures are useful in the diagnosis of plantar fasciitis? According to the guidelines published by the orthopedic section of the APTA, the following test and measures are recommended to diagnose plantar fasciitis:

- Palpation of proximal plantar fascia insertion
- Active and passive talocrural dorsiflexion
- Tarsal tunnel test
- Windlass mechanism
- Longitudinal arch angle
- Foot posture index

28. What are the risk factors associated with plantar fasciitis? According to the guidelines published by the orthopedic section of the APTA in 2008, the multifactorial risks include limited ankle dorsiflexion and a high body mass index in nonathletic populations. Running and work-related weight-bearing activities that occur under conditions of poor shock absorption are also risk factors.

29. What is the best treatment for plantar heel pain? According to the American Physical Therapy Clinical Practice Guidelines for Heel Pain of 2014, the following clinical practices are recommended based on the strength of the evidence:

- Manual therapy—talocrural joint posterior glides, subtalar joint lateral glide, anterior and posterior glides of the tarsometatarsal joint distraction manipulation; Level A evidence
- Night splints—use of a night splint for 1 to 3 months; Level A evidence
- Foot orthosis—orthosis with medial longitudinal arch and heel cushion; Level A evidence
- Taping—antipronation taping of the foot and kinesio taping of the gastroc/soleus and plantar fascia; Level A evidence
- Stretching—plantar fascia specific stretching and gastroc/soleus stretching; Level A evidence
- * Modalities such as ultrasound, phonophoresis, low-level laser, and iontophoresis have limited evidence to support their use; Level C evidence.

30. What nerves are involved with plantar heel pain? There is a clear distinction between entrapment of the medial calcaneal nerve and the first branch of the lateral plantar nerve (ie, the nerve to the abductor digiti quini brevis). The medial calcaneal nerve innervates the skin under the heel and may innervate the subcalcaneal bursa. In most cases, this nerve plays no role in plantar heel pain. More likely, the heel pain is from irritation of the first branch of the lateral plantar nerve (known as Baxter’s nerve) or the inferior calcaneal nerve. It innervates the plantar fascia at its origin on the calcaneus, and it also innervates the periosteum of the calcaneus. It often becomes entrapped during chronic plantar fasciitis. Patients will deny first-step pain but, on the contrary, they will complain of symptoms worsening with prolonged activity. They may complain of laterally radiating pain or paresthesia and may be unable to abduct the fifth digit. Traditional treatment for plantar heel pain, as described, would be helpful as well as neural mobilization.

31. How can adverse neurodynamics cause plantar heel pain, and why do patients feel better with neural mobilization? Chronic irritation may cause reduced microcirculation, decreased axonal transport, and altered mechanics, resulting in a painful cycle. In addition, the nerve is a continuum with multiple sites of potential compression that may result in a double-crush phenomenon, exacerbating the pain. It is hypothesized that sliding between the neural tissue and interface tissue can decrease adhesions and promote healing. Neural tissue can shorten and lengthen and has considerable remodeling capabilities. Restoring normal neural mobility appears to be important in abolishing symptoms.

32. Describe the common cause and usual management of heel pain in children. Calcaneal apophysitis of the os calcis (Sever’s disease) is related to activity. The child usually complains of pain with running or jumping as well as tenderness over the insertion of the Achilles tendon. The patient should be referred to a physician. Radiographs are useful for diagnosis when
pain has been prolonged and recalcitrant. Treatment should include decreased activity guided by the child’s symptoms, foot taping, or, in severe cases, immobilization with a brace. A heel lift or improved shoe wear also helps reduce the traction pull on the tendinous apophyseal attachment. The key is to restore heel cord flexibility.

33. What are some clinically useful outcome measures that can be used for patients with heel pain or plantar fasciitis?
- Foot and Ankle Measure (FAAM)
- Foot Health Status Questionnaire (FHSQ)
- Foot Function Index (FFI)

34. Summarize the differential diagnosis for pain in the lateral aspect of the ankle after inversion sprain.
- Osteochondral fracture of the talus
- Distal fibula fracture
- Avulsion fracture of the fifth metatarsal
- Jones fracture (metaphyseal-diaphyseal junction of the fifth metatarsal)
- Peroneal tendon injury
- High ankle sprain of the anteroinferior tibial fibular ligament
- Peroneal or sural nerve irritation
- Cuboid subluxation
- Achilles tendon injury
- Subtalar joint ligament injury

35. How common are the various ankle sprains?
Brostrom reported that 65% of ankle sprains involved complete rupture of the ATFL, and 20% had combined injury to the ATFL and CFL. Isolated injury to the posterior talofibular ligament (PTFL) was rare; isolated injury to the CFL was not found. The anteroinferior tibiofibular ligament (high ankle sprain) was injured in 10% of patients and the deltoid in only 3%. In grade III sprains, the anterior deltoid ligament may be involved through the plantar flexion component of the injury. In summary, the order of ankle ligament injuries occurs in this order (most common to least common): ATFL, ATFL and CFL, PTFL, and CFL.

36. When are radiographs warranted for ankle and foot injuries?
The Ottawa ankle and foot rules are 100% sensitive and 40% specific in the identification of ankle and foot fractures.
- Radiography of the ankle is indicated if any of the following is present on physical examination:
  - Bone tenderness of the medial malleolus
  - Bone tenderness of the lateral malleolus
  - Inability to bear weight
- Radiography of the foot is indicated if any of the following is present on physical examination:
  - Tenderness at the base of the fifth metatarsal
  - Tenderness over the navicular
  - Inability to bear weight

37. What is the best method for measuring ankle swelling?
Both the figure-of-eight tape measure and volumetric immersion are valid measurements of swelling. The figure-of-eight tape measure is a simple method to track rate and amount of progress during rehabilitation. The patient should be in a long sitting position with the distal one third of the leg off the plinth in a plantar-flexed position. The tape measure surrounds the most superficial aspect of the malleolus and then travels around the foot medially over the superficial aspect of the navicular and laterally over the cuboid bone to meet at the dorsum of the foot, resulting in a figure-of-eight pattern.

38. What are the guidelines for return to activities and sports after ankle sprains, and what is the best evidence to prevent recurrent sprains?
Although each patient should be treated individually, suggested criteria for return to sports after an ankle sprain include:
- Full range of active and passive motion at the ankle
- No limp with walking
- Strength equal to 90% of the uninvolved side
• Single-leg hop, high jump test, and 30-yard zig-zag test at least 90% of the uninvolved side
• Ability to reach maximal running and cutting speeds
  Coordination/balance training and bracing have been proven to help reduce the severity of future ankle sprains. It is also necessary to strengthen all muscles of the lower extremity. For example, if the hip abductors are weak, one may compensate with lateral trunk flexion, which causes the center of mass to deviate laterally, potentially creating an inversion force to the ankle and hindfoot.

39. What disorders may cause chronic pain after an ankle sprain?
• Tension neuropathy of the superficial peroneal nerve—Inversion sprains may stretch the superficial peroneal nerve and lead to chronic pain localized to the dorsum of the foot. Compression is found most often at the site where the nerve exits the deep fascia of the anterior compartment of the leg. Pain most often is localized to the anterolateral ankle and radiates to the anterior foot. It can be reproduced by plantar flexion and reduced by dorsiflexion. Careful physical examination and local nerve blocks are most helpful in correct diagnosis.
• Anterior or lateral soft tissue impingement—Hypertrophied synovial tissue or scarring of the ATFL can become entrapped in the joint during dorsiflexion. Entrapment is most severe in the anterolateral gutter of the ankle. A less common cause of pain is talar impingement by the anteroinferior tibiofibular ligament. Bassett and Spear hypothesized that after severe sprain, the ATFL has increased laxity, which causes the talar dome to protrude more anteriorly. During dorsiflexion, the distal fascicle of the anteroinferior tibiofibular ligament may cause impingement on the talus. Management requires removal of the fascicle.
• Cuboid subluxation—This fairly common, but often unrecognizable, condition has been reported in the literature. Most commonly the cuboid is subluxated in the plantar direction and requires dorsal manipulation. The peroneals are often weak as a result of the displaced bone.

40. What is a syndesmotic ankle sprain?
Injury of the anterior and posterior inferior tibiofibular ligaments and damage to the interosseous membrane are known as a high ankle sprain. The common mechanism is external rotation of the tibia on a planted foot. High ankle sprains are common in football and baseball. They must be differentiated from routine lateral ankle sprains. Patients have tenderness and swelling over the anterior distal leg and may have swelling and ecchymosis on both sides of the ankle. External rotation of the foot while the leg is stabilized creates pain at the syndesmosis. The squeeze test is pain elicited distally over the syndesmosis with compression of the tibia and fibula at midcalf level.
  It may be critical to rule out concurrent fracture of the fibula. Patients with a syndesmotic sprain should be referred to an orthopedic surgeon. Complete diastasis of the syndesmosis should be evaluated by radiograph, and instability may require surgery. The syndesmotic sprain typically produces longer disability than the more routine ankle sprain.

41. What are shin splints?
"Shin splints" is not a specific diagnosis. The evidence is clear that shin splint pain has many different causes from tibial stress fractures to compartment syndrome. It is preferable to describe shin splint pain by location and etiology, for example, lower medial tibial pain, resulting from periositis or upper lateral tibial pain caused by elevated compartment pressure.

42. What is the most common cause of tibial overuse syndromes?
Tibial overuse injuries are a recognized complication of chronic, intensive, weight-bearing exercise or training, commonly practiced by athletic and military populations. The most common tibial overuse injuries are anterior stress syndrome and posterior medial stress syndrome.

43. Why is anterior tibial stress syndrome (shin splints) often associated with runners?
Reber et al. using fine-wire EMG, identified that during running, the tibialis anterior muscle increased in activity and fired above the fatigue threshold for 85% of the time. This may account for the high number of fatigue-related injuries to the tibialis anterior muscle seen in runners.

44. What is the cause of posterior medial tibial stress syndrome?
Beck and Osternig identified that the soleus, the flexor digitorum longus, and the deep crural fascia were found to attach most frequently at the site where symptoms of medial tibial stress syndrome occur. These data contradict the contention that the tibialis posterior contributes more to this particular condition. Therefore specific modalities and stretching to these muscles should be beneficial.
45. **What is the best treatment for shin splints?**
Generally, the most effective treatment is considered to be rest, often for prolonged periods. In a recent review of the literature, Thacker et al. found limited evidence for the use of shock-absorbent insoles, foam heel pads, heel cord stretching, and alternative footwear as well as graduated running programs among the military. They did identify the most encouraging evidence for effective prevention of shin splints was the use of shock-absorbing insoles.

46. **Define sinus tarsi syndrome.**
The sinus tarsi is an oval space laterally between the talus and the calcaneus and continuous with the tarsal tunnel. The sinus tarsi and tarsal canal are filled with fatty tissue, subtalar ligaments, an artery, a bursa, and nerve endings. Tenderness in the tarsal sinus indicates disruption or dysfunction of the subtalar complex. Chronic ankle sprains have been cited as a common cause of sinus tarsi syndrome. Arthroscopic reports indicate scarring and synovial inflammation in the lateral talocalcaneal recess.

47. **Define tarsal coalition.**
In this structural abnormality, a fibrous or osseous bar abnormally spans two of the tarsal bones, most commonly the talocalcaneal or calcaneonavicular joint. It is most often recognized in the early teenage years. Ankle sprains, slight trauma, or growth-plate ossification are common factors that provoke pain and lead to the discovery of this condition via radiograph. Typically the pain is unrelenting. Common findings are loss of rear-foot motion and concomitant rigid pes planus. A talocalcaneal coalition is difficult to identify on radiographs; magnetic resonance imaging or computed tomography may be required. Treatment focuses initially on rest and then on treatment to increase flexibility and decrease stiffness. Surgery may be necessary to resect the bar; extreme cases may require fusion.

48. **Describe the normal mobility of the first ray. How is it assessed clinically?**
The first metatarsal should lie in the same plane as the lesser metatarsals. Normal mobility is assessed with stabilization of the lateral four toes while the examiner’s other hand applies dorsal or plantar force on the first metatarsal. Motion in plantar and dorsal directions should be equal, and during dorsal testing, the inferior aspect of the first metatarsal should reach the plane of the lesser metatarsals.

49. **What are hallux rigidus and hallux limitus, and what is the best treatment?**
Hallux limitus is restriction in metatarsophalangeal (MTP) extension. Normal walking requires 65 degrees of extension during terminal stance. Hallux rigidus is further loss of motion, characterized by the development of osteoarthritis, as evidenced by spurring or loss of joint space. Common problems associated with these two disorders include trauma to the forefoot, congenital variations in the head of the first metatarsal, and a dorsiflexed first ray.

Conservative treatment for both should focus on strengthening of the flexor hallucis longus, and taping the great toe (at the MTP joint) into flexion can limit painful dorsiflexion. Mobilization of the first MTP joint by in a dorsal direction to increase extension is indicated in hallux limitus. However, in hallux rigidus the arthritic joint needs to be protected. In most cases, mobilizing the joint will not reduce symptoms and may cause irritation.

In more chronic cases, treatment is focused on decreasing the force to the MTP by using a stiff-soled shoe, external metatarsal bar, or orthotic modifications such as a metatarsal bar and full contact orthoses.

50. **What is the consequence of a hypomobile first ray?**
Patients with a hypomobile first ray present with callus formation under the first metatarsal and hallux, suggesting shear and compressive forces. The problems result from inability of the first ray to dorsiflex with weight acceptance, which causes increased plantar pressure under the first ray. Patients report pain with walking, primarily at the end of stance, and with passive extension as well as decreased range of motion in dorsiflexion of the first MTP joint.

51. **Describe bunions and hallux abducto valgus (HAV) deformity and discuss risk factors.**
A bunion refers to just the bony prominence or exostosis on the medial aspect of the first MTP. A bursa can form over the enlarged joint, which can then become inflamed and painful.

HAV deformity consists of abduction or medial deviation of the first metatarsal and adduction or lateral deviation of the hallux (phalanges), which then can lead to the bunion. HAV deformity is one of the most common and disabling pathologies of the foot.

The cause of HAV is multifactorial but commonly includes genetics and improper shoe wear. It is interesting that the incidence of HAV is approximately 2% in cultures that do not wear shoes.
and 44% in women and 22% in men who wear shoes. Abnormal biomechanics with excessive pronation and first ray hypermobility contribute to altered forces that can stretch the MTP joint ligaments and weaken the intrinsic muscles of the foot. In addition, the altered positioning shifts the forces at the MTP joint, changes the moment arms angles, and thus propitiates the pronation and abnormal alignment of the MTP joint out of the sagittal plane.

There are many other conditions associated with developing HAV from arthritic causes like RA, OA, neuromuscular conditions such as MS and CVA, and genetic causes such as Down syndrome. Therefore any disorder that can affect the balance of the toe flexors and extensors can lead to HAV.

52. Should you treat asymptomatic HAV?
No, treatment is not warranted.

53. What interventions are helpful in those with painful HAV?
The goal of nonoperative treatment is to stop or even reverse the progression of degeneration at the first MTP and the compensatory overload on the second toe and resultant second toe deformities.

- To treat the pain and immediate symptoms
  - Night splint to align the MTP joint and balance the pull of ligaments
  - Wider shoe to decrease pressure on irritated bunion
  - Padding the bunion to reduce direct pressure
  - Taping arch or taping MTP sesamoids or combination for stabilization

- To treat the progression of the problem
  - Foot exercises to balance muscle strength and improve proximal hip stability and first ray stability
  - Seated calf raises with focus on first MTP joint extension
  - Foot fists to work on foot intrinsics
  - Lunge progression with foot in neutral arch and plantarflexed first ray
  - Gait training with focus on shifting weight under the first MTP joint at preswing
  - Achilles and soleus stretching if short or stiff
  - A custom-made, full-contact orthosis to decrease abnormal pronation. An extension under the first MTP joint may be helpful.
  - Manual therapy
  - Plantar glides of first metatarsal head with goal of increasing first MTP motion
  - Sesamoid glides; proximal and distal
  - Talocrural glides or mobilization with movement with goal of improving normal ankle ROM

54. What is the typical surgery for a bunion or hallux abductus valgus (HAV)?

It is very important to differentiate surgery for a bunion verses surgery for HAV.

Surgery to remove the bony prominence and/or inflamed bursa at the base of the MTP joint is called a bunionectomy. On the other hand, many different surgical techniques exist for HAV. The most common is an osteotomy to realign the bones of the foot that are causing the deformity. Repair of tendons and ligaments, which are imbalanced, is often combined with an osteotomy. Other corrections may be removal of bone from the end of the first metatarsal, reshaping the MTP joint (resection arthroplasty), fusion of the MTP joint, fusion of the first cuneiform and first metatarsal (Lapidus procedure) or implant of an artificial joint. It is important to communicate with the surgeon to understand the precise surgery performed on your patient.

55. What should be considered with rehabilitation after HAV?
The recovery from HAV surgery can be painful and often more involved than the patient expected. The foot is initially protected for the first 3 to 4 weeks in a stiff walking shoe or boot.

Active and passive ROM of the first MTP is started early on to avoid irreversible stiffness. In the early stages, patient education regarding swelling and pain management should be implemented.

Patients are often fearful to bear weight medially under the first metatarsal, so gait training is key. The goal is to restore balance, strength, and normal biomechanics to the foot and the entire lower kinetic chain. Schuh et al. recently confirmed by plantar pressure analysis that postoperative physical therapy and gait training lead to improved function and weight bearing of the first ray after hallux valgus surgery.

56. Describe the windlass mechanism. How can abnormal mechanics lead to pathology?
From midstance to terminal stance in gait, full body weight is transferred to the metatarsal heads. As a result, the MTPs extend and activate the windlass mechanics, tightening the tissues on the plantar aspect of the foot and elevating the arch.
Dorsal movement of the navicular results in plantar flexion of the first ray. Plantar flexion of the first ray allows the phalanges to glide, resulting in dorsiflexion of the first MTPs. If plantar flexion of the first ray is not achieved, dorsiflexion cannot occur at the MTPs, and the windlass mechanism is lost. This leads, in turn, to loss of the structural stability of the foot. If the foot remains excessively pronated for any number of reasons, the windlass loses its effect. The loss of the windlass mechanism may result in the following clinical pathologies:

- Joint laxity of the metatarsals
- Metatarsalgia
- Formation of hallux valgus

57. Describe hammer toes and mallet toes. How are they treated?
A hammer toe is MTP extension with proximal interphalangeal (PIP) flexion, which may be a flexible or fixed deformity. Pain often results from a callus on the dorsum of the PIP and under the metatarsal head. Hammering of the second toe often is accompanied by a hallux valgus deformity. A mallet toe is a toe deformity that results in flexion of the distal interphalangeal (DIP). Treatment for hammer and mallet toes includes stretching of the dorsal extrinsics in a position of ankle plantar flexion and MTP extension, strengthening of the intrinsics, padding over the dip or pip, and toe tip, and wearing a deeper shoe.

58. Define claw toes. How are they treated?
Claw toe is also an extension deformity of the MTP joint with concomitant flexing or “clawing” of the toe at both the proximal and distal interphalangeal joints. The claw toe results from muscle imbalance in which the active extrinsics are stronger than the deep intrinsics (lumbricals; interosseus) and may indicate a neurologic disorder. It is commonly seen with high arches (cavus foot). Stretching, as with the hammer toe, is often successful with flexible deformities, and shoes should avoid unnecessary pressure.

59. What is sesamoiditis?
Active people may develop a problem in the two small bones (sesamoids) that lie in the tendon of the flexor hallucis brevis muscle under the first MTP joint. The medial digital plantar nerve also runs in close proximity to the medial sesamoid and can be irritated. Patients with an inflamed sesamoid find it quite painful to ambulate. They have palpable pain at the first MTP joint, pain on extension of the great toe, and often swelling at the head of the first metatarsal. The differential diagnosis should include fracture of the sesamoid and bipartite medial sesamoid.

60. What is metatarsalgia?
Metatarsalgia refers to an acute or chronic pain syndrome involving most commonly the second and third metatarsal heads. Pain also prevents extension at the MTP joint and is provoked by gait. The various causes include overuse, anatomic misalignment, foot deformity, and degenerative changes. Other nonmechanical causes include RA, gout, or other arthropathies. A cavus foot, which places more weight on the distal end, is commonly seen with this disorder.

Metatarsalgia of the first MTP joint often results from a traumatic episode or degenerative arthritis. Patients should be screened for a hallux valgus rigidus as well as sesamoiditis.

61. In general, what is the best conservative treatment for forefoot disorders?
- Change pressure under the tender area with a metatarsal pad proximal to the pain or cut-out under orthoses.
- Change ill-fitting shoes and educate on improper shoe wear, such as avoiding high heels and shoes that are too flexible.
- Improve MTP flexion and IP extension by strengthening intrinsics with manual and weight-bearing exercises.
- Maintain correct arch position by strengthening in an arched or short-foot position.
- Taping to unload fat pad or claw-hammer toe correction
- Taping for arch support with the modified low-dye technique
- Joint mobilization: first MTP joint, sesamoid glides, and subtalar and ankle mobilization joints and calf stretching to allow for normal progression over foot during gait

62. Where is the most common site of a neuroma? Describe the symptoms of a neuroma.
Neuromas are found most commonly in the third web space between the third and fourth metatarsals. Neuromas at the first and fourth web spaces are rare. Patients complain of deep burning pain and may have paresthesia extending into the toe. The main symptom is pain in the
plantar aspect of the foot, which is increased by walking and relieved by rest. The neuroma is secondary to irritation of the intermetatarsal plantar digital nerve as it travels under the metatarsal ligament. Pain often is elicited with MTP extension, which tightens the ligament and compresses the nerve.

63. How is a neuroma diagnosed?
Palpation in the interspace as opposed to over the joint should provoke the patient’s pain. Mulder’s sign is where the clinician squeezes the forefoot while palpating the involved interspace with the thumb and index finger of the other hand. A positive test is reproduction of pain and a possible audible click.

64. What is the suggested treatment for neuromas?
Physical therapy intervention includes shoe modification (specifically a wider toe box), metatarsal pads, and orthosis. Foot orthotic therapy can be very effective and, compared with metatarsal pads, can reduce plantar pressures across the forefoot and restore loading of the first metatarsal.

Joint mobilization to increase intertarsal motion is often necessary as well as deep soft tissue mobilization. Neurodynamics should be assessed and treated because the nerve may be compressed more proximally as well as locally. Steroid injection is often helpful to decrease inflammation, and, in chronic, unrelenting cases, referral for surgical neurectomy may be necessary.

65. How is the level of protective sensation tested?
The Semmes-Weinstein microfilament test is a simple, inexpensive, and effective method for assessing sensory neuropathy in patients at risk for developing foot ulcers. Patients unable to feel the nylon filament with a 10-gram bending force are diagnosed with loss of protective sensation. They benefit from protective footwear and a foot care education program.

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CHAPTER 76 QUESTIONS

1. All of the following are present in stage II posterior tibial tendinosis except:
   a. The “too many toes” sign
   b. Rigid subtalar joint
   c. Inability to perform a heel rise
   d. Tenderness along the tendon

2. Which is the most appropriate intervention during the first 4 months after Achilles tendon repair?
   a. High-volume eccentric exercise
   b. Calf and Achilles stretching
   c. Ankle joint mobilization
   d. Early ROM and weight bearing

3. According to the American Physical Therapy Association’s published guidelines for heel pain, evidence is strongest for which of the following interventions?
   a. Iontophoresis
   b. Intrinsic foot exercises
   c. Laser therapy
   d. Foot orthotics

4. Which of the following is the most appropriate exercise for a patient with stage II PTTD?
   a. Open chain plantar flexion and inversion with a theraband
   b. Resisted foot adduction with the foot in contact with the floor, in a windshield-wiper type of motion
   c. Eccentric heel lowering
   d. Exercise should not be performed in this stage
1. How are ankle fractures classified?
Ankle fractures are described by the number of malleoli involved:
- Single malleolar fracture is a lateral or medial malleolar fracture.
- Bimalleolar fracture is a fracture of both the medial malleolus and the lateral malleolus.
- Trimalleolar fracture is a fracture of the lateral malleolus, medial malleolus, and posterior aspect of the distal tibial articular surface.

There are two major classification systems for ankle fractures: the Weber/AO classification and the Lauge-Hansen classification (more complex). Fractures are classified to dictate treatment, simplify communication between medical personnel treating the fracture, and predict outcome.

The Weber/AO classification is the simplest method to classify ankle fractures:
- Weber A—below the level of the syndesmosis
- Weber B—at or near the level of the syndesmosis; 50% have disruption of the syndesmosis
- Weber C—above the level of the syndesmosis; >50% have disruption of the syndesmosis

The four Lauge-Hansen classes are (the first term in parentheses refers to the foot position and the second term describes the external force applied to the ankle):
- Supination-Adduction (SA, 10%–20%)
- Supination-External Rotation (SER, 40%–75%)
- Pronation-Abduction (PA, 5%–20%)
- Pronation-External Rotation (PER, 5%–20%)

2. What are the indications for surgical treatment of an ankle fracture?
- Intraarticular displacement of the distal tibial surface of 2 mm or more requires surgical intervention.
- Distal tibiofibular ligament rupture, causing widening of the ankle mortise, is an indication for surgery.
- Any injury that causes two breaks in the ankle joint “ring” requires surgery. The ankle is a hinge joint in which the malleoli are connected to the talus through the collateral ligaments. Bimalleolar fractures are inherently unstable and require surgical stabilization. A fibular fracture combined with a deltoid ligament tear is a bimalleolar equivalent fracture and also requires surgery.
- Any fracture that allows the talus to shift laterally or medially in the mortise is treated surgically.
- Ankle fractures that involve only one malleolar disruption and do not disturb the stability of the ankle mortise are treated nonoperatively; the patient wears a short-leg walking cast or fracture boot for 4 to 6 weeks.

3. Describe the radiographic views and alignment guides used in assessing ankle fractures.

**ANTEPOSTERIOR VIEW**
- Measured 1 cm proximal to the ankle joint, the tibiofibular overlap should be >6 mm.
- Measured 1 cm proximal to the ankle joint, the tibiofibular clear space should be <6 mm.

**LATERAL VIEW**
- Assess joint line, talus, calcaneus, and posterior tibial fracture

**MORTISE VIEW**
- Tibiofibular line should be continuous
- Talocrural angle—normally 8 to 15 degrees or within 2 to 3 degrees of opposite side
• Talar tilt—0–1.5 degrees; talus may tilt upward to 5 degrees in a normal ankle with inversion stress
• Medial clear space—normally equal to the superior clear space (should be <4 mm)
• Tibiofibular overlap should be >1 mm

SPECIALIZED STRESS VIEWS
• Mortise view with inversion stress—talar tilt is normally <5 mm; twofold difference from uninjured ankle is abnormal; tear >10 to 15 degrees indicates tear of the anterior talofibular ligament (ATFL) and calcaneofibular ligament (CFL)
• Mortise view with external rotation stress—this view stresses the deep deltoid ligament. It is performed to test deep deltoid ligament stability in a patient with a distal fibular fracture. If the medial tibiotalar space widens more than 4 mm with this test, then the deep deltoid ligament is torn and surgical stabilization of the distal fibula is warranted.
• Lateral view with anterior drawer—anterior talar shift >8 to 10 mm indicates ATFL tear

4. Describe the complications and outcomes of ankle fractures.
Potential complications after open reduction and internal fixation (ORIF) include nonunion (about twice as common in diabetic patients), malunion, wound breakdown (2%–3%), infection (2%), reflex sympathetic dystrophy, and arthritis (10% in anatomic reductions, 90% with malreduction).

5. Describe other fracture patterns around the ankle.
• Maisonneuve—pronation-external rotation fracture with fracture of the proximal fibula
• Curbstone—isolated posterior malleolus fracture
• Le Fort-Wagstaffe—anterior fibular avulsion; supination-external rotation injury
• Tillaux-Chaput—anterotibial avulsion
• Pronation-dorsiflexion fracture—fracture of anterior articular surface
• Nutcracker fracture—avulsion fracture of the navicular with comminuted compression fracture of the cuboid
• Pilon—high-energy compression fracture through tibial plafond (50% develop arthrosis or other major complication; 26% require fusion)

6. What is the Hawkins classification of talar neck fractures?
Talar neck fractures usually result from hyperdorsiflexion injury, as in a motor vehicle accident or fall from a height. Hawkins classified the different types of fracture patterns as follows:
• Type I—vertical nondisplaced talar neck fracture
• Type II—displaced fracture with subluxation or dislocation of the subtalar joint
• Type III—type II with talonavicular dislocation
• Type IV—type III with talonavicular dislocation

7. Describe the treatment and outcomes for talus fractures.
Treatment is usually surgical in light of problems with late displacement and prolonged immobilization. The risk of avascular necrosis (AVN) increases with Hawkins type (type I = 0%–13%, type II = 20%–50%, and type III = 20%–100%). Approximately 40% to 90% of patients suffer late arthritis.

8. What is Canale’s view?
This radiographic view provides optimal visualization of the talar neck. The radiograph is taken with the foot in maximal planar flexion and pronated at 15 degrees; the x-ray tube is directed 15 degrees cephalad to the vertical.

9. What radiographic views and lines are used to evaluate calcaneal fractures?
• Broden’s view—foot in neutral position and leg internally rotated 30 to 40 degrees. Views are angled 10, 20, 30, and 40 degrees cephalad. Broden’s view allows visualization of the posterior facet but has largely been replaced by CT scan.
• Bohler’s angle—first line from anterior calcaneus to highest posterior articular surface and second line from posterior articular surface to posterior tubercle. The normal angle is 25 to 40 degrees. A decreased angle indicates posterior facet collapse.
• Angle of Gissane—formed by the two cortical struts, one along the posterior facet and the other to the anterior process of the calcaneus. The normal angle is approximately 140 degrees. An increased angle indicates posterior facet collapse.

10. What are the outcomes of calcaneal fractures?
Surgical treatment generally provides better outcomes than nonoperative treatment. Nonoperative treatment includes casting and strict nonweight bearing for 6 to 8 weeks, followed by progression
of weight bearing. Surgical treatment is ORIF. Complications include arthritis, peroneal impingement (10%–20%), widening of heel, decreased dorsiflexion, weak plantar flexion, leg length discrepancy, wound dehiscence, and sural nerve injury. Approximately 65% of patients are limited in vigorous or sports activities, 50% are able to ambulate over any surface, and 40% are unable to return to previous employment.

11. How is a bipartite sesamoid distinguished from a sesamoid fracture?
Bipartite sesamoids occur in 10% to 30% of the population and may be easily confused with an acute fracture. Bipartite sesamoids are bilateral in 85% of cases, have smooth sclerotic borders, and exhibit no callus after several weeks of immobilization. A bone scan will show increased uptake of technetium in a fractured sesamoid but will be normal in the case of a bipartite sesamoid. MRI may show increased signal around a fractured sesamoid.

12. What is a pilon ankle fracture and how is it treated?
A pilon fracture is an intraarticular fracture of the distal tibia produced by dorsiflexion and/or axial loading forces. The term pilon refers to the talus as the pestle driving into the mortar-like ankle mortise, producing a fracture of the weight bearing surface of the tibia.

The Ruedi-Allgower classification describes pilon fractures as follows:
- **Type I**—nondisplaced
- **Type II**—displaced
- **Type III**—displaced with joint surface comminution and impaction

The recommended treatment of displaced fractures is surgery. In low-energy fractures without significant soft tissue energy and swelling, ORIF is indicated 10 to 14 days after soft tissue swelling subsides.

13. What complications can occur after pilon fractures?
The most frequent complication after pilon fracture treatment is posttraumatic arthritis (50%–70%). Other complications include wound healing problems, dehiscence, nonunion, malunion, and pin tract infections. In type III fractures, the goal is often to achieve soft tissue healing and sufficient bony healing of the metaphyseal bone to allow for fusion at a later date.

14. What common fractures are frequently misdiagnosed as ankle sprains?
- Talar osteochondral fracture (a divot fracture involving bone and cartilage usually from the anterolateral or posteromedial talar dome)
- Lateral talar process fracture
- Anterior calcaneal process fracture
- Posterior talar process fracture
- “Flake fracture” of the posterior distal fibular rim, indicating a tear of the superior peroneal retinaculum and peroneal tendon dislocation
- Navicular fracture
- Lateral or medial malleolar fracture

15. What is the pathophysiology of stress fractures of the foot?
A stress or fatigue fracture is a break that develops in bone after cyclical, submaximal loading. In states of increased physical activity, bone is resorbed faster than it is replaced, which results in physical weakening of the bone and the development of microfractures. With continued physical stress, these microfractures coalesce to form a complete stress fracture. Middle-aged and older adult patients with osteoporosis, diagnosed with a T score of lower than 2.5 on dual photon spectrometry (DEXA scan), are also at risk for stress fractures. Amenorrheic athletes are predisposed to stress fractures; amenorrhea is present in up to 20% of vigorously exercising women and may be as high as 50% in elite runners and dancers.

16. What are common locations for stress fractures of the foot?
Common locations for foot stress fractures are the metatarsals, calcaneus, and navicular. Distal tibial stress fractures and lateral malleolar fractures are less common. Symptoms of stress fracture are localized pain and swelling with weight bearing of insidious onset. A thin sclerotic line may be seen in a stress fracture of metaphyseal bone. Although initial radiographs may be negative, a technetium bone scan is positive as early as 48 to 72 hours after onset of symptoms.

17. Describe the Sanders classification of calcaneal fractures.
Any displaced calcaneal fracture should be evaluated with a CT scan to determine the degree of posterior facet displacement and comminution. The Sanders classification uses CT scanning in the
coronal plane to describe the number of posterior facet fragments and their location. Sanders classifies an extraarticular fracture as a type I. A posterior calcaneal facet fracture with two pieces is a type II, a fracture with three pieces is a type III, and a fracture with four pieces is a type IV. Using this system, Sanders has reported results of ORIF of displaced intraarticular fractures by fracture type: 73% of type II fractures had a good or excellent result, 70% of type III fractures had a good or excellent result, and only 9% of type IV fractures had a good or excellent result.

18. What injuries are commonly associated with calcaneal fractures?
The calcaneus is the most fractured tarsal bone. Approximately 75% of calcaneal fractures are intraarticular. Approximately 10% of patients have associated fractures of the spine, 25% have extremity injuries, 10% are bilateral, and 5% are open.

19. How are calcaneal fractures treated?
- Nondisplaced fracture—6 weeks of splinting, elevation, nonweight bearing, and early motion typically yield acceptable results.
- Displaced fractures—treatment is controversial. Surgery is performed 10 to 21 days post injury to allow for edema resolution and healing of fracture blisters. Operative treatment of displaced intraarticular fractures through an extensile L-shaped incision yields excellent exposure but carries a higher risk of complications than nonoperative treatment. Results at 1 year post injury have been shown to be similar between nonoperative and operative treatment. Operative treatment yielded better results at 8 to 12 years’ follow-up along with a lower incidence of posttraumatic arthritis. Calcaneal reductions through smaller incisions and percutaneous techniques have been developed to avoid wound complications while allowing for fracture reduction to prevent posttraumatic subtalar joint arthritis. Contraindications for calcaneal open reduction internal fixation include poor vascular supply, insulin-dependent diabetes, tobacco abuse, venous stasis, neuropathy, unhealed fracture blisters, and elderly or inactive patients.
- Open calcaneal fractures—immediate operative debridement, intravenous antibiotics, and splinting are the course of treatment. No internal fixation or limited percutaneous fixation is used in open fractures because of the significant risk of wound dehiscence, infection, chronic osteomyelitis, and amputation.

20. What are expected outcomes after calcaneal fractures?
Young, active patients with closed noncomminuted fractures are potential candidates for ORIF through an extensile lateral L-shaped incision. Surgery is performed 10 to 21 days after fracture to allow time for edema reduction. Type II and III fractures have shown good to excellent results in 70% to 85% of patients following this protocol. This compares favorably to 40% to 60% acceptable results following nonoperative management. A splint is used postoperatively until wound healing is documented; then early motion is begun. Weight bearing is delayed for 8 to 10 weeks.

21. What fractures of the foot are at risk for avascular necrosis and why?
In the foot the bones at risk for AVN are the talus and navicular. The talus is 60% to 70% articular cartilage and has no tendinous attachments. Most of the talar body blood supply enters the undersurface of the talar neck and flows posteriorly. The talus is at particular risk for osteonecrosis with displaced fractures of the talar neck. The weakened bone of the osteonecrotic segment of the talar dome may then collapse, causing pain and arthritis in the ankle and subtalar joints. In some instances, talar osteonecrotic segments will heal spontaneously over a course of 2 to 3 years in a process known as “creeping substitution,” in which the dead bone is resorbed and replaced by live bone. The large articular surface area of the navicular also limits blood supply to the dorsal and plantar aspects. Blood perfusion is diminished to the central third of the navicular. AVN with late partial collapse of the navicular is common in comminuted navicular fractures.

22. What is a Lisfranc joint injury?
The Lisfranc joint, or tarsometatarsal joint, consists of the articulations between the five metatarsals, three cuneiforms, and cuboid. Stability is enhanced by the archlike configuration of the joint in the coronal plane and by the dorsal and plantar tarsometatarsal and intermetatarsal ligaments. The recessed second metatarsal base is connected to the medial cuneiform by the important Lisfranc ligament. Most Lisfranc injuries occur by forceful external rotation and pronation of the foot. A fall onto a maximally plantar-flexed foot can cause dorsal displacement of the metatarsals. Direct crushing injuries are less frequent causes of Lisfranc injury.
23. What are the classification patterns and treatments for Lisfranc injuries?
Lisfranc injuries are classified into three injury patterns:
• Homolateral—all metatarsals are displaced in the same direction.
• Divergent—metatarsals are displaced in both sagittal and coronal planes in differing directions.
• Isolated—one or more metatarsals are separated from the remaining Lisfranc complex.
An anatomic reduction is required for the best result. Despite anatomic fixation, some patients will develop posttraumatic arthritis, requiring fusion at a later date. Fifty percent of patients will have some long-term disability.

24. What are the classification and treatment of a fifth metatarsal base fracture (Jones fracture)?
The fifth metatarsal metaphyseal-diaphyseal region is a watershed area of blood supply, which makes this fracture location notorious for delayed union and nonunion. The Torg classification describes three types of fractures based on anatomic location. A type I fracture involves the tuberosity and extends into the metatarsocuboid joint. A type II fracture (true Jones fracture) extends into the fourth and fifth metatarsal joints. A type III fracture involves the proximal fifth metatarsal shaft and is distal to the fourth and fifth metatarsal joints. Type III fractures may also occur as a stress fracture in athletes. The more the distal the fracture, the greater the risk of nonunion.

Type I fractures can be successfully managed in a fracture boot or postoperative shoe for 6 to 8 weeks or until healing occurs. Type II and III fractures should be managed with a period of nonweight bearing cast immobilization or immediate intramedullary screw fixation. Recent studies suggest that screw fixation is the preferred treatment for the acute type II or III fracture in the athlete. Intramedullary screw fixation is also recommended for treatment of delayed union or nonunion of these fractures.

25. What is a Charcot arthropathy or neuropathic arthropathy, and how is it classified?
A Charcot arthropathy, or neuropathic arthropathy, is a process of chronic, noninfective, painless joint destruction. Charcot first described this condition associated with tabes dorsalis in 1868. Approximately 0.1% to 0.5% of diabetic patients will develop a neuroarthropathic joint. Two theories explain the development of a Charcot joint. The neurotraumatic theory states that decreased protective sensation and cumulative mechanical trauma lead to fracture and joint destruction. The neurovascular theory states that a neurally initiated vascular reflex leads to increased resorption by osteoclasts. Studies have shown increased osteoclastic activity without a concomitant increase in osteoblastic bone formation in the feet of diabetic patients.

The presenting symptoms of a Charcot foot include the spontaneous onset of a warm, swollen foot that is associated with no pain or vague pain. The midfoot is most commonly involved, followed by the hindfoot and ankle. The midfoot will lose its arch over time, and the forefoot will dorsiflex and abduct, producing a rocker-bottom foot deformity. The patient is then predisposed to plantar ulceration of the prominent plantar arch. The ankle will develop a significant varus or valgus deformity with eventual corresponding pressure ulceration over the prominent malleolus.

Early radiographs may show osteopenia with intact joints. Later radiographs will show fractures, joint subluxation or dislocation, and bone destruction and fragmentation. The following clinical and radiographic stages of Charcot joints have been described:
• Stage 0—neuropathic patient with a history of sprain or fracture
• Stage 1—inflammatory stage with edema, hyperemia, erythema, and bone fragmentation on x-ray
• Stage 2—reparative stage with less swelling and erythema; radiographs show new bone formation at site of fracture and dislocation
• Stage 3—consolidation phase with resolution of swelling; radiographs show bony healing of fractures and dislocations

Brodsky has classified the Charcot foot based on anatomic location. Type 1 involves the tarsometatarsal and naviculocuneiform joints. Type 2 involves the subtalar and/or Chopart joint. Type 3A involves the ankle joint. Type 3B involves a fracture of the calcaneal tuberosity.

26. How is a Charcot arthropathy/neuropathic arthropathy diagnosed and treated?
The key to treatment of the Charcot foot is making the diagnosis. A clinician should have a high index of suspicion for the neuropathic patient with a warm, swollen foot. The acute Charcot foot is warm, erythematous, and swollen, mimicking a diabetic foot infection. Radiographs, bone scans, and MRI scans show findings indistinguishable from osteomyelitis. Charcot patients will typically be afebrile and have a
normal white blood cell count, normal C-reactive protein, and normal erythrocyte sedimentation rate. The swelling and erythema in a Charcot foot will usually resolve with bed rest and elevation. A combination, three-phase technetium bone scan and indium-labeled WBC scan has been shown to have high sensitivity and specificity rates for differentiating Charcot from infection. The mainstay treatment of Eichenholtz Stage 1 Charcot is a total contact cast. This type of cast incorporates felt padding over the malleoli and tibial crest, unloads plantar prominences with a plastizote insert, and encloses the toes. The cast is changed every 2 to 4 weeks for 2 to 4 months until radiographic healing progresses to Eichenholtz Stage 2. Weight bearing is allowed in the cast. A Charcot restraint orthopedic walker (CROW) boot is an alternative to total contact casting. In Stage III, the patient may progress to walking in extra depth shoes with custom diabetic inserts. Surgical treatment is reserved for recalcitrant ulceration, unbraceable deformities, acute fractures and dislocations, and infections. Surgery ranges form the simple removal of a bony prominence through plantar exostectomy to extensive midfoot or hindfoot fusion with internal or external fixation.

27. How are talar fractures classified?
Fractures of the talus are characterized by location. The different types of fractures are talar body fractures, fractures of the lateral talar process, fractures of the posterior talar process, osteochondral fractures of the talar dome, and talonavicular fractures. Any of these fractures may be misdiagnosed as an ankle sprain.

Hawkins and Canale have classified talar neck fractures as these four types:
- Type I—nondisplaced vertical fracture of talar neck
- Type II—displaced talar neck fracture with subluxation or dislocation of the subtalar joint
- Type III—displaced talar neck fracture with dislocation of both subtalar and ankle joints
- Type IV—type III fracture with talonavicular subluxation or dislocation

28. How are talar fractures treated?
Type I fractures of the talar neck are treated with immobilization for 3 months. Type II to IV fractures are treated with closed reduction. If an acceptable reduction is achieved, nonweight bearing cast immobilization is used for 3 months. In the case of displaced fractures, which are irreducible, open reduction and screw fixation are indicated. Outcomes related to talar fractures treated with ORIF have demonstrated an incidence of osteoarthritis of up to 100%, osteonecrosis 50%, and nonunion 12%. Osteonecrosis after a talar neck fracture occurs only when the subtalar joint is dislocated. In cases of talar body osteonecrosis, 44% will spontaneously revascularize without collapse.

29. What is the Hawkins sign?
The Hawkins sign is the appearance of talar dome subchondral atrophy or lucency on the ankle AP view at 6 to 8 weeks after a talar fracture. This indicates that the talar body is vascular and excludes the diagnosis of osteonecrosis. If the talar body appears denser and more sclerotic than the surrounding bone, then osteonecrosis is present.

30. How are osteochondral talar dome fractures classified?
Osteochondral talar dome fractures or talar osteochondritis desiccans lesions are believed to be caused by trauma, although idiopathic avascular necrosis may also be a factor. Location in the talar dome is either posteromedial or anterolateral. The classic Brendt and Harty classification is based on plain radiographic appearance and is most commonly used to classify these lesions:
- Type I—compression of subchondral bone
- Type II—incomplete fracture
- Type III—complete, nondisplaced fracture
- Type IV—completely detached, displaced fragment

These lesions are best evaluated by CT scan or MRI. More recent CT classifications include subchondral cystic lesions in the talar dome.

31. How are osteochondral talar dome fractures treated?
Initial treatment is immobilization of nondisplaced acute lesions (types I to III); type IV fractures require surgery. Ankle arthroscopy allows for inspection and treatment of the lesion with removal of loose cartilage or bone and drilling of the subchondral bone to stimulate growth of fibrocartilage. In cystic lesions with intact articular cartilage, drilling and bone grafting can be achieved through the talar body. For lesions larger than 1 cm², drilling is inadequate to restore cartilage coverage of the lesion, and osteochondral grafting taken from the ipsilateral femoral condyle or from allografts is indicated.
32. What is compartment syndrome of the foot, and how is it diagnosed?
When a foot is subjected to significant blunt trauma, crush injuries, or high-energy fracture, swelling occurs, which leads to increased compartment pressure. Pressures greater than 30 to 40 mm Hg for longer than 8 hours result in permanent muscle injury, loss of sensation, and muscle contractures.

The foot has five muscle compartments—medial, central, lateral, interosseous, and calcaneal. Classic signs of compartment syndrome are swelling, pain out of proportion to injury, pain on passive stretch of toes, and paresthesias or sensory loss. Loss of pulse or poor capillary refill is not a sign of compartment syndrome but of vascular compromise. Definitive diagnosis is made by measurement of compartment pressures using a handheld pressure monitor. This is accomplished surgically through two longitudinal dorsal forefoot incisions and one medial midfoot incision. Wounds are left open for later secondary closure or skin grafting.

33. How does the presence of diabetes affect ankle fracture outcomes?
It is estimated that one third of the U.S. population born after 2000 will develop diabetes. The presence of diabetic neuropathy or vasculopathy complicates soft tissue and bony healing after an ankle fracture. Diabetics have higher complication rates to include infection rates, nonunions, delayed unions, delayed wound healing, and amputations. Postoperative emphasis on blood sugar control is important to optimize wound healing. Neuropathic patients may benefit from superconstructs using multiple syndesmotic screws, rigid locked plating, or external fixation in conjunction with internal fixation. A longer period of postoperative immobilization and nonweight bearing is needed with diabetic patients compared with nondiabetic patients after internal fixation to ensure bony healing.

BIBLIOGRAPHY


**CHAPTER 77 QUESTIONS**

1. Why do ankle fractures in poorly controlled diabetic patients have poorer outcomes than nondiabetic patients?
   a. Wound healing in diabetic patients is compromised.
   b. Diabetics with neuropathy are more likely to be noncompliant and walk on their injured extremity against medical advice.
   c. Infection rates are higher in diabetic patients.
   d. All of the above

2. Which of the following bones in the foot and ankle are not prone to nonunion?
   a. Lateral malleolus
   b. Talus
   c. Navicular
   d. Fifth metatarsal base

3. A diabetic patient with numb feet presents to the ER with a minimally painful, warm, swollen, red foot. Foot radiographs show severely displaced fracture dislocations of the Lisfranc joint. The skin is intact. The patient is afebrile and has a normal leukocyte count. When the patient is placed at bed rest with the foot elevated, the swelling and erythema both improve. The most likely diagnosis is:
   a. Osteomyelitis of the midfoot
   b. A destructive neoplasm of the midfoot
   c. Gangrene of the foot
   d. Charcot arthropathy, or neuropathic arthropathy, of the midfoot

4. Which of the following ankle fractures can be safely treated without surgical intervention?
   a. A displaced bimalleolar ankle fracture
   b. A lateral malleolar fracture with a medial tibiotalar clear space of 5 mm
   c. A lateral malleolar fracture displaced 2 mm but with no evidence of talar shift. External rotation stress view shows no evidence of medial clear space widening
   d. A distal tibial fracture with 3 mm of articular surface displacement
FOOT ORTHOSES AND SHOE DESIGN

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1. Define the subtalar neutral position. Why is it important?
   The subtalar neutral position is the position in which the head of the talus is aligned with the navicular. Radiographically, it is defined as the position where the joint lines of the talonavicular joint and the calcaneocuboid joint are continuous. The subtalar joint neutral position is used by clinicians to evaluate the amount of pronation and supination on either side of neutral position. This position is used to assess the foot for structural deformities. Subtalar neutral position also is used during weight-bearing assessment to evaluate foot structure and to determine how far from neutral the patient is functioning. It is considered to be best practice for a foot to be maintained in the subtalar joint neutral position while casting for most types of foot orthoses.

2. How is subtalar neutral position determined?
   Other than radiographic analysis, there are two common clinical methods: 1) divide the total amount of heel eversion (pronation) and inversion (supination) into thirds. The position is found by positioning the rear foot in a position that is one third from maximal pronation or two thirds from maximal supination. 2) Palpate “congruency” at the talonavicular joint. This procedure is based on creating the talonavicular alignment as described. The head of the talus is palpated on its medial and lateral aspects. Then the foot is moved between a pronated and a supinated position until the examiner feels talonavicular alignment, or “congruency.” It has been purported that the talonavicular and subtalar joints are at maximal congruency at the same times during passive range of motion assessment.

3. How reliable and valid are these methods?
   Concerns have been raised about the validity of the first method noted. The validity has been questioned secondary to variability of the axis of inclination of the subtalar joint. For the second method, the interrater reliability is poor to moderate. Intrarater reliability is much higher and may allow each clinician to develop a repeatable method for his or her own use. The reliability of the second method appears to be positively related to examiner experience.

4. What is the primary goal of a foot orthosis?
   There could be many goals of a foot orthosis. One common goal of a foot orthosis is to make the subtalar joint function around the neutral position. Functionally, foot orthoses are often utilized to facilitate and normalize the speed and magnitude of motion about the subtalar joint and midtarsal joints during gait. They are used, in this format, to facilitate more normal amounts of pronation during the initial part of stance and supination during the latter part of stance. A recent meta-analysis demonstrated that there is notable evidence that indicates that specifically molded orthotics can be utilized to decrease loading rates and vertical impact forces.

5. Does the subtalar joint function around neutral position?
   This well-accepted principle of foot function has recently been questioned as a result of research about human locomotion. Two independent research groups found that the subtalar joint demonstrates the predicted pronation/supination pattern but usually functions in a pronated position. Pierrynowski and Smith found that the subtalar joint usually was pronated during stance. During stance the “normal” foot is pronated (position) but is supinating (motion) for a longer period of time during the gait cycle. McPoil and Cornwall demonstrated that the subtalar joint did not reach a supinated position before heel rise and that it had a tendency to function around the relaxed standing position. Despite this evidence, the principle of assessing the cause of abnormal motion and reducing the amount and speed of motion has not changed.
6. Do orthotics actually control motion?
Many studies demonstrate no effect, whereas others document significant reduction in subtalar joint motion. Possible reasons for contradictory findings are errors in methodology, measurement of shoe motion instead of bone motion, measurement of skin over bone versus actual bone or joint motion, differences in the composition and types of orthoses, lack of specific orthotic prescription, and the patient’s need for foot orthoses based on foot structure. This last factor is extremely important. If a patient has no need for foot orthoses and is functioning optimally, it is unreasonable to anticipate notable improvement. Several studies have demonstrated that the effects of medial foot orthoses on reducing motion were “small” and “subject-specific.” It has been demonstrated that antipronation devices fabricated with both rearfoot and forefoot varus posting have the greatest positive effects.

7. Why do orthotics function better in the clinical setting?
Elimination of symptoms requires a reduction of the stress on the symptomatic tissue. Stress may result from the amount, speed, or timing of subtalar joint motion. Alteration in any of these three variables may reduce the stress below the symptomatic threshold or to a level that allows for healing. The studies that demonstrate the greatest effect of orthoses are performed with patients instead of subjects. If foot orthoses are designed for a specific patient, considering the function of the entire lower extremity as well as foot structure, the chance for resolution of symptoms is maximized.

8. How do foot orthoses control motion?
Motion at the subtalar joint occurs in all three cardinal planes but primarily in the frontal and transverse planes. The amount of motion expected in these planes can vary depending on the axis of inclination of the subtalar joints. Since pronation and supination are motions occurring in all three cardinal planes simultaneously, controlling motions in either the frontal or transverse planes will reduce the total amount of motion. For this reason, a medial (varus) motion, which reduces the amount of eversion in the frontal plane, also reduces the motions in the other planes.

9. When can a foot orthosis be utilized to “increase” motion?
When a patient has a rigid forefoot valgus, pronation of the subtalar joint may be blocked when the medial side of the forefoot contacts the ground. A foot orthosis that includes a forefoot valgus post allows for proper lateral-to-medial loading of the forefoot, minimizing the supinatory moment of the forefoot valgus. This allows the subtalar joint to pronate in a more normal fashion.

10. Does exercise prescription provide an added benefit to foot orthosis application?
There is no universal answer to this question. Exercises to strengthen muscles within the foot have been employed for many years. More recently, functional exercises involving the more proximal joint of the lower extremity, as well as trunk exercises, have been employed to treat symptoms caused by excessive tissue stress. Clinical judgments regarding the magnitude of dysfunction must be made to determine whether to use exercises alone, foot orthoses only, or both in providing the most efficacious care to patients. One additional concept to keep in mind is that a foot that excessively pronates will develop a morphologically shortened Achilles tendon, and a shortened Achilles tendon will produce an increase in speed and/or magnitude of overpronation. Because of these concepts, it is critical to ensure that a patient has adequate available dorsiflexion before the application of foot orthotics. Often, static stretching or dynamic stretching should be employed.

11. Are there any clinical tests that, if positive, suggest that a patient requires a foot orthosis?
One functional test addresses the general question of whether foot dysfunction is present and may require intervention. With the patient standing relaxed, the patient is asked to rotate the trunk in the transverse plane as far as possible; some have called this a pronation/supination test and other a rotary stability test. This trunk rotation should cause a reaction at the STJ. When the trunk is rotated to the right, the right STJ should supinate and the left STJ should pronate. If the foot does not react, it most likely indicates that the feet are dictating lower extremity function and that exercise alone may not be sufficient. The use of foot orthoses is not precluded if the feet do react. There are other functional tests such as a heel rise test, maximum pronation test, or the foot function index that may indicate further biomechanical assessment is indicated, but they do not necessarily predict the need for orthotic intervention.

12. What is the significance of heel eversion in a relaxed standing position?
Eversion of the calcaneus past a vertical position indicates that the rear foot is compensating for another structure because the calcaneus is resting in a less-than-optimal position for its own stability. Reasons include eversion to bring a forefoot varus deformity to the ground, compensation for a severely
tight calf group by pronating to unlock the MTJ, functional shortening of a long leg, and transverse plane problems in the spine, pelvis, and hip that cause the leg to rotate internally.

13. Why do some patients stand with most of the weight on the outside of the feet? The patient may be in a supinated position to compensate for a rigid forefoot valgus, partially compensated rear foot varus, or uncompensated rear foot varus. The subtalar joint may also be volitionally held supinated to avoid pain (eg, heel pain syndrome); however, the foot may not actually be supinated because a foot with cavus architecture looks similar. To distinguish between the supinated position of the subtalar joint and architecture, place the subtalar joint in a neutral position and assess the compensation from neutral to relaxed position.

14. What types of symptoms can be caused by a rigid plantar-flexed first ray? Symptoms of plantar fasciitis, sesamoiditis, and hallux limitus or rigidus can be caused by a rigid plantar-flexed first ray. If the plantar-flexed first ray creates a forefoot valgus, the patient may suffer from chronic ankle sprains, lateral knee pain, central patellofemoral pain, and low back pain.

15. In what situation does the heel evert very little while the STJ pronates excessively? In patients with a more vertical inclination of the subtalar joint axis, the frontal plane component (eversion) decreases and the transverse plane (abduction) component of pronation increases. Because clinicians usually assess and measure only the frontal plane component, the amount of subtalar joint pronation is often underestimated. In addition, feet with a compensated rearfoot varus deformity typically pronate excessively to reach the supporting surface. Although notable pronation has occurred, this is similarly underestimated because the foot appears normal in weight bearing. In many cases, these types of feet require more rear foot varus posting than indicated by minimal heel eversion. A deep heel on the orthotic shell also may enhance control of the predominantly transverse plane motion.

16. Why does a rear foot with a varus position fail to pronate at the STJ during weight bearing? Ligamentous structures, osseous structures, or pathology, such as arthrofibrosis, may restrict the STJ motion. A rigid forefoot valgus may prevent the use of available pronation. In addition, a patient with pain or limited hip internal rotation may voluntarily prevent pronation.

17. With restricted calcaneal motion caused by limited STJ pronation, would there ever be a case for posting the heel medially? Using a medial (varus) wedge when there is insufficient pronation would seem to be contraindicated. However, the patient may develop symptoms at end range or may avoid end range by voluntarily limiting pronation. In these cases, a medial wedge that prevents the STJ from reaching end range but does not limit the beneficial pronation can be very effective at eliminating symptoms. There is some evidence that utilizing a softer compound, total contact shell is also beneficial in these cases.

18. Can a foot that pronates excessively still lack enough pronation? When a foot has a large forefoot and/or rear foot varus deformity, all the motion available may be used just to get the foot to the ground. During locomotion this would be seen as excessive pronation. With the foot on the ground, any attempt to pronate further, for example, in order to jump, would be blocked. For the function of jumping, this abnormally pronated foot does not have enough available motion to allow for additional pronation. This is detrimental because the foot is functioning at end range during high-demand activities.

19. Can a foot that relaxes close to STJ neutral be abnormal and require orthotic intervention? A foot that has a rear foot varus and a rigid forefoot valgus has a tendency to relax in STJ neutral position during weight bearing. The rear foot varus wants the STJ to pronate, but the forefoot valgus does not allow for pronation. Orthotic posting is required in the rear foot and forefoot for normal functioning during gait. Specifically, an orthosis with a rear foot varus post and a forefoot valgus post is indicated. The forefoot valgus post allows for more normal pronation, but the rear foot varus post prevents excessive pronation.

20. What is the difference between forefoot varus and forefoot supinatus? Traditionally, forefoot varus is described as a single-plane (inversion) bony deformity, whereas forefoot supinatus is described as a triplanar soft tissue contracture. Assessment of joint mobility and symmetry of motion may distinguish between the two conditions. The orthotic treatment differs because the soft tissue supinatus may resolve, but the varus will not.
21. Are posting strategies different in children?
Most children pronate more than adults. As the calcaneus and talus endure developmental derotations, the pronation decreases. In designing orthoses for children with rear foot and forefoot varus deformities, it is probably better to post the rear foot more aggressively and to use smaller forefoot varus posts in the hope that the forefoot varus will decrease. Except for special circumstances, the concept of treating the cause of the pronation does not change.

22. What is the role of the arch of the orthotic shell?
The arch of the shell plays an important role in capturing the inclination angle of the calcaneus to control the amount of mobility at the talonavicular articulation and to capture the architecture of the foot to optimize the effects of corrective posts. In most patients, the decrease in arch height is not the cause of pronation but rather a result of STJ and midtarsal joint pronation. If the shell is used as the primary corrective component, it may need to be fabricated from a more flexible material to be tolerated by the patient.

23. What is an extended forefoot post, and when is it indicated?
In most orthoses, the shell ends behind the metatarsal heads. Therefore, the forefoot posting exerts its influence on the metatarsal shafts. Some orthoses are fabricated with a flexible post that extends under the metatarsal heads, often called a runner’s wedge or a foot post to the sulcus. This type of post may be more effective because it exerts its influence directly under the metatarsal heads. This also places correction farther under the forefoot, which can prevent pronation late into the stance phases when ground reaction forces are higher. A primary limitation of this orthotic feature is that the orthotic is significantly more bulky and thus is more difficult to fit into certain types of shoes.

24. How does function improve with a first ray cut-out?
Propulsion occurs off the medial side of the foot. It is purported that as much as 60% to 70% of forefoot stresses are through the first ray. Additionally, as the heel rises from the ground, the first metatarso-phalangeal (MTP) joint dorsiflexes (up to 70 degrees). The first metatarsal must plantar flex to allow for normal MTP dorsiflexion. If the patient has a rigid plantar-flexed first ray, excessive weight bearing under the first metatarsal head may prevent the typical plantar flexion of the first metatarsal that would occur with great toe extension during terminal stance. The first ray cut-out increases weight bearing under the second metatarsal head and provides room for requisite plantar flexion of the first metatarsal.

25. What is more important in orthotic fabrication: material selection, posting, or specific contouring of the device?
Although there is evidence that suggests that both custom molding a device to the contours of a patient’s foot and posting are both effective strategies for fabrication, Mundermann et al. suggests a total contact design may be the most effective component. There is some evidence that suggests that more rigid devices do not necessarily provide greater levels of control. Device rigidity is likely a factor of patient tolerance and comfort.

26. What problems may be associated with insufficient rearfoot varus posting with a substantial forefoot varus post?
If the rear foot pronates excessively, the MTJ is “unlocked” (mobile). The forefoot post becomes less effective at reducing motion and may actually cause the MTJ to collapse (the forefoot inverts and dorsiflexes relative to the rear foot), thus rendering the orthotic to function less than optimally.

27. Why would a patient with a large rear foot and forefoot varus complain that he or she is sliding off the lateral side of the orthosis?
Many patients with severe pronation gradually acquire a shortening of the calf muscles. Dorsiflexion at the MTJ compensates for loss of ankle dorsiflexion. One objective of the orthosis is to stabilize the MTJ. When walking with the orthosis, a patient who lacks sufficient ankle dorsiflexion may attempt to pronate on top of the orthosis, producing the feeling of sliding laterally as well as producing a complaint of an increase in local arch pressure. This compensation may also cause blisters under the shaft of the first metatarsal.

28. How does the forefoot adjust to a large degree of rear foot posting?
The mobility of the MTJ allows the medial side of the forefoot to reach the ground with a moderate amount of rear foot posting. At some point, the rear foot posting exceeds the ability of the MTJ to
compensate, and the posting creates a pseudoforefoot varus. The foot must pronate more to bring the medial side of the foot to the ground, in turn, creating a secondary problem.

29. Why should the midtarsal joint be considered when designing an orthosis?
Motion between the rear foot and forefoot occurs at the MTJ. The ability of the MTJ to compensate for surface irregularities, foot deformities, and orthotic posts depends on the amount of available motion. The amount of available motion is a function of the position of the STJ and general flexibility characteristics. Midtarsal joint mobility may influence the magnitude of both rearfoot and forefoot posts, depending on the particular posting strategies of individual clinicians.

30. How many miles can a running shoe sustain?
There is no universal answer because of the variety of midsole and outsole constructions. Generally, the average running shoe is built for a male to weigh approximately 185 pounds and a female to weigh approximately 150 pounds. Given these averages, midsoles predominantly made of EVA will last approximately 400 miles. In recent years, many shoe manufacturers have reduced the amount of EVA in the midsoles and replaced this material with neocomposites, which further complicate this answer as generally the midsoles made from neocomposites will be more durable.

31. How can orthotics relieve symptoms of a Morton’s neuroma?
The first metatarsal is purported to bear 60% to 70% of the weight at toe-off in the gait cycle. With excessive or abnormal pronation at toe-off, the hallux assumes a more dorsiflexed position, and the lesser metatarsals bear more weight than they are designed to sustain. Relative varus of the forefoot causes excessive STJ pronation at toe-off, thus creating a mobile lever at push-off instead of a rigid lever. Thus the medial longitudinal and transverse arches of the foot are compromised, causing compression and shearing of the interdigital nerves. The most severely compromised area is the third metatarsal interspace, where the medial and lateral plantar nerves converge. A biomechanical orthosis addresses the faulty mechanics, and a metatarsal pad placed proximal to the involved metatarsal heads elevates the metatarsal shafts, taking pressure off of the interdigital nerves. The apex of the metatarsal pad should be placed between the affected metatarsals.

32. What is the function of an external metatarsal or rocker bar?
The function of the metatarsal bar is to delay and decrease loading of the metatarsal heads during gait as well as to decrease early MTP joint extension as the foot moves from midstance to toe-off. The bar is placed at an apex point proximal to all five metatarsal heads and thus shifts the foot pressure proximally. External metatarsal bars significantly change the dynamics of the gait cycle and require increased patient balance; therefore, they should be used only as a secondary treatment option.

33. List common problems with foot orthotics and their possible causes.
• Arch pain or blisters on plantar foot surface—probably the arch or medial posting is too high and needs to be lowered, or the patient did not follow the break-in procedure of increasing wear by 1 hour per day for a 2-week period. This is also a common symptom when the patient lacks appropriate levels of dorsiflexion (DF), and compensation is occurring through the oblique midtarsal joint.
• Sensation of rolling to the outside of the foot—this sometimes may be normal because the medial longitudinal arch is not accustomed to weight bearing.
• This symptom will commonly resolve without intervention within the first few weeks. However, it also may indicate that the medial post is too high and that the orthotic shell is too rigid for the patient’s foot type or body weight.
• Sensation that the heel is coming out of the shoe—wearing a shoe that has a low throat and heel quarter or using an orthotic that is too thick or slick may cause this sensation.
• Symptoms persist—reevaluate biomechanics and determine whether more correction is necessary.
• Pain or blisters under metatarsal heads—ensure that all rigid shell materials end slightly proximal to the metatarsal heads.

34. Are injury rates reduced with minimalist shoes?
It has been proposed that running barefoot may potentially reduce a running injury. Many have also attempted to correlate minimalist shoe running with barefoot running. Generally, these are not the same, even for the most minimalist shoe choices. There is some evidence to suggest that barefoot running will more commonly produce a forefoot strike pattern during running. The evidence is very inconsistent that minimalist shoes can or will mimic this effect. There are instances where forefoot striking may be
beneficial, but this is certainly not true for everyone. Those who are prone to injuries or symptoms in the forefoot or Achilles tendon are not likely to be successful with a barefoot or minimalist approach. Forefoot striking during running has been demonstrated to decrease torques at the knee as well as vertical impact forces, but there are no strong correlations to a shoe being responsible for this altered impact pattern.

35. What are the effects on the foot and body of wearing high-heeled shoes?
In a normal standing position, approximately 50% of the weight is borne by the rear foot and 50% by the forefoot. A 2-inch heel shifts weight distribution: 10% is borne through the rear foot and 90% through the forefoot. With a flat-soled shoe, the angle between the body’s weight line and the horizontal is 90 degrees. With a 2-inch heel, the angle is changed to 70 degrees. Thus the body must compensate by changing joint position and muscle functions of the feet, ankles, hips, and spine to maintain an erect position. Furthermore, a 1-inch heel tilts the pelvis forward 5 degrees and a 2-inch heel 20 degrees. High-heeled shoes also force the knees to stay in relative knee flexion throughout the gait cycle. Finally, the chronic wearing of high-heeled shoes causes muscle imbalances, such as shortening of the Achilles tendon. This decreases the calf muscles’ mechanical advantage to develop power, causing loss of the natural heel-to-toe gait pattern and necessitating muscle compensations from the rest of the lower quadrant.

36. How should the shoe be checked for improper wear?
A normal sole is worn just laterally to the center of the heel, bisecting the sole and running medially toward the ball and great toe of the foot. Check the heel first to see that it is worn slightly laterally to center, indicating that the heel is supinated at heel strike. Next check the counter, making sure that it is firm and positioned perpendicular to the sole and has not migrated medially or laterally. A medially migrated counter or one that leans inward may indicate increased pronation during gait. Check the stability and flexibility of the sole by grasping the shoe from heel and toe; then twist and bend the shoe. The normal shoe should provide stability through the midfoot and shank area but flexibility at the toe break and forefoot. The front quarters should have a slight crease from the first MTP to the fifth MTP. An oblique crease may indicate a shoe that is too long or a condition such as hallux rigidus. The front quarter also should be perpendicular to the sole without medial or lateral migration. A front quarter that has migrated laterally is also an indication of an increased pronation response because the foot excessively abducts in the transverse plane. Finally, check the arch and midsole to make sure that the arch of the foot is not collapsing over the sole.

37. How can the patient ensure a proper shoe fit?
- Fit a shoe only after you have been active or at the end of the day so that your foot size and shape are typical. This is when the foot is at its largest as well.
- Allow ½ inch between the longest toe and the end of the toe box to ensure proper toe-off without jamming of the toes into the toe box of the shoe.
- The widest part of the shoe should coincide with the widest part of the forefoot.
- The shoe should be snug along the instep; therefore, the dimensions from the heel to the ball of the foot and the shoe instep should be equal.
- The quarter, vamp, and toe box of the shoe should not gap or fold excessively.
- The heel counter should be rigid and should fit snugly around the heel of the foot, limiting excessive heel motion and slippage. Allow space for only 1 finger to slip beside the calcaneus and the heel counter of the shoe.
- When standing in the shoe, perform a pinch test at the fifth metatarsal head. You should be able to slightly pucker the material, indicating that there is adequate width.
- Purchase a shoe that was designed for your foot type and that is immediately comfortable. Do not try to “break in” your shoes; they will probably break you.
- A normal foot lengthens and widens with age so assessing shoe fit routinely is critical. Do not assume it will fit because it is “your size.”

38. What is the leading cause of diabetic foot ulcers? What are the appropriate recommendations for therapeutic footwear?
Most diabetic ulcers result from peripheral neuropathy, which leads to an insensate foot. The insensate foot is unable to recognize increased shear and pressure forces that cause skin breakdown and ulceration. Skin breakdown is most common over the bony prominences of the metatarsal heads, which bear most of the weight during walking. Once the ulcer has healed, therapeutic shoe wear is essential to prevent recurrence. Several research studies have shown that patients who return to normal footwear have a recurrence rate of 90%, whereas those who use modified shoes and orthoses have a recurrence rate of 15% to 20%. Therapeutic footwear should fulfill the following objectives:
- Redistribute and relieve high-pressure areas, such as the metatarsal heads, by using an accommodative total contact orthosis.
- Provide shock absorption by decreasing vertical load forces.
- Reduce shear by decreasing horizontal movement of the foot in the shoe.
- Accommodate deformities such as loss of fatty tissue or ligamentous support.
- Stabilize and support flexible deformities toward a more normal or neutral position while accommodating rigid deformities.
- Reduce painful joint motion or stress.

39. What is a last?
A last is a three-dimensional positive model or mold from which the upper and lower aspects of the shoe are constructed. There are three basic last types: a straight last, a standard last, and an inwardly curved last. The forefoot and rear foot are in neutral alignment with a straight last, whereas a curved last is angled medially at the forefoot. In general, the straighter the last, the greater the stability and control the shoe will have; the curved last is more mobile during the gait cycle.

40. Describe the anatomy and construction of the shoe.
The upper portion of the shoe includes the quarter, counter, vamp, throat, toe box, and top lining. The quarter is a horseshoe-shaped material that cradles the heel of the foot. The counter is a rigid piece of material surrounding the heel posteriorly to stabilize motion. Shoes often include an extended medial heel counter to limit midfoot motion in the overpronator. The vamp is the portion of the shoes that covers the dorsum of the foot to the upper ball of the foot. The throat is the line that connects the proximal portion of the vamp and distal portion of the quarter. The two most common styles are Blucher and Balmoral. The Blucher style is designed for a wider forefoot; the front edges of the quarter are placed on top of the vamp and not sewn together, yielding more room at the throat and instep. In the Balmoral style, the quarter panels are sewn together on the back edge of the vamp. The toe box then covers the end of the toes and refers to the depth of the toe region.

The sole of the shoe includes the outsole, midsole, innersole, and shankpiece. The outsole is the portion of the shoe that contacts the ground. Important outsole properties should include stability, flexibility, durability, and traction. The outsole is made of various materials, depending on the function of the shoe. Outsoles are typically made of leather or a synthetic material. Shankpieces are commonly used in dress and orthopedic shoes to provide rigidity to the midsection of the shoe. The shankpiece helps reduce the twisting or torsion of the forefoot in relation to the rear foot as well as provides support for the midfoot region. The shank refers to the portion of the shoe from the heel to the metatarsal heads. In athletic shoes, the midsole replaces the use of the shank. The midsole is made of various materials, depending on individual needs. For example, the overpronator may benefit from an athletic shoe that uses a dual-density midsole. A dual-density midsole uses a softer durometer material on the lateral side to decrease the lever arm ground reaction forces at heel strike and decrease the rate of pronation. The medial side of the midsole is made of denser or firmer durometer material to decrease the magnitude of pronation. The innersole attaches the upper part of the shoe to the soling and acts as a smooth filler for the foot to rest upon.
41. What are the three basic types of athletic shoe constructions?
The three basic types are board-lasting, slip-lasting, and combination-lasting. In board-lasting the upper shoe is glued to a rigid fiberboard; the board-lasted shoe provides stability and motion control. A slip-lasting shoe is sewn together at the center of the sole, much like a moccasin, and is then cemented to the midsole. It affords little stability but significant flexibility. Finally, a combination-last is used to provide rear-foot stability and forefoot flexibility. The rear portion of the foot is board-lasted and the forefoot is slip-lasted. More recently, a technique called Strobel or Force lasting has become popular. This approach provides for moderate levels of stability and cushion. Generally, a foam or welted material is stitched about the periphery of the shoe, allowing for a very strong connection between the upper and sole of the shoe. Because Strobel or Force lasting is very economical, it is becoming commonplace in athletic and other styles of shoes.

42. What is a rocker sole?
A rocker sole is used to facilitate a heel-to-toe gait pattern while reducing the proportion of internal energy of the foot and ankle for the gait cycle. The toe of the shoe is curved upward to simulate dorsiflexion and allow the metatarsal heads to move through a decreased range of motion at toe-off. Ground reaction forces also are reduced on the ankle because the take-off point is moved posteriorly. In addition, a rocker sole may be used to reduce pressure on specific areas of the foot, such as the heel, midfoot, metatarsals, and toes. Two of the more common types of rocker soles include the forefoot rocker sole and the heel-to-toe rocker sole. A forefoot rocker sole reduces shock at toe-off by placing the apex of the rocker sole just proximal to the metatarsal heads. A forefoot rocker provides stability at midstance but unloads the forefoot at toe-off. A heel-to-toe rocker sole uses a rocker at both the posterior aspect of the heel and just proximal to the metatarsal heads. This type of rocker sole is able to dissipate ground reaction forces at heel strike and increase propulsion at toe-off.

43. What mechanisms can be used to capture a patient’s foot in which to fabricate an orthotic?
There are five basic approaches that one might capture an impression of the patient’s foot. These types include suspension-slipper casting with plaster, foam crush boxes, digital scanning of the foot, tracings of the foot (manually or with a digital photograph), and wax impressions. Wax impressions are, by far, the least common. The gold standard approach is considered to be negative suspension slipper-plaster casting with the foot in a subtalar neutral position. This may be performed in either prone or supine positions with equal results. Foam crush boxes can be utilized effectively, specifically for more rigid foot types. The quality of digital impression is highly variable on the scanner type.

44. What are the proposed mechanisms by which a foot orthotic has a positive effect on pain and function in patients with patellofemoral knee pain?
Some of the most common theories on how foot orthotics decrease knee pain and increase function in patients with patellofemoral knee pain include the following: 1) reduction of lower limb internal rotation; 2) reduction in Q-angle; 3) decrease in laterally directed soft tissue tension forces of the vastus lateralis, iliotibial band, and patellar tendon; and 4) reduction in lateral patellofemoral contact forces.

45. Does the use of a foot orthotic reduce the incidence of lower limb stress reactions in younger, active adults?
It appears the use of a shock-absorbing orthotic can reduce the incidence of lower limb stress reactions, especially in military recruits. The best outcomes seem to be associated with a total contact shell and rear foot and forefoot posting as indicated. As far as over-the-counter products, comfort seems to be the most important variable. The use of a shock-absorbing orthotic as a preventative measure may be a wise choice for those participating in activities that often cause stress reactions of the lower limbs (eg, running, walking—especially in boots).

46. Does the type of prophylactic foot orthosis have any effect on the incidence of lower limb overuse injuries?
The limited research in this area suggests that there is not a significant difference in overuse injury rates based on the type of orthotic used (soft custom, soft prefabricated, semirigid biomechanic, and semirigid prefabricated). Because there is no significant difference between the various types of orthotics, clinical judgment is likely the key deciding factor. Because of methodologic issues within the research and the often small sample sizes, definitive conclusions are unable to be drawn.

47. Are laterally wedged orthotics helpful to patients with medial knee osteoarthritis?
Yes, there appears to be ample evidence to suggest that treating symptomatic medial compartment DJD of the knee with laterally wedged insoles can be effective in reducing pain.
BIBLIOGRAPHY


CHAPTER 78 QUESTIONS

1. Which of the following is true regarding the proper “fitting” of a shoe?
   a. Allow ¾ inch between the longest toe and the end of the toe box.
   b. Allow space for only two fingers to slip beside the calcaneus and the heel counter.
   c. Fit a shoe only after you have been active or at the end of the day.
   d. In standing, pinch the first and fifth MTP together; there should be no puckering of material.

2. After 5 days of wear, a patient complains that she is getting blisters on the sole of her foot and arch pain.
   The most likely cause is:
   a. Orthotic is too thick
   b. Orthotic shell is too flexible
   c. Arch is too low
   d. Medial posting is too high
3. Minimalist shoes:
   a. Decrease injury rates of the posterior tendonitis
   b. **Increase stress to the Achilles tendon and metatarsals**
   c. Mimic barefoot running exactly
   d. Produce a forefoot strike pattern predominantly
ANSWERS TO STUDY QUESTIONS

CHAPTER 1 ANSWERS:
1. C
2. A
3. D

CHAPTER 2 ANSWERS:
No answer

CHAPTER 3 ANSWERS:
1. E
2. B
3. D

CHAPTER 4 ANSWERS:
1. C
2. D
3. D

CHAPTER 5 ANSWERS:
1. A
2. A
3. D

CHAPTER 6 ANSWERS:
1. B
2. B
3. D

CHAPTER 7 ANSWERS:
1. C
2. C
3. D

CHAPTER 8 ANSWERS:
1. A
2. D
3. D
CHAPTER 9 ANSWERS:
1. D
2. A
3. C

CHAPTER 10 ANSWERS:
1. C
2. D
3. C

CHAPTER 11 ANSWERS:
1. D
2. C
3. B

CHAPTER 12 ANSWERS:
1. A
2. D
3. C
4. C

CHAPTER 13 ANSWERS:
1. E
2. D
3. E

CHAPTER 14 ANSWERS:
1. A
2. B
3. B

CHAPTER 15 ANSWERS:
1. C
2. A
3. D
4. A
5. D

CHAPTER 16 ANSWERS:
1. B
2. B
3. D

CHAPTER 17 ANSWERS:
1. C
2. D
3. C. These are all signs of thrombocytopenia
CHAPTER 18 ANSWERS:
1. C
2. D
3. A

CHAPTER 19 ANSWERS:
1. D
2. A
3. A
4. C
5. C

CHAPTER 20 ANSWERS:
1. B
2. A
3. C

CHAPTER 21 ANSWERS:
1. C
2. B
3. D
4. C
5. B

CHAPTER 22 ANSWERS:
1. D
2. B
3. A

CHAPTER 23 ANSWERS:
1. B
2. B
3. D
4. A

CHAPTER 24 ANSWERS:
1. C
2. D
3. A

CHAPTER 25 ANSWERS:
1. C
2. C
3. A
4. D
5. D
CHAPTER 26 ANSWERS:
1. D
2. D
3. C

CHAPTER 27 ANSWERS:
No answer

CHAPTER 28 ANSWERS:
1. B
2. A
3. B

CHAPTER 29 ANSWERS:
1. A
2. D
3. B

CHAPTER 30 ANSWERS:
1. B
2. A
3. D

CHAPTER 31 ANSWERS:
1. B
2. A
3. D

CHAPTER 32 ANSWERS:
1. D
2. B
3. A

CHAPTER 33 ANSWERS:
1. A
2. A
3. B

CHAPTER 34 ANSWERS:
1. B
2. A
3. C
4. D
5. A
CHAPTER 35 ANSWERS:
1. B
2. A
3. D

CHAPTER 36 ANSWERS:
1. A
2. B
3. C

CHAPTER 37 ANSWERS:
No answer

CHAPTER 38 ANSWERS:
No answer

CHAPTER 39 ANSWERS:
1. C
2. A
3. A
4. D
5. C
6. D

CHAPTER 40 ANSWERS:
1. D
2. C
3. D

CHAPTER 41 ANSWERS:
1. A
2. D
3. A

CHAPTER 42 ANSWERS:
1. B
2. B
3. D

CHAPTER 43 ANSWERS:
1. C
2. B
3. D
CHAPTER 44 ANSWERS:
1. C
2. D
3. E

CHAPTER 45 ANSWERS:
1. C
2. B
3. B

CHAPTER 46 ANSWERS:
1. A
2. D
3. B

CHAPTER 47 ANSWERS:
1. C
2. C
3. D

CHAPTER 48 ANSWERS:
1. B
2. C
3. D

CHAPTER 49 ANSWERS:
No answer

CHAPTER 50 ANSWERS:
1. C
2. C
3. D
4. B

CHAPTER 51 ANSWERS:
1. B
2. C
3. D
4. A
5. B

CHAPTER 52 ANSWERS:
1. B
2. D
3. B
CHAPTER 53 ANSWERS:
No answer

CHAPTER 54 ANSWERS:
1. D
2. D
3. D

CHAPTER 55 ANSWERS:
1. A
2. C
3. A

CHAPTER 56 ANSWERS:
1. C
2. B
3. D
4. D
5. A

CHAPTER 57 ANSWERS:
No answer

CHAPTER 58 ANSWERS:
No answer

CHAPTER 59 ANSWERS:
1. C
2. D
3. C

CHAPTER 60 ANSWERS:
1. C
2. B
3. C

CHAPTER 61 ANSWERS:
1. B
2. B
3. C

CHAPTER 62 ANSWERS:
1. A
2. A
3. D
CHAPTER 63 ANSWERS:
1. D
2. B
3. B

CHAPTER 64 ANSWERS:
No answer

CHAPTER 65 ANSWERS:
No answer

CHAPTER 66 ANSWERS:
1. D
2. C
3. B

CHAPTER 67 ANSWERS:
1. A
2. A
3. A

CHAPTER 68 ANSWERS:
1. C
2. C
3. A

CHAPTER 69 ANSWERS:
1. C
2. B
3. B

CHAPTER 70 ANSWERS:
1. C
2. B
3. D

CHAPTER 71 ANSWERS:
1. A
2. D
3. B

CHAPTER 72 ANSWERS:
1. C
2. A
3. B
CHAPTER 73 ANSWERS:
1. C
2. B
3. D

CHAPTER 74 ANSWERS:
1. C
2. C
3. D

CHAPTER 75 ANSWERS:
1. A
2. D
3. A

CHAPTER 76 ANSWERS:
1. B
2. D
3. D
4. B

CHAPTER 77 ANSWERS:
1. D
2. A
3. D
4. C

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1. C
2. D
3. B
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