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Physical Agents Used in the Management of Chronic Pain by Physical Therapists

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The primary role of physical therapy in the treatment of patients suffering from chronic pain is to prescribe, facilitate, and pace therapeutic activities for functional physical restoration [1–4]. Within their sphere of practice, physical therapists have at their disposal and the expertise to administer a wide choice of physical agents (frequently referred to as physical modalities) that may be used to attenuate pain [5–8].

Physical agents may influence pain by resolving inflammation [7,8], facilitating tissue repair [7,8], activating temporary analgesia [8,9], altering nerve conduction [8], providing a counterirritant [8], modifying muscle tone or collagen extensibility [8], reducing the probability of maladaptive central neuropathic changes developing into chronic pain-generation loci [10–12], or otherwise providing palliative relief from pain sensations [6]. In a physical therapy setting, agents are rarely used in isolation; rather, they are used to enhance the effectiveness of other therapeutic interventions directed at functional restoration [4,13].

When prescribing physical agents for the treatment of chronic pain, two essential patient-specific issues must be considered. First, although agents may be useful in a variety of ways for treating chronic pain, they are frequently implemented for temporary attenuation of pain sensations [5,7]. Administering physical agents for passive palliative relief to patients with chronic pain is controversial [4] and should be considered on an individual case basis. For a given patient, providing temporary relief via physical agents may create a therapeutic window of opportunity for the therapist to mobilize tissue or address movement impairments [4]. For others, palliative treatment may psychologically reinforce a maladaptive cycle of pain behavior or generate disincentives for the patient to approach pain

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management in an active or functional manner. This may hinder progress toward functional recovery [4].

The second consideration relates to supposition regarding the locus of pain generation and appropriately matching the physical agent's effects with what affects the pathology or symptoms arising from that pain generation site or process. As an example, an agent such as pulsed ultrasound, whose therapeutic value is to aid in the resolution of inflammation at local tissue, is of little or no value for treating central thalamic pain. In chronic pain cases, the pain generation site may shift over time [11,14–19]. Dorsal horn or cortical neuroplastic changes may result in chronic central pain generation after an inciting distal lesion [11,16,17]. New secondary pain generation sites may also develop from excessively restricted mobility [14,15,18,19]. The original lesion may have resolved, with the current pain complaint now being generated by secondary structural and pathophysiologic changes associated with lack of active motion [14,15]. The applied physical agent must address the specific source of pain generation, neurologically interrupt pain transmission by operating on peripheral nerve conduction or central gating mechanisms, or provide an effective counterirritant. When nociceptive pain is being generated by damaged or inflamed tissue, locally effective agents may be applied. If pain symptoms are largely being generated by neuropathic or central neuroplastic remodeling components, then only agents capable of influencing neural transmission or central processing are likely to be beneficial.

To articulate the strength and quality of evidence supporting the use of physical therapy agents for specific indications, a rating system was developed by Canadian task force groups, proposing a hierarchy of three grades (grades I–III) [7,20,21]. Applications of physical agents with evidence from controlled studies on human volunteers, published in peer-reviewed journals, regardless of level of randomization or blindness, are rated ''grade I''; noncontrolled human studies are ''grade II''; and human case studies are ''grade III'' [7]. This article uses this rating system, as applied by Belanger [7], as a first-order approximation of the quality of evidence for each physical agent genre.

Superficial thermal agents: heat and cold

Thermotherapy

Thermotherapy in rehabilitation is the therapeutic application of superficial mild heat to increase circulation, enhance healing, increase soft tissue extensibility, and control pain. Heat may be delivered to superficial tissues via conduction (eg, hot packs, paraffin dips, microwavable rice-filled cloth bags, electric heating pads), convection (eg, hydrotherapy, fluidotherapy), or radiation (eg, infrared lamps for treating dermal ulcers and psoriasis) [8]. In the context of pain management, potential therapeutic benefits of

superficial heat are due to its effects on metabolic, neuromuscular, and hemodynamic activity.

Although the therapeutic mechanisms attributable to superficial heat primarily influence tissue healing and acute nociceptive pain generation, thermotherapy may have utility in the comprehensive treatment of chronic pain. With mild increases in tissue temperature, the oxygen–hemoglobin dissociation curve shifts to the right, making more oxygen available for tissue repair. Increases in enzymatic activity increase oxygen uptake by the cell, thus enhancing healing [8]. Increased skeletal muscle temperature (to 42°C) has been reported to decrease firing rates of gamma and type II muscle spindle efferents while increasing Golgi tendon organ type Ib fiber firing rates [22–24]. This may reflexively reduce skeletal muscle tone and spasm by lowering alpha motor neuron firing rates [25]. Reducing skeletal muscle activity may be useful in breaking the pain-spasm-pain exacerbation cycle [26].

Superficial heat has been reported to elevate nociceptive threshold [27]. Although it does not travel over large-diameter fibers, the afferent thermoreceptive message of superficial heating has been hypothesized to produce inhibitory modulation of dorsal horn pain gates [8] or to provide a counterirritant stimulus to cortically compete with pain perception. Pain may be significantly influenced indirectly via local vasomotor effects and increased blood flow. Cutaneous thermoreception directly results in the release of bradykinin, leading to local vasodilation in the heated area [28]. After synapsing in the dorsal horn, input from thermal receptors inhibits sympathetic vasomotor efferents in the intermediolateral gray area, thereby decreasing neurogenic vasoconstriction [8]. In addition to the decrease in sympathetic vasomotor outflow, local vasodilation and increased vascular perfusion may influence pain by decreasing tissue ischemia [29], helping to resolve hyperalgesia, thus returning nociceptors to normal firing thresholds and clearing the region of exacerbating metabolites such as prostaglandins. Although increases in blood flow of up to 30 ml per 100 g of tissue have been reported [22], these effects primarily influence cutaneous blood vessels and the tissue regions they supply with less evident vasodilation in deep muscle vasculature due to the minimal ability of superficial agents to carry increased temperature to deep structures [8].

Superficial heat, in the form of hot packs, paraffin, and hydrotherapy, has been broadly evaluated for effectiveness in treating rheumatoid arthritis. Although six controlled studies have found it a beneficial adjunct [30–34], two have found it ineffective [35,36], with the possibility of heat harming the condition through increased collagenase activity damaging compromised articular cartilage [37]. Uncontrolled grade II comparative studies report beneficial effects of superficial heat for chronic low back pain [38–42], neck and shoulder pain [43], and trigger point pain in the neck and back [44].

Contraindications to thermotherapy include applying heat over regions of acute injury, inflammation, hemorrhagic areas, malignancy, impaired

sensation, and thrombophlebitis; hemorrhagic areas; abdomens of pregnant women; or patients manifesting relevant cognitive impairments [7,8]. Precautions should be taken when applying heat over areas with impaired circulation, edema, or superficial metal implants or open wounds; with patients manifesting poor thermal regulation, cardiac insufficiency, or acute inflammatory disorders [37]; or with hypotensive patients or patients prone to syncope when heating large body areas [7,8].

Cryotherapy

In a rehabilitation context, cryotherapy withdraws heat from the body through the use of mild superficial cooling agents. Cryotherapy is used to control pain, edema, and inflammation; to enhance movement; and to attenuate spasticity [8]. The body surface may be exposed to cold though conduction (eg, cold packs, ice massage, cryopressure garments combining cold with compression, bags of frozen corn), convection (eg, cold whirlpool immersion, contrast baths), or evaporation (eg, vapocoolant sprays). The therapeutic effects of cold generally result from its actions on metabolic, neuromuscular, and hemodynamic processes [8].

The application of cold may decrease nociceptive input and pain perception through local and central nervous system mechanisms. In response to cold, the vasoconstrictive response decreases the release of local vasodilating substances, which decreases nociceptor sensitization [26]. Due to metabolic axonal changes, for every 1°C drop in interstitial temperature, nerve conduction velocity of somatosensory afferent fibers drops approximately 2 m/s, with A-delta fibers being the most sensitive to cold-mediated attenuations in velocity [22]. Cold application for 10 to 15 minutes may go beyond immediate changes and produce pain reductions for more than 1 hour [8]. Continued analgesia may be caused by conduction blocking of A-delta nociceptive fibers, inhibitory gating of pain by thermoreceptive fibers, and the maintenance of subnormal deep tissue temperature for 1 to 2 hours after cold exposure [8,45]. Prolonged application of cold has also been demonstrated to produce reversible total nerve conduction blocks [46]. Cold application theoretically interrupts the pain-spasm-pain cycle, reducing muscle spasm and extending pain relief after tissue temperature has recovered to normal values [8]. Finally, by applying vapocoolant sprays over skeletal muscle, so-called ''cryostretch'' is possible [8,47,48]. Immediate analgesia is afforded by evaporative cooling reduces muscle spasm and allows muscle with excess neurogenic tone to be stretched for increased range of motion [48,49].

Although the existing literature strongly supports the efficacy of cryotherapy in the management of acute trauma, cryotherapy may play a role in treating chronic pain conditions. Two uncontrolled comparative studies [47,50] and case studies [51,52] have reported cryotherapy to be a beneficial adjunct in treating muscle spasms and myofascial pain. Comparative

grade II studies found cryotherapy to be a beneficial adjunctive tool in the management of low back pain [41,53], chronic headache [54,55], trigeminal neuralgia [56], and chronic osteoarthritis [57].

Contraindications to cryotherapy include cold urticaria; cold intolerance or hypersensitivity; Raynaud disease or phenomenon; cryoglobulinemia or paroxysmal cold hemoglobinuria; and deep open wounds, regenerating peripheral nerves, areas of circulatory compromise or peripheral vascular disease, and skin areas of impaired somatosensory discrimination [7,8].

Therapeutic ultrasound

In contrast to superficial agents, deep-heating agents are capable of producing temperature elevations at tissue depths of 3 cm or greater through conversion of a nonthermal energy source into heat within tissue [8,58]. One of the most commonly used deep-heating agents is ultrasound, with several authors reporting it to be the most widely used physical agent available to clinicians [7,59,60]. Therapeutic ultrasound is clinically used in three forms: continuous, for raising deep tissue temperature; pulsed, for activating nonthermal physiologic effects; and as a phonophoresis driving agent for transdermal delivery of topical medication [7,8].

Unlike ultrasound used for medical imaging, therapeutic ultrasound is used to deliver energy to deep tissue sites, via propagation of ultrasonic waves, to produce increases in tissue temperature or nonthermal physiologic changes [6,58]. Rather than transmitting ultrasonic waves through tissue and then processing a returning echo to generate an image of underlying structures, therapeutic ultrasound is one-way energy delivery. Via a reverse piezoelectric effect, a crystal sound head transmits acoustic waves typically at 1 or 3 MHz and at amplitude densities of between 0.1 and 3 w/cm^2 [3,8]. Although still comfortably in the ultrasonic range, this is a lower frequency than that used for imaging but is a notably higher wattage.

Ultrasonic energy causes soft tissue molecules to vibrate from exposure to the compression and rarefaction caused by the acoustic wave. Increased molecular motion leads to microfriction between molecules, and frictional heat is generated, thus increasing tissue temperature [7]. In addition to heat generation through microfriction, heat may be generated at specific tissue interfaces due to changes in sonic impedance within the tissue. Different tissue types have varying abilities to attenuate ultrasonic acoustic waves [58]. When passing from tissue of low sonic impedance to one of high impedance (such as from muscle to bone), heat is generated at the interface through shearing and reflection of the wave [22]. This is true at the periosteum, where continuous application of ultrasound can produce periosteal pain due to differential heating [22]. Referred to as ultrasound's ''thermal effects,'' this heating is reported to produce increased collagen extensibility, increased nerve conduction velocity, altered local vascular perfusion, increased

enzymatic activity, altered contractile activity of skeletal muscle, and increased nociceptive threshold [22,29,58,61].

Administering ultrasound discontinuously at a specified duty cycle of on–off pulses produces cavitation and streaming [7,8,58]. The cyclic drop in pressure created by acoustic waves causes normally present minute gas pockets in the tissue to develop into microscopic bubbles, or cavities. With therapeutic ultrasound, stabile acoustic cavitation results, whereby the microbubbles pulsate without imploding. This pulsation leads to microstreaming of fluid around the pulsating bubbles [7,58]. When occurring around cells, this process is reported to alter cell membrane activity, vascular wall permeability, and facilitate soft tissue healing [7,58,63]. Increases in skin and cell membrane permeability from pulsed ultrasound are thought to be partially responsible for the ability of ultrasound to deliver medication to deep tissue sites transdermally.

Pulsed ultrasound has been reported to produce a variety of effects. Some of these are contradictory, such as improved blood flow and increased vasomotor activity [64,65]. Many ultrasound effects may be intensity dependent, with physiologic reversals occurring at different dosing levels [58]. Although usually used for nonthermal effects, pulsed ultrasound produces a concomitant therapeutic effect, meaning that heating and nonthermal effects occur simultaneously [7,66].

Clinical indications for continuous ultrasound relate to the usefulness of deep tissue heating. The heating of collagen increases its extensibility by altering its tertiary molecular bonding. This makes ultrasound a useful aid for therapists treating scar tissue, joint contractures, tissue adhesions, and maladaptive shortening of connective tissue [7,8,58], all of which could be structural contributors to chronic pain [14,15]. Pain reduction via increased nociceptive threshold may be achieved with continuous ultrasound [7,8,22,58]. Proposed mechanisms for increased nociceptive thresholds include counterirritation, heat activation of large diameter afferent fibers, or alteration of nociceptive receptor sensitivity [58]. In numerous studies, varying in rigor, ultrasound has been reported effective in treating pain from a variety of origins including soft tissue lesions [67], muscle spasms [68], tendonitis [69], myofascial trigger points [58], carpal tunnel syndrome [70], back pain [71], epicondylitis [72], complex regional pain syndrome (CRPS) [73], and phantom limb pain [74].

Although the mechanisms remain unclear, pulsed ultrasound has long been used for treating acute and chronic inflammation [75,76] and to promote tissue healing [58]. A further application of pulsed ultrasound for treating pain and inflammation is via phonophoresis. A preparation of a steroid (eg, dexamethasone) or analgesic (eg, lidocaine) is used as the coupling medium between the soundhead and skin surface [8,58]. Pulsed ultrasound transdermally drives the medication deep into tissue by altering the permeability of the stratum corneum and then deep cell membranes [8,77]. Although administered for local tissue effects, drugs delivered through

phonophoresis become systemic, and their systemic contraindications must be considered [8].

Controlled grade I studies have found ultrasound to be useful in the treatment of soft tissue lesions [67], shoulder pain [79], shoulder adhesive capsulitis [80], and pain associated with prolapsed intervertebral discs [71]. For osteoarthritis [81,82], carpal tunnel syndrome [70,83], shoulder calcific tendonitis [69,84], and elbow epicondylitis, grade I investigations are divided into those that report benefits from ultrasound and those that do not. Available grade I studies assessing ultrasound's usefulness for treating postextraction dental pain [85,86], shoulder peritendinitis [87], perineal postlabor pain [88,89], and subacromial bursitis [90] report no significant beneficial effects over controls.

Contraindications to ultrasound include directing acoustic energy over malignant lesions, pregnant abdomens, plastic implants, hemorrhagic regions, cemented areas of prosthetic joints, ischemic regions, insensate areas, infected lesions, electronic implants (including neurostimulators), areas that have been exposed to radiotherapy within the past 6 months, fractures, epiphyseal growth plates in skeletally immature patients, thrombotic areas, orbits of the eyes, gonads, and spinal cord after laminectomy [7,8]. The most common adverse effect is periosteal pain from continuous ultrasound [8], although some authors feel this is the indicator that therapeutic temperature has been reached in deep tissue [22].

Although efficacy evidence for therapeutic application of ultrasound is mixed, ultrasound is widely used by physical therapists as an adjunct to the management of pain and inflammation [3,91,92]. Aside from possible placebo effects, its therapeutic actions are almost exclusively at the tissue level. This makes it a potential tool for nociceptive pain but of limited or no use for central pain or chronic pain exacerbated by neuroplastic remodeling. Prescribing its use for patients with chronic pain should result from reasonable evidence that the pain is, at least in part, generated by an active lesion at the nociceptive level.

Diathermy

Diathermy is the use of shortwave (wavelength 3–30 m, frequency 10–100 MHz) or microwave (wavelength 0.001–1 m, frequency 300 MHz to 300 GHz) electromagnetic radiation to produce heat within body tissue through conversion [8]. The United States Federal Communications Commission has assigned 13.56, 27.12, and 40.68 MHz for medical applications of shortwave and 2450 MHz for microwave medical applications [7,8]. Shortwave diathermy (SWD) is typically generated using the 27.12-MHz band [7,8].

Diathermy has potential advantages over other agents used to heat subcutaneous tissue. First, diathermy can produce heat at deeper tissue levels than superficial agents [8]. Second, it can heat larger areas than other

penetrating agents (eg, ultrasound) [8]. Third, shortwave radiation does not experience a transmission impedance change while passing from soft tissue to bone, as does sound energy. Therefore, unlike ultrasound, it is not reflected by bone and does not cause differential heating at tissue interfaces or present risk of periosteal burning [8].

Microwave diathermy (MWD) has two disadvantages limiting its potential use. Unlike SWD, MWD reflects when encountering even slight variations in soft tissue density, thus producing shearing, standing waves, and local hot spots in relatively superficial tissue [8]. The high frequency of MWD, combined with its high reflectivity at tissue interfaces, means that MWD tends to bring superficial tissues to intolerably high temperatures before therapeutically useful temperature increases are achieved at the deeper target tissue levels. For this reason, the clinical use of MWD has been nearly abandoned in most countries in favor of SWD and ultrasound [7].

Shortwave energy can be delivered as continuous electromagnetic radiation (continuous shortwave diathermy [CSWD]) for deep heating of soft tissue or in pulsed form (pulsed shortwave diathermy [PSWD]) to induce nonthermal effects [7]. As electromagnetic energy is delivered to the tissue via CSWD, increased average molecular kinetic energy leads physiologically to thermal heating effects of vasodilation, increased rate of nerve conduction, increased collagen extensibility, acceleration of enzymatic activity, changes in skeletal muscle strength, and possibly increased nociceptive threshold [8]. In contrast with superficial heating, which produces physiologic heating effects within a few millimeters of the dermis, CSWD may be used to produce these effects within deep muscle [8].

By pulsing the delivery of shortwave energy with low amplitude, short-duration pulses at a low-duty-cycle SWD do not generate sustained increases in tissue temperature due to dissipation of transient heat from vascular perfusion of the area [8]. However, as with ultrasound, when pulsed energy is applied at subthermal levels, a number of nonthermal changes occur [93]. Although the mechanisms of nonthermal effects are speculative, they are broadly attributed to modified ion binding, which affects cellular functions of protein synthesis and ATP production [7,94–96]. The influence of electromagnetic fields on ion binding has been reported to produce a cascade of physiologic responses that may include growth factor activation in fibroblasts and neurons, macrophage activation, and alterations in myosin phosphorylation [8,97].

There is evidence that PSWD application for 40 to 45 minutes increases microvascular perfusion of local tissue in normal subjects and adjacent to ulcer sites in patients with diabetic ulcers [8,98,99]. Increased local perfusion has the capability to increase oxygenation of deep tissue, decrease anaerobic metabolism, enhance nutrient availability, and assist phagocytosis [8]. Although it is most probable that CSWD and PSWD produce thermal and nonthermal effects, a result of either mode of application is increased cellular metabolism and functioning, which may have implications for the promotion of healing [7].

Due to its ability to heat large areas of deep tissue, potential indications for the clinical use of CSWD include augmentation of healing, decreased joint stiffness in large areas such as the hip or diffuse spinal regions, and increased joint range of motion when combined with stretching [8]. Possible clinical indications for the use of nonthermal PSWD include pain control via edema reduction and enhanced healing of soft tissue wounds (eg, burns, pressure ulcers, and surgical wounds), peripheral nerve lesions, and fractures [8]. The possible clinical benefit of SWD to beneficially address these conditions must be considered not only on its demonstrated effects but also on the strength of clinical efficacy evidence.

Most of the recent literature on clinical efficacy of SWD evaluates potential tissue healing effects, with a few available studies addressing SWD application in chronic pain management. Pulsed electromagnetic fields (PEMF) have been reported to be a useful therapy for nonunion fractures [100,101], failed arthrodeses [102], and osteonecrosis [103]. Another form of PEMF, magnetotherapy, has been applied to treat chronic pain of various origins [104–106], venous ulcers [107,108], and tendonitis [109,110]. Although both are pulsed delivery of electromagnetic radiation, PEMF and PSWD are not synonymous [7]. Two studies found significant decreases in neck pain and increases in range of motion in patients who had cervical spine injuries after using PSWD for 3 weeks compared with a placebo device [111,112]. Two early (1959 and 1964) grade I controlled studies [113,114] reported beneficial results treating osteoarthritis with SWD, whereas three more recent investigations did not find SWD therapy to produce significant reductions in osteoarthritic pain intensity over control subjects [115–117]. Three controlled investigations failed to demonstrate significant benefits of SWD in treating ankle sprains [118–120]. A single uncontrolled study reported positive outcomes using PSWD treating post-traumatic algoneurodystrophy (CRPS) [121]. A single available controlled study found SWD beneficial for managing low back pain [122].

The nature of the radiant energy that allows SWD to increase tissue temperature gives rise to special precautions and contraindications. Some materials absorb disproportionate amounts of electromagnetic energy, such as metals, fat, and tissue with high free water concentrations [8]. Other materials (eg, drops of perspiration) act as lenses, focusing the energy. High absorption and focusing may lead to hazardous increases in adjacent tissue temperature [7,8]. Burning or fire could be caused by the presence of metal implants, pacemakers, neurostimulators, or copper-bearing intrauterine contraceptive devices within the body or any metal outside the patient's body (eg, jewelry, coins, clothing zippers) or in close proximity to the patient within the shortwave radiant field (eg, metal parts in a treatment table, zippers in pillow cases) [7]. The immediate environment must be cleared of metal and electronic or magnetic equipment. "Well, the first time I lit a patient on fire with diathermy…" began a therapist's anecdote relating how she had forgotten about the metal zipper of the inner pillowcase beneath the patient's head.

Special precautions must be taken when treating obese patients, when treating high adipose regions, or under circumstances when the patient begins perspiring [8] and over moist wound dressings or ischemic areas [7]. Because of variation in absorbency, some tissue areas may be burned while others are spared [8]. The patient's skin must be kept dry during treatment to prevent scalding from hot perspiration [8].

Contraindications to SWD include pregnancy, malignancy, and applying SWD over insensate skin regions. Because of potential damage due to heat generation, CSWD should not be applied over the eyes, testes, or epiphyseal growth plates in skeletally immature patients.

Although SWD has good tissue penetration properties and the ability to heat or deliver pulsed energy to deep structures, it is rarely used in the treatment of pain. This is because of the many ways that patients can be harmed via soft tissue burns. Using a pharmacologic analogy, its therapeutic index is uncomfortably low compared with other physical agents. Most clinics have abandoned its use, and it is a rare physical therapy facility that has the equipment available and in use with therapists adequately trained and experienced in its application. Recent promotion of the clinical use of SWD is for wound-healing applications.

Laser

Laser therapy uses light that is monochromatic, coherent, and highly directional [8]. Proposed uses for laser therapy in physical rehabilitation settings include the promotion of wound healing and pain management [7,8]. Although laser therapy has been widely used in Europe for more than a decade [123], it was not until February of 2002 that the US Food and Drug Administration approved the therapeutic use of laser therapy for the temporary relief of pain.

Special properties of laser light allow the potential for direct delivery of electromagnetic light energy to tissue depths slightly below the dermis and possible indirect physiologic effects at deeper levels [8,124]. The ability of laser light to penetrate is a function of tissue type and the laser's wavelength and resistance to scatter [125]. The most commonly used wavelengths for clinical application of laser light range from 600 to 1300 nm, allowing a direct tissue penetration depth of 1 to 4 mm [8]. A second variable parameter of laser light affecting its clinical use is power or wattage [7,8]. Cold lasers with output powers of less than 500 mW, at a power density of about 50 mW/cm^2, have been used in rehabilitation settings to theoretically promote healing and manage pain via photobiomodulation of chromophores within the affected tissue. The term ''low-level laser therapy'' (LLLT) is used to describe the therapeutic application of cold lasers to facilitate photobiomodulation [7,125,126]. The most frequently used lasers for LLLT are semiconductor diode types (904-nm gallium-arsenide lasers or gallium-aluminum

arsenide lasers) with wavelengths that may vary based on aluminum content [7].

Although the physiologic effects of low-wattage lasers are not well established or understood, there is consensus in the literature that LLLT can induce photobiomodulation effects [7,126,127]. As laser light penetrates the skin, its photons are absorbed by cellular chromophores (light-absorbing molecules) that undergo photobiomodulation via influence over respiratory chain enzymes in the form of photobiostimulation or photobioinhibition according to the Arndt-Schultz law of photobiologic activation [7,125]. This asserts a dose-response interaction effect whereby low dosages trigger a photobiostimulation response and higher dosages trigger a photobioinhibition response [125]. Wound-healing effects are attributed to photobiostimulation, whereas pain management has been reported to be a function of photobioinhibition [7]. Photobiomodulation effects via cold laser on calcium channels have been reported to cause increased fibroblast, macrophage, and lymphocyte activity [128–132].

For temporary analgesia, the effect of LLLT on nerve conduction velocity has been addressed by numerous grade I controlled studies. Some have shown small increases or decreases in peripheral nerve conduction velocity with corresponding slight changes in distal latencies [133–136], whereas others report finding no effect [137,138]. The ability of LLLT to influence nerve conduction velocity in a clinically significant way seems uncertain at this time. That is not to say that LLLT modulation of pain from peripheral nerve involvement could not be influenced via another, as yet uncertain, mechanism.

The two primary indications for LLLT are wound healing and pain management. Efficacy studies related to both applications yield varied results. Of 17 English language studies reviewing the clinical impact of LLLT on cutaneous wounds and ulcers, 14 have demonstrated beneficial outcomes. Of three grade I controlled studies on wound healing, two conducted before 1992 using He-Ne lasers [139,140] reported no benefit over control subjects, whereas a more recent work published in 1999 [141] reported a beneficial effect, citing the importance of appropriate candidate selection for LLLT.

The effect of LLLT has been addressed in numerous studies of varying quality for a wide spectrum of conditions that generate pain. For many disorders, outcomes of controlled studies are decidedly split between those that show some clinically significant beneficial effect over control subjects and those that do not. Regarding arthritic conditions, four [142–145] of seven [142–148] controlled studies reported beneficial results for patients with rheumatoid arthritis, and five [149–153] of seven [149–155] studies yielded positive therapeutic responses for osteoarthritic conditions. Two controlled studies addressing the ability of LLLT to relieve pain secondary to trigger point stimulation reported beneficial results [156,157], whereas the treatment of myofascial pain per se displays a different clinical picture, with three studies [158–160] reporting no significant effect over controls. Three [161–163] of

four [161–164] studies assessing the impact on teninopathies report no beneficial findings, with regional epicondylitis showing a positive response to therapy in one [165] of five [165–169] grade I controlled studies.

Regarding pain originating from a specific locus, controlled studies reporting LLLT benefits have been published regarding trigeminal pain [170,171], postherpetic pain [172], perioral herpes pain [173], and postsurgical abdominal pain [174]. Laser therapy has not demonstrated significant benefits in available grade I studies concerned with ankle pain [175], temporomandibular joint disorder [176], muscle soreness [177,178], plantar fasciitis [179], chondromalacia [180], or orofacial pain [181].

The foremost contraindication to the use of LLLT is exposure of the eye to laser light. Additional contraindications include exposing any of the following regions to low-level laser light: locally to endocrine glands [8]; photosensitive skin areas; hemorrhagic areas; any area within 4 to 6 months after radiation therapy; neoplastic lesions; or over the heart, vagus nerve, or sympathetic innervation routes to the heart of cardiac patients [7,8]. Precautionary application should be considered when using LLLT over epiphyseal regions of long bones in children, gonads, infected areas, or areas with compromised somatosensation and when treating patients who display mental confusion, fever, or epilepsy [7,8]. Although there are few reports of adverse responses to LLLT, episodic tingling, burning sensations, mild erythema, numbness, increased pain, and skin rash associated with LLLT have been reported in individual cases [8].

The available literature shows a mixed picture of efficacy findings regarding the therapeutic effects of LLLT for various pain conditions. Proposed mechanisms have plausibility, yet they are incompletely understood. The use of LLLT is increasing in North America, and recent approval by the US Food and Drug Administration may accelerate its clinical implementation for the temporary reduction of pain. It is not in widespread use by physical therapists, and, although some clinics are providing this service, most do not have the apparatus or training to offer it.

Electrical current

Traditionally, the use of electrical currents to modulate pain is via transcutaneous electroneural stimulation (TENS). Unlike physical agents, whose primary site of action in pain control is the tissue level, TENS is thought to operate by facilitating interruption of the neural transmission of pain [9]. Using capacitance coupling, surface electrical current produced by the TENS unit generates action potentials in underlying peripheral nerves. Specific axons affected are determined by three interacting factors: fiber diameter, anatomic proximity of nerve fibers to the skin surface, and external current intensity [8]. There are choices for the placement of stimulating electrodes: around or near the lesion site, along the course of the peripheral

nerve carrying the nociceptive message, on the back near spinal nerve root entry, or at related acupuncture points [8]. Four levels of stimulus intensity may be delivered by TENS units: subsensory, sensory, motor, and noxious.

Subsensory-level TENS uses a phasic charge of insufficient amplitude to depolarize peripheral nerve axons, reach sensory threshold, or depolarize muscle membranes [9]. This approach is sometimes referred to as subliminal stimulation [182], low-intensity direct current [5], or microcurrent electrical nerve stimulation (MENS) [8,9,183]. In the absence of neural stimulation, it is uncertain which mechanism microcurrents use to modulate pain. Postulated mechanisms include placebo effects, augmented tissue healing, and altering energy flow along acupuncture meridians [8]. Two authors have stated that there is no evidence for the use of subsensory-level electrical currents in pain management [8,9]. Several studies have found MENS to be no more effective than no treatment or placebos and significantly less effective than sensory-level TENS [183–189].

Although subsensory microcurrent does not operate by exciting peripheral nerves, it may enhance tissue healing [7]. Numerous studies have reported microcurrents being generated by the skin in areas around wounds [189–192]. These naturally occurring microcurrents, called "currents of injury" [189], have been observed in the skin of regenerating newt stumps [193,194]. It has been hypothesized that exogenous microcurrent may augment this endogenous activity and enhance or maintain skin healing [7]. Grade I investigations have found microcurrent efficacious in augmenting healing for epicondylitis [195], peritendinitis [196], and indolent and diabetic ulcers [197,198]. Two controlled studies found microcurrent to be of no benefit in the treatment of delayed-onset muscle soreness [199,200]. Mixed results have been reported in controlled studies for the treatment of pressure ulcers [201,202]. However, in reference to pressure ulcers, the United States Agency for Health Care Policy and Research concluded in 1994 that "At this time, electrical stimulation is the only adjunctive therapy with sufficient supporting evidence to warrant recommendation" [7]. From the perspective of pain management, microcurrent application may assist wound resolution but seems to be of no value in attenuating pain that is not associated with an active nociceptive lesion.

Operating at higher current amplitude than microcurrent, sensory-level (or "conventional") TENS is thought to attenuate the perception of pain via stimulation of large-diameter afferent peripheral nerve fibers and subsequent interruption of pain transmission at the dorsal horn due to the gate control mechanism [8,9,203]. Because it primarily operates neurally through the ascending analgesia pathway, sensory-level TENS produces a rapid onset of pain reduction, yet its effects typically cease quickly after stimulation has stopped [8]. Sensory-level TENS units are often worn for many hours during the day and use frequent random modulation of the stimulus wave to prevent neural habituation. Some studies have suggested that this level of stimulation may trigger limited endorphin release in instances where its effects seem to outlast the period of electrical stimulation [204,205].

Sensory-level TENS is primarily indicated for acute and subacute pain but also has utility in chronic pain conditions. One suggested chronic pain application is to reduce pain as early as possible in the development of the condition to fight dorsal horn remodeling of N-methyl-D-asparate (NMDA) receptors as a central pain generation site [11]. It may also be plausible to use sensory-level stimulation at a body site other than the painful region to provide a competing attentional counterirritant to fight long-term cortical remodeling.

To achieve a more prolonged analgesic response from TENS, current amplitudes may be increased to induce motor- or noxious-level stimulation, which activates the descending endogenous-opioid–based analgesic pathway [7–9,206,207]. Motor-level stimulation occurs when TENS amplitude is high enough to produce visible skeletal muscle contractions [8,9]. Rhythmic muscle contractions may be induced electrically without exciting nociceptive afferent fibers [9]. These contractions have been shown to stimulate therelease of enkephalins and dynorphins [208]. Analgesic responses to motor level TENS have a slower onset (15–60 minutes) but have longer duration after stimulation is discontinued (several hours) than sensory TENS [8].

Noxious-level TENS helps reduce pain perception by stimulating nociception at a site near or remote to the painful region. Current amplitudes are great enough to produce painful stimulation with or without skeletal muscle contraction [9]. Pain relief onset occurs within seconds or minutes after initiating the stimulus and may last for several hours [8]. Studies have demonstrated that noxious-level–induced decreases in pain last longer and are more pronounced than the relief generated from sensory- or motor-level TENS [206,207,209–211]. It is hypothesized that noxious-level stimulation may cause rapid pain modulation via ''hyperstimulation analgesia,'' which interferes with central patterned-reverberation pain circuitry [9].

Although sensory-level TENS is the most widely used modality, due to patient intolerance for rhythmic muscle contractions and painful stimuli presented to a person already experiencing pain, motor- and noxious-level applications may be indicated if insufficient relief has been achieved with sensory-level stimulation [8]. Motor-level TENS is recommended for patients who have chronic pain and low endogenous endorphin levels (eg, from prolonged opiate use) [8]. Noxious-level TENS may be indicated for patients who have chronic pain and have not had a successful response to motor-level TENS [8].

Clinical efficacy literature related to chronic pain applications of TENS is extensive and has yielded relatively consistent findings. Six controlled studies have demonstrated significant clinical effectiveness for TENS in the management of pain associated with osteoarthritis [212–217]. Other conditions for which TENS has demonstrated effectiveness in grade I studies include trigeminal neuralgia [218], postamputation and phantom limb pain [219,220], neck pain [221], pain due to peripheral neuropathy [222,223], painful shoulder secondary to stroke [224], and migraine headache [225]. Mixed results have been reported for rheumatoid arthritis [226–228], low

back pain [229–235], and myofascial pain [236,237]. Conditions for which the literature consistently shows TENS to be of no benefit over control subjects include a limited number of acute and post-surgical pain conditions [7].

In addition to TENS, electrical currents are used to enhance healing, resolve inflammation, and transdermally deliver topical medication [8,238]. Interferential current and iontophoresis are of potential utility in managing chronic pain.

Interferential current (IC) involves intersecting two alternating current sources of slightly different middle frequencies to create an interference pattern at a target tissue site. The resulting IC is in the form of a low-frequency "beat," whose frequency is the arithmetic difference between the two intersecting currents, typically in the 1 to 200 Hz range [8]. Suggested indications are for pain modulation via inhibitory gating at the dorsal horn and edema management.

Efficacy literature supporting the use of IC is lacking. Evidence for its use is largely based on clinical anecdotes and unsupported beliefs [239]. Regarding analgesia, one study reported that healthy subjects receiving IC showed significantly increased thresholds for "ice-pain" compared with control subjects not receiving IC [240]. Conversely, IC failed to show any effect on pain when using the RIII reflex as an experimental pain model [241]. Although there is a case report indicating successful treatment of a patient who had migraine headache using IC [242], grade I controlled studies applying IC to the treatment of acute low back pain [243] and jaw pain [244] have failed to demonstrate beneficial results. No benefit for low back pain was observed for IC in an investigation comparing it with motorized lumbar traction and massage [245]. In spite of the paucity of supporting evidence, IC is widely used in physical therapy clinics.

Similar in function to phonophoresis, iontophoresis uses direct current to assist the local transdermal delivery of ionizable medications, such as local anesthetics and antiinflammatories [238]. Positively charged ionic compounds are repelled from anode electrodes and attracted to cathodes, whereas negatively charged compounds manifest the opposite behavior [246]. For managing chronic pain conditions, several medications have been recommended that are capable of forming ionic compounds in solution: lidocaine for soft tissue pain and inflammation, dexamethasone and hydrocortisone for inflammation, magnesium sulfate for skeletal muscle spasms, and salicylates for acute and chronic muscle and joint pain [238]. Because iontophoresis relies upon direct current, it is important to note that a sodium hydroxide alkaline reaction naturally occurs beneath the cathode electrode and hydrochloric acid concentrates beneath the anode [238]. With excessive use, electrochemical skin burns may occur beneath the electrodes due to these pH changes. Although changes in tissue pH beneath the electrodes may affect drug ionization and stability, there is evidence that iontophoresis can effectively deliver some medications to the site of interest [247–249].

Few studies have investigated the use of iontophoresis for managing chronic pain. It has been found to be effective in a grade I study using dexamethasone to manage plantar fasciitis [247], beneficial using a combination of dexamethasone and lidocaine to treat shoulder myofascial syndrome in a grade II comparative study [248], and effective as an adjunct to managing post-herpetic neuralgia pain in an uncontrolled follow-up investigation [249].

There are other electrical current configurations with reported use in managing chronic pain whose application and availability in the United States is limited. Three of these are multiplexed anodal stimulation, high-voltage pulsed current [7,250,251], and diadynamic current [7,252,253].

Contraindications to the use of electrical stimulation include applying current over the anterior cervical region, carotid sinuses, heart, transthoracic area, insensate skin, and the abdomen of a pregnant woman; in conjunction with a cardiac pacemaker, implanted defibrillator, or any other implanted electrical device; during ECG testing or while operating diathermy devices; and for patients with venous or arterial thrombosis or thrombophlebitis [7,8].

Precautions should be taken delivering electrical stimulation over tissues susceptible to hemorrhage or hematoma; on craniofacial regions for patients with a history of cerebrovascular accidents or seizures; on patients who have movement control disorders, impaired cognition, malignancies, osteoporosis (motor-level TENS), or cardiopathies; and on patients while driving or operating heavy machinery [7,8]. Iontophoresis is specifically contraindicated for use over open skin lesions and for patients with known sensitivity to the therapeutic ions [7]. Precautions should be taken to prevent skin damage due to adhesive irritants and electrochemical pH changes under the electrodes.

Supporting evidence is strong for the use of TENS as adjunctive therapy for treating many pain conditions. Most physical therapy clinics are equipped to administer interferential current and iontophoresis (provided the patient brings to the clinic the ionic medication preparation prescribed by the referring physician), to conduct a TENS trial, and to arrange for the acquisition of a home TENS unit. A TENS trial frequently requires some time and experimentation to determine an effective electrode placement site.

Desensitization

In contrast to the normal hyperalgesic response of body tissue to acute injury, allodynia is a painful response to a non-noxious somatosensory stimulus such that the affected individual may guard the limb from even the most delicate tactile contact, even refraining from wearing clothing over the painful site [12,254,255]. It is one of the hallmark symptoms of CRPS, with 74% of patients reportedly experiencing allodynia [256]. For physical and occupational therapists, the treatment of allodynia via desensitization

is an essential component in helping to restore functional use of the affected body part [257]. A number of authors cite desensitization training as one of the essential core therapeutic elements in the physical or occupational therapists' management of CRPS [4,257–260]. Using this technique, the therapist may directly treat pain symptoms that are restricting function [259].

Somatosensory desensitization therapy for allodynia generally involves having the patient rub the affected body region over time with a series of progressively coarser and more irritating tactile stimuli [257]. A complete treatment protocol may span 10 to 15 weeks, including home practice and at least weekly in-clinic rechecks and progressions [255,260].

Although the operating mechanism of desensitization has yet to be established or may be multidimensional, several plausible theories are offered. For a person experiencing allodynia, restricting or avoiding tactile contact to the painful area has become a way of life [4]. By reintroducing tactile stimulation, the person may rehabituate to formerly irritating somatosensory input [261]. Repeated exposure to progressively irritating materials may reset altered central processing of somatosensory input at the dorsal horn or cortically [257,261] or may prevent the development of permanent pain pathways in the central nervous system by manipulation of cortical centers responsible for pain perception [262]. Reintroducing normal tactile input may restore large-fiber–diameter afferent inhibition of pain, which had been eliminated through restricted normal tactile contact [257]. As a goal of desensitization, the patient may begin normalizing exposure of the effective body area to the distal environment [263]. This helps reestablish the benefits of ascending analgesia from large-diameter somatosensory fibers and, with guidance from the therapist, aids in reintroduction of the limb or body area into functional usage. Enhanced usage may create a positive spiral of analgesia and activity, thus turning normal activity into a continuation of the desensitization therapy [12,257].

Although clinical use of desensitization is common and considered part of standard care when working with patients manifesting allodynia [259] and individual patients are reported to manifest notable functional usage gains after its implementation [252–260], efficacy evidence supporting its use is sparse [264] and is predominantly limited to case studies of grade III with an absence of available controlled studies.

The earliest reports of using desensitization therapy come from prophylactic intervention against postamputation phantom limb pain [260,263,264]. An early reported use of treating chronic allodynia used the chemical irritant capsaicin as the desensitizing agent in the treatment of CRPS [265]. Multiple reports have indicated that patients desensitized to light touch or never manifesting light touch allodynia may experience painful responses to non-noxious levels of thermal variation, pressure, or vibration [10,62,255,257,266]. There are case reports of treatment success in managing thermal-[62] and pressure-related allodynia when the desensitizing agents were matched to the specific somatosensory modality producing the painful

response, resulting in functional improvements along with reductions in pain intensity and pain medication usage [255,266]. This suggests that desensitization therapy may be somatosensory specific and that desensitizing agents should be chosen to represent the particular problematic sensory stimulus type that is triggering the allodynia [255,257,266].

Desensitization therapy may be indicated for conditions involving somatosensory allodynia. The clinician should consider the scarcity of supporting evidence and evaluate individual patient response. Its application is contraindicated when working with any painful skin field where there is an active lesion that may be physically harmed by exposure to somatosensory irritating agents.

Clinical implications: application of physical agents to prototypical cases

Physical therapy treatment approaches for the following prototypical cases may vary considerably, based on a therapist's treatment philosophy and the patient's functional goals. However, the following cases represent examples of how physical agents might be used in each case.

Case 1: Chronic right lower extremity pain secondary to closed head trauma

Because the patient's right lower extremity pain is not primarily nociceptive, a TENS trial is indicated. Placement of stimulating electrodes could be near the painful region, on the contralateral limb, or over the spinal nerve root. Given that the primary pain generator is most likely rostral to any possible stimulation site, TENS would serve as a potential counterirritant. With secondary pain aggravation due to spasticity, cryotherapy is indicated to ease the spasticity in the form of cold packs or vapocoolant sprays. A prolonged consequence of diminished movement and spasticity is contracture, which could create secondary structural pain-generation sites. This may be addressed with ultrasound, to facilitate collagen extensibility, combined with stretching. The patient's diminished cognitive status requires assessment to determine if comprehension and communication are adequate for the safe use of these agents and their potential for home implementation.

Case 2: Chronic bilateral distal lower extremity pain secondary to diabetic polyneuropathy

To treat bilateral distal lower extremity pain due to diabetic polyneuropathy, TENS is indicated for analgesia. Specific electrode placement sites need to be explored for effectiveness and convenience. If TENS analgesia is effective to help her increase activity, she decreases the possibility of developing new pain-generation sites secondary to inactivity. The patient's history of hypertension and myocardial infarction does not present elements that would contraindicate TENS.

Case 3: Chronic neck and back pain/possible fibromyalgia syndrome

It seems plausible that the spread of pain for this patient is due to diminished movement and muscle guarding after the initial episode of posterior neck pain. Myofascial release and treating the trigger points of the upper trapezius and levator scapulae muscles is beneficial in this case. This may be addressed in a variety of ways. One approach would be cryostretch of hypertonic, shortened muscles via vapocoolant sprays. The trigger points may also respond to superficial heat. Laser therapy may be helpful with trigger points, but the literature is divided over such treatment. A consideration with laser application is that LLLT penetrates 1 to 4 mm below the skin surface, which may not be deep enough to affect the trigger points in question. Patient response to each of these options should be assessed to determine the most effective option or combination of agents. TENS may also be applied to provide analgesia that might allow increased neck, back, and shoulder girdle mobility. Gradually increasing motion may break the exacerbation cycle spreading the pain and is consistent with therapeutic approaches for patients manifesting the tender points and restricted movements associated with fibromyalgia syndrome.

Case 4: Chronic low back pain without radicular symptoms

This patient may represent a case where physical agents are not indicated. With no specific pain generator identified, agents operating at the tissue level are not likely to be beneficial. A TENS trial might be useful, but careful attention should be paid to the psychobehavioral impact of a passive analgesia approach. Although the patient's interventional history includes extensive physical therapy, it may be appropriate to ascertain what specific treatment approaches were used. The development of deactivation pain is a concern, so the patient may be a likely candidate for reactivation therapy, with careful attention to physical activity dosing and pacing.

Summary

Evidence supporting the use of specific physical agents in the management of chronic pain conditions is not definitive; it is largely incomplete and sometimes contradictory. However, the use of agents in chronic pain management programs is common [78]. Within the broad use of physical agents, they are rarely the sole modality of treatment. A 1995 American Physical Therapy Association position statement asserts that "Without documentation which justifies the necessity of the exclusive use of physical agents/modalities, the use of physical agents/modalities, in the absence of other skilled therapeutic or educational intervention, should not be considered physical therapy" [13]. Physical agents may serve as useful adjunctive modalities of pain relief or to enhance the effectiveness of other elements

in therapy geared toward resolution of movement impairments and restoration of physical function.

Given that a conclusive aggregate of findings is unlikely to exist for all permutations of patient conditions, combined with interacting therapeutic modalities, an evidence-based approach to pain management is not always possible or beneficial to the patient. In the face of inconclusive evidence, a theory-based approach may help determine if the therapeutic effect of a given physical agent has the possibility of being a useful clinical tool in the context of treating a particular patient's mechanism of pain generation. Until controlled efficacy findings are definitive, careful individual patient response monitoring of thoughtful theoretical application of adjunctive physical agents may be a prudent approach to the management of chronic pain.

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