TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION (TENS)

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INTRODUCTION

The field of pain management is a rapidly expanding one, and new treatment modalities are being discovered or rediscovered. Electroanalgesia has a long and sometimes dubious history, dating back to the ancient Egyptians. However, the publishing of "the gate control theory" of pain transmission in 1965 by Melzack and Wall transformed our understanding of pain, its transmission, and how it is modulated. With this discovery, electroanalgesia underwent a revolution, and transcutaneous electrical nerve stimulation (TENS) was developed and is continually being refined. Today, our understanding of the mechanism by which TENS produces analgesia continues to expand does its potential applications.

This article provides a review of pain, its definition, types, and physiology. It provides background information and theories surrounding the mechanism of analgesic action of TENS and the development of electroanalgesia. It discusses the usage, design, applications, and warnings surrounding TENS.

WHAT IS PAIN?

Pain is an unpleasant sensory and emotional experience associated with actual or potential damage, or described in terms of such damage. Pain serves as an essential defense mechanism to protect one's body from potential damage. Indeed, the disastrous consequences of diminished or absent pain signaling become readily apparent in diseases and conditions that result in partial or complete damage of the nerves that innervate the extremities (e.g., diabetic neuropathy, tables dorsalis, tuberculoid leprosy, and many others). While serving an essential function, pain can often present for physiologically inappropriate reasons, continue far past the removal of noxious stimuli, remain long after wound healing, or even present for purely psychological reasons. This maladaptive and uncontrolled pain cycle afflicts an estimated 40 million Americans (1), and research into the causes and cures of pain is a rapidly expanding branch of medical science and forms the basis for a multibillion dollar a year, multidisciplinary industry.

Pain can be categorized either temporally as in acute or chronic pain or by the mechanism. Nocioceptive–inflammatory pain is produced after an appropriately perceived tissue injury. Neuropathic pain, however, is produced by nerve injury that is inappropriately perceived due to neuroplasticity. Often described as a burning or electric sensation, neuropathic pain can persist long after an injury or for completely idiopathic reasons. Even simple light touch or changes in temperature are enough to trigger severe bouts of extreme pain, lasting seconds to hours or longer (i.e., trigeminal neuralgia).

Phantom limb pain is another incompletely understood neuropathic phenomenon and occurs in 50–67% of postsurgical amputation patients (2). It is often described as a minor-to-severe cramping or, less commonly, as a burning sensation (3). While this commonly subsides with time, in $\sim$10% of patients, this pain persists and is often refractory to NSAID or opiate therapy, traditional first and second line agents in the treatment of pain.

THE PHYSIOLOGY OF PAIN

The process of nocioception is complicated, but can be divided into four distinct physiological processes transduction, transmission, modulation, and perception. Transduction, the translation of noxious stimuli into electrical activity at the sensory endings of nerves, occurs at unspecialized mechano-, thermo-, or polymodal (thermal and chemical) nociceptors, as well as at unspecialized nerve endings.

Polymodal nociceptors respond to a variety (i.e., chemical, mechanical, and temperature extremes) of intense noxious stimuli. Thermonociceptors are distinct from thermoreceptors that transmit non-noxious temperature information. This class of nocioreceptors functions from temperature ranges of roughly <5 to >45 $^{\circ}$C. Mechanonociceptors are activated when intense pressure stimulates them; as with thermonociceptors, the mechanonociceptors are distinct from the receptors that transmit non-noxious light and strong touch, vibratory information, and so on. Additionally, visceral "silent" nociceptors exist in a default dormant state and are usually activated only in the presence of inflammatory mediators. These silent nociceptors likely are involved in hyperalgesia as discussed below.

In the peripheral nervous system, small unmyelinated C polymodal nocioceptive fibers, as well as the larger,

Encyclopedia of Medical Devices and Instrumentation, Second Edition, edited by John G. Webster

lightly myelinated Aδ mechano- and thermonocioceptive fibers transmit noxious stimuli to the dorsal horn of the spinal column. The small C fibers are responsible for what is termed slow pain and transmit data at under $2.5\,m{\cdot}s^{-1}$. These small fibers outnumber the larger, lightly myelinated Aδ fibers, responsible for fast pain, which conduct at a rate of $4–30\,m{\cdot}s^{-1}$, by a ratio of $\sim 7:1$ in the epithelium. The concept of slow and fast pain is easily conceptualized by a hypothetical injury of one stepping on a nail. The initial sharp sensation, or fast pain, is transmitted by the larger Aδ fibers, while the nagging dull ache, or slow pain, is transmitted by the smaller, unmyelinated C fibers (Fig. 1).

The Aδ fibers synapse with projecting neurons in lamina 1 of the dorsal horn of the spinal cord. In addition to this direct, afferent input, these projecting neurons receive indirect input from the stalk cell neurons in lamina II. These stalk cell interneurons of lamina II receive their afferent input from the C fibers that synapse with them. The projecting neurons of lamina V receive afferent input from the large myelinated Aβ, non-noxious, sensory fibers via dendtritic synapse in lamina IV, from synapse with Aδ fibers in lamina V, and project both to lamina III as well as higher cortical centers (5,6) (Fig. 2).

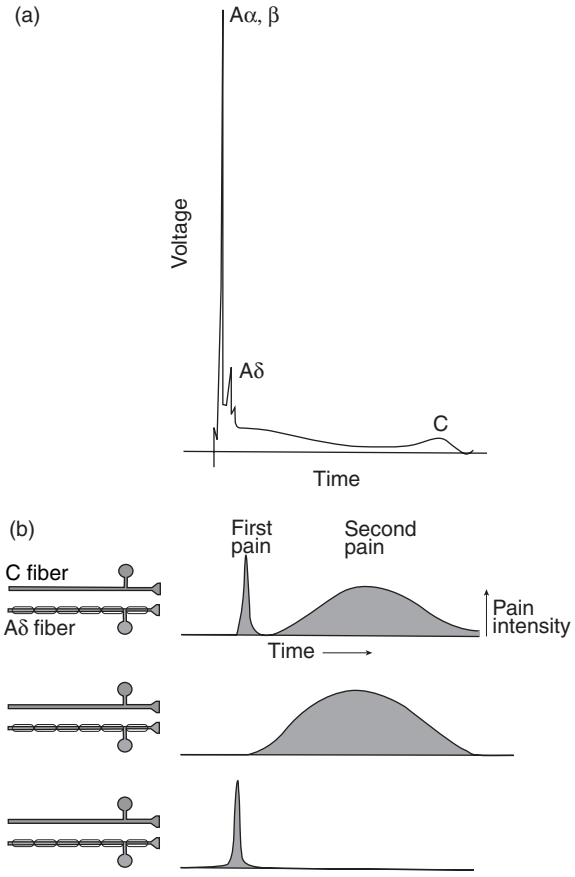

Figure 1. Propagation of action potentials in sensory fibers results in the perception of pain. (Modified from Ref. 4). (a) This electricle recording from a whole nerve shows a compound action potential representing the summated action potentials of all the component axons in the nerve. Even though the nerve contains mostly nonmyelinated axons, the major voltage deflections are produced by the relatively small number of myelinated axons. This is because action potentials in the population of more slowly conducting axons are dispersed in time, and the extracellular current generated by an action potential in a nonmyelinated axon is smaller than the current generated in myelinated axons. (b) First and second pain are carried by two different primary afferent axons. First pain is abolished by selective blockade of Aδ myelinated axons (middle) and second pain by blocking C fibers (bottom).

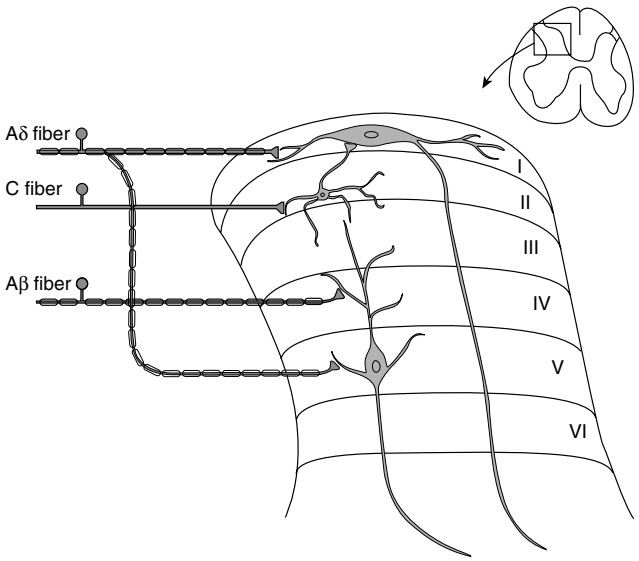

Figure 2. Nociceptive afferent fibers terminate on projection neurons in the dorsal horn of the spinal cord. Projection neurons in lamina I receive direct input from myelinated (Aδ) nociceptive afferent fibers and indirect input from unmyelinated (C) nociceptive afferent fibers via stalk cell interneurons in lamina II. Lamina V neurons are predominately of the wide dynamic-range type. They receive low threshold input from the large-diameter myelinated fibers (Aβ) of mechanoreceptors, as well as both direct and indirect input from nociceptive afferent fibers (Aδ and C). In this figure the lamina V neuron sends a dendrite up through lamina IV, where it is contacted by the terminal of an Aβ primary afferent. A dendrite in lamina III arising from a cell in lamina V is contacted by the axon terminal of a lamina II interneuron. (Adapted from Ref. 4.)

In the dorsal horn of the spinal cord at the synapse level, the afferent pain signal can be modulated to either lessen or amplify the body's response to the pain signal. Serotonin as well as norepinephrine act either directly presynapticly to inhibit the propagation of the pain signal or via activating inhibitory interneurons. The enkephalins, endogenous δ and μ opiate receptor agonists, function at this level to serve a similar inhibitory function. The neuromodulator peptide, substance P is released, along with glutamate from the C fibers, and both work alostericly to amplify the pain signal transmission to higher levels.

Once in the dorsal horn of the spinal cord and after synapse, the afferent pain signal is transmitted to higher cortical centers via either the spinothalamic, spinoreticular, spinomesencephalic, cervicothalamic, or spinohypothalamic pathways. Perception is the final process where all above processes as well as prior physical and psychological experiences interact and create the final subjective and emotional experience of pain. The opioids, both endo-

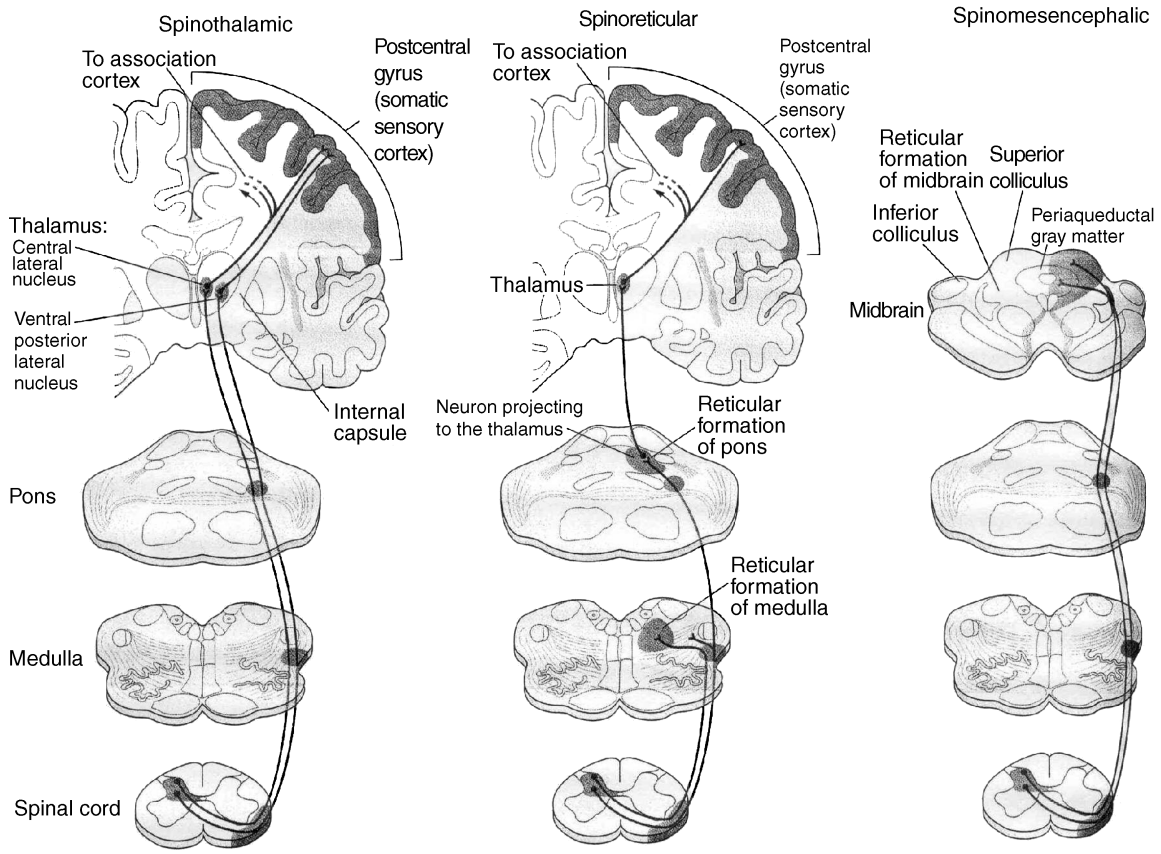

Figure 3. Three of the major ascending pathways that transmit nociceptive information from the spinal cord to higher centers. The spinothalamic tract is the most prominent ascending nociceptive pathway in the spinal cord. (Adapted from Ref. 7.)

genous and exogenous, function to alter perception of pain at the cortical level, as well as to activate inhibitory interneurons in the periaqueductal gray area (Fig. 3).

HYPERALGESIA AND SENSITIZATION

In certain situations, nociceptors can become exquisitely sensitive to stimulation or activated in greater numbers than usual. This results in hyperalgesia and is termed sensitization; this process occurs via distinct mechanisms both peripherally as well as centrally. While peripheral sensitization occurs in both acute and chronic phases of injury, central sensitization generally occurs in the chronic phase of insult, after repetitive noxious events.

Upon peripheral injury, for example, an epithelial incision, inflammation is affected via a large number of chemical mediators, such as prostaglandins, leukotrienes, bradykinin, serotonin, substance P, histamine, potassium, and others, released from both damaged, as well as surrounding tissues (5). These inflammatory mediators serve not only to result in inflammation, but also serve to decrease the threshold to stimulate surrounding nociceptors. This can be done by directly acting to affect sensitization or by working in tandem to sensitize nociceptors via another chemical mediator. For example, bradykinin is an important and extremely potent mediator of hyperalgesia. It works not only to directly sensitize the nociceptive fibers (i.e., C and Aδ fibers), but also serves to stimulate local tissue to produce prostaglandins, which themselves result in sensitization. In addition to bringing about sensitization of nociceptors, some chemical mediators directly activate nociceptors, for example, histamine activating polymodal nociceptors (Table 1).

With continued C fiber pain signal transmission due to persistent noxious insult, increased glutamate is released from their end plates in the dorsal horn. With this increased glutamate release, continued opening of postsynaptic calcium ion channels results. This is mediated by postsynaptic *N*-methyl-d-aspartate (NMDA)-type glutamate receptors. This process, termed "wind-up", results in a continual increase in dorsal horn neuron response to the pain signal. This is an example of pain signal modulation. In addition to this progressively increasing response to the pain signal, dorsal horn neurons can become more easily excitable to a lesser peripheral signal. This process, termed central sensitization, is also mediated by NMDA-type glutamate receptors. Additionally, there is an upregulation in production of a variety of neurotransmitters, neurohormones, and their receptors. Effectively, these changes of excitability and magnitude of C fiber response constitute a pain "memory" and also result in progressively larger areas of peripheral tissue coverage of the dorsal horn neuron. Central sensitization with resultant

Table 1. Naturally Occurring Agents that Activate or Sensitize Nociceptors[a]

Substance	Source	Enzyme Involved in Synthesis	Effect on Primary Afferent
Potassium	Damaged cells		Activation
Serotonin	Platelets	Tryptophan hydroxylase	Activation
Bradykinin	Plasma kininogen	Kallikrein	Activation
Histamine	Mast cells		Activation
Prostaglandins	Arachidonic acid–damaged cells	Cyclooxygenase	Sensitization
Leukotrienes	Arachidonic acid–damaged cells	5-Lipoxygenase	Sensitization
Substance P	Primary afferents		Sensitization

[a]Modified from Ref. 4.

hyperexcitability helps explain allodynia, the perception of a non-noxious stimulus, such as light touch, as a painful stimulus. In light of these changes, it is obvious not only why chronic pain can be so difficult to treat, but also why it is important to break pain "cycles" before chronic changes begin to occur.

Allodynia is classically seen in several different chronic neuropathic pain syndromes for reasons that are not always completely understood, but likely stem from the chronic changes outlined above. Herpes zoster is perhaps the best known of these conditions with many sufferers reporting a severe dermatomal burning pain long after the peripheral nerve damage has healed. Allodynia is common following an attack, and severe bouts of pain can be precipitated from the friction between ones shirt and ones skin. Trigeminal neuralgia is another such chronic condition where allodynia is common. In this condition, lightly stroking one's cheek or the process of eating can precipitate attacks of severe, stabbing transient pain, followed by longer periods of a moderate to severe burning sensation.

PSYCHOLOGICAL ASPECTS OF PAIN

As mentioned earlier, perception of pain is an individualized phenomenon. It is affected by culture, mood, and individual experiences (8,9). Chronic pain can be termed as pain that persists for a certain period, usually ~6 months, after an injury has healed or when the noxious source is idiopathic and central sensitization has occurred. The field of pain management employs a diverse, polymodal disciplinary strategy toward treating pain that extends far beyond simple pharmacotherapy. It includes interventional therapy, physical therapy, meditation, biofeedback, acupuncture, psychiatric therapy, electroanalgesia, and many other treatment modalities. There is a definite psychological component to chronic pain that can cause it to be perceived as much more severe than acute pain and make it refractory to traditional therapy, and chronic pain is frequently associated with depression.

THEORIES OF PAIN

Gate Control Theory

The gate control theory, published in 1965 by Ron Melzack and Pat Wall (10), was the theory from which modern electrotherapy has evolved and that has helped revolutionize treatment and our understanding of pain. The theory states that pain perception depends on the balance of large, afferent sensory Aβ and small diameter afferent nociceptive Aδ and C fiber activity. According to the theory, nonnociceptive sensory fibers can activate neurons in the substantia gelatinosa. These neurons can decrease or inhibit the pain signals of nociceptive neurons prior to higher level transmission. This theory explains the common practice of rubbing an acute wound to lessen pain. It is worth noting that the inhibitory effect of nonnocioceptive neurons is a local one. No analgesic effect exists when rubbing one's toes after an injury to one's fingers (Fig. 4).

The theory's emphasis on the modulation of inputs in the spinal dorsal horn and the dynamic role of the brain in pain processes had a clinical, as well as a scientific impact, and after this theory several methods were developed to modulate the input. Physical therapists and other healthcare professionals began developing and refining different modulation techniques, such as implantable dorsal spinal electrostimulation, and later transcutaneous electrical nerve stimulation devices as well as rediscovering old ones, such as acupuncture. After this discovery, TENS became an important part in treating the acute and chronic pain. See below for a much more thorough discussion of the history of electroanalgesia.

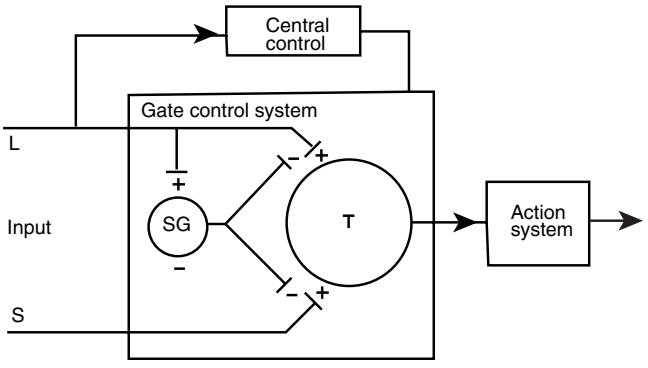

Figure 4. Schematic diagram of the Melzack–Wall gate control theory of pain mechanisms. Large- (L) and small-diameter (S) afferent fibers project to the substantia gelatinosa (SG) and first central trasmission (T) cells. The inhibitory effect (–) of SG on the afferent terminals is increased (+) by activity in L fibers and decreased by activity in S fibers. A specialized system of L fibers (the central control trigger) activates certain cognitive processes that influence the modulating properties of the apinal gating mechanism via descending fibers. (From Ref. 10 R. Melzack and P. D. Wall, *Science*, 150:971–979, 1965, © 1965, AAAS.)

Other Theories Regarding TENS' Analgesic Effect

Other theories have been developed to explain the effectiveness of TENS, namely, the enkephalin and endorphin theories, and likely all three contribute to the analgesic effect. Multiple studies have demonstrated an increase in dorsal horn enkephalin production (11,12) as well as have demonstrated that blockade of opiate receptors lessens or even prevents analgesia from TENS (13–15). As briefly described earlier, enkephalins are μ and δ opiate receptor agonists and function as inhibitory neurotransmitters. Release of enkephalins from inhibitory interneurons decrease Ca^{2+} influx into the nocioceptive neuron, thereby preventing, or lessening depolarization time, prevents or lessens excitatory neurotransmitters, such as glutamate, release, thereby negatively modulating the pain signal. Additionally, enkephalins function postsynapticly to activate K^{+} conductance, thereby hyperpolarizing the dorsal horn projecting neuron and inhibiting pain transmission to further higher cortical centers (Fig. 5).

While enkephalins have a short half-life and function locally, recent studies (16–18) have demonstrated increased levels of circulating endorphins. In contrast to enkephalins, endorphins are circulating μ agonist neurohormones. As such, they act not only on the μ receptors in the dorsal horn of the spinal cord, but also function on central μ receptors to alter the perception of pain and negatively modulate the signal. The discovery that TENS increases endorphin levels is significant. The effect of increasing enkephalins produces a transient effect that lasts only as long as the electrical signal is applied, as is with direct nonnocioceptive stimulation as described in the gate theory. However, use of TENS produces an increase in circulating endorphins that is proportional to usage. The net effect is an analgesic effect that persists after the TENS unit is removed and increases in potency and duration with repeated usage.

The Evolution of Electroanalgesia

The use of electroanalgesia is an ancient practice, and to thoroughly understand the theory and application of TENS, it is important to understand the evolution of electroanalgesia. The powers of certain fish, namely, the Nile Catfish *(Malopterurus electricus)*, Torpedo Fish *(Torpedo mamorata)*, and the Electric Eel *(Gymnotus electricus)* to deliver electrical shocks resulting in paralysis and temporary sensory loss in affected limbs has long been known. The Nile Catfish appeared on walls of various Egyptian tombs, dating from ~2750 bc, and represents the earliest known documentation of this phenomenon of electrical discharge. While it is not known exactly when ancient man discovered the analgesic or anesthetic properties of such fish, it is quite likely that since their discovery by primitive man, these properties were readily apparent.

Exactly when the electrical properties were used for medicinal benefit is unclear, but the earliest known writings describing this benefit were made by made in ad 46 by Scribonus Largus, a Roman physician who described the usage of the torpedo fish as a treatment for intractable gout and headache pain (19). Quoting from his treatise *Compositiones Medicae*, Largus describes these remedies.

> *For any type of gout a live black torpedo should, when the pain begins, be placed under the feet. The patient must stand on a moist shore washed by the sea and he should stay like this until his whole foot and leg up to the knee is numb. This takes away present pain and prevents pain from coming on if it has not already arisen. In this way Anteros, a freedman of Tiberius, was cured* (20).
>
> "*Headache even if it is chronic and unbearable is taken away and remedied forever by a live torpedo placed on the spot which is in pain, until the pain ceases. As soon as the numbness has been felt the remedy should be removed lest the ability to feel be taken from the part. Moreover several torpedoes of the same kind should be prepared because the cure, that is, the torpor which is a sign of betterment, is sometimes effective only after two or three*" (21).

As time progressed, the usage of electroanalgesia spread as a treatment for varying medical conditions. Pedanius Discorides around 80 ad describes the usage of the torpedo fish for rectal prolapse. This represents likely the first description of electrical stimulation for intentional muscular contraction (19). Likewise, these treatments were used and espoused by Galen in the second century.

The knowledge of the usage of the electrical properties of such fish was not limited to Europe. Ibn-Sidah, an Islamic physician described placing an electric catfish on someone suffering a seizure sometime in the eleventh century (21). The use of the electric fish continued to grow and by the sixteenth century the number of remedies had expanded and included treatments for various arthralgias, myalgias, headaches, epilepsy, vertigo, and for inducing sleep both by European, Indian, and Middle Eastern physicians. By the seventeenth century the application of artificially generated electricity was made possible by the development of the electrostatic generator by Otto Von Guericke.

Major revolutions in electroanalgesia came in the mid-nineteenth century from Guillaume Benjamin Amand Duchenne. He introduced the usage of moistened electrodes, as opposed to the more painful dry electrode, as a means of delivering electroanalgesia for treatment of neuropathic pain. His focus on muscle contractions from electrotherapy and making strides toward to a somewhat standardized system of electrode placement were important advances as well.

Throughout the world, electrotherapy became increasingly popular toward the end of the nineteenth and beginning of the twentieth centuries. However, with this rise in popularity came a rise in dubious to downright fraudulent applications and practitioners treating all manners of maladies from skin ailments to weight loss. With an ever increasingly savvy public, the rise of fraudulent applications, the rise of modern pharmacotherapy, X rays, and the like, electrotherapy begin to fall out of favor, or at least popularity, in the early twentieth century (19).

However, technological advances in electrical storage and delivery along with new understandings of pain have produced a resurgence in application and research in electroanalgesia. Shortly after the publishing of the gate control theory, a flurry of exciting discoveries in this

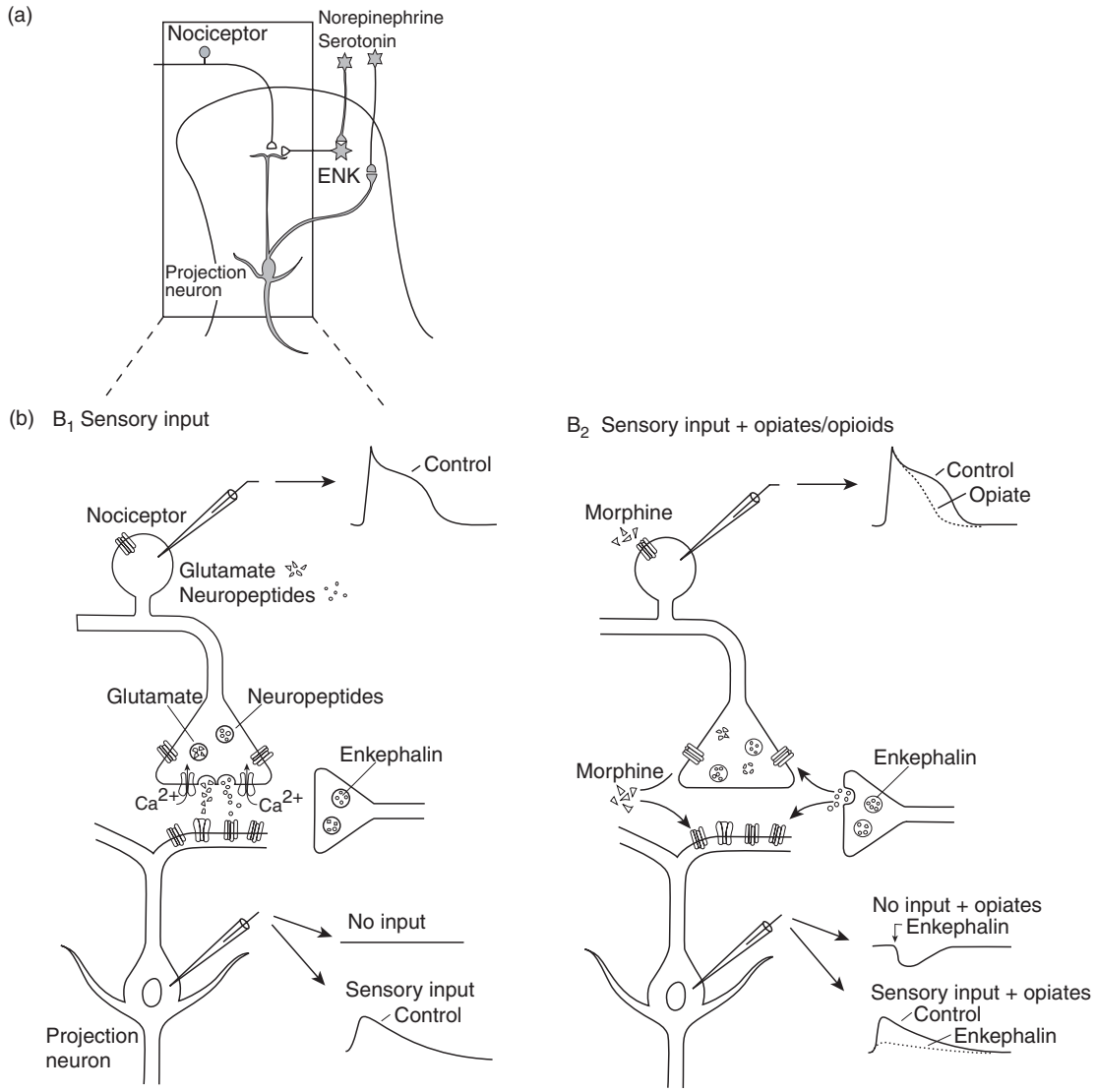

Figure 5. Local-circuit interneurons in the superficial dorsal horn of the spinal cord integrate descending and afferent pathways. (a) Possible interactions between nociceptor afferent fibers, local interneurons and descending fibers in the dorsal horn of the spinal cord. Nocicaptive fibers terminate on second-order spinothalamic projection neurons. Local enkaphalin-containing interneurons (ENK) exert both presynaptic and postsynaptic inhibitory actions at these synapses. Serotonergic and noradrenergic neurons in the brain stem activate the local opioid interneurons and also suppress the activity of spinothaiamic projection neurons. (b) 1. Activation of nociceptors leads to the release of glutamate and neuropeptides from sensory terminals in the superficial dorsal horn, thus depolarizing and activating projection neurons. 2. Opiates decrease the duration of the nociceptor's action potential. probably by decreased Ca^{2+} influx and thus decrease the release of transmitter from primary afferent terminals. In addition, opiates hyperpolarize the mambrane of the dorsal horn neurons by activating a K^{+} conductance. Stimulation of the nociceptor normally produces a fast excitatory postsynaptic potential in the dorsal horn neuron opiates decrease the amplitude of the postsynaptic potential.

realm took place starting with the 1967 demonstration by Sweet and Wall that *In vivo* electrostimulation of peripheral nerves produces analgesia as well as Shealy and Long's work in the area of dorsal and anterior spinal cord surgically implanted stimulators. Shealy and Long discovered that peripheral nerve stimulation done in surgical candidates prior to an electrospinal implant placement produced nearly comparable analgesia to the actual dorsal horn implant (17)! This discovery laid the foundation for TENS development and widespread utilization today (22,23).

While somewhat beyond the scope of this article, it is worth noting that electroaccupunture experienced a widespread rediscovery in China in the 1950s. Though based on a different system of understanding of human physiology than traditional western medicine, this modality of

Figure 6. This represents one of the earliest families of stimulators, with the original model seen on the *left*. The first personal patient treatment model is depicted on the *right*, and a prototype for a miniaturized design is shown in the *center*. The original sponge electrodes are depicted in the *foreground*.

electroanalgesia is beginning to garner increasing interest interest in western medicine (19).

Transcutaneous Electrical Nerve Stimulation

Today electrotherapeutic treatment is one of the most important parts of multidisciplinary approach to treat acute and chronic pain. The TENS units themselves have undergone an evolution from large bulky units to the much smaller units of today. While there are numerous units available, each generally consists of one or more channels for electrodes, a display (either analog or digital), and various options to adjust the various parameters of the electrical current.

One of the First TENS Units Created (below)

The unit (*left*) is one of the first TENS unit available and is large and bulky with an all analog interface. Subsequent units (*center and right*) still remain analog but were more compact, though nowhere near the level of today's units (Fig. 6).

Several Modern TENS Units (below)

Pictured are just several of the numerous commercially available TENS units. Note the compact size of the models compared to older units as well as the digital TENS unit (*bottom*) (Fig. 7 and Table 2).

Electrode

The discovery that transcutaneous peripheral nerve stimulation provided nearly identical analgesic levels as dorsal root stimulation revolutionized electroanalgesia, and it almost goes without saying that the noninvasive, easy to employ nature of TENS is one of the modality's biggest assets. The electrode, the interfacing agent between the skin and machine, has undergone almost as much evolution as the TENS unit itself. The very nature of peripheral transcutaneous nerve stimulation is such that electrical currents must be applied for longer periods of

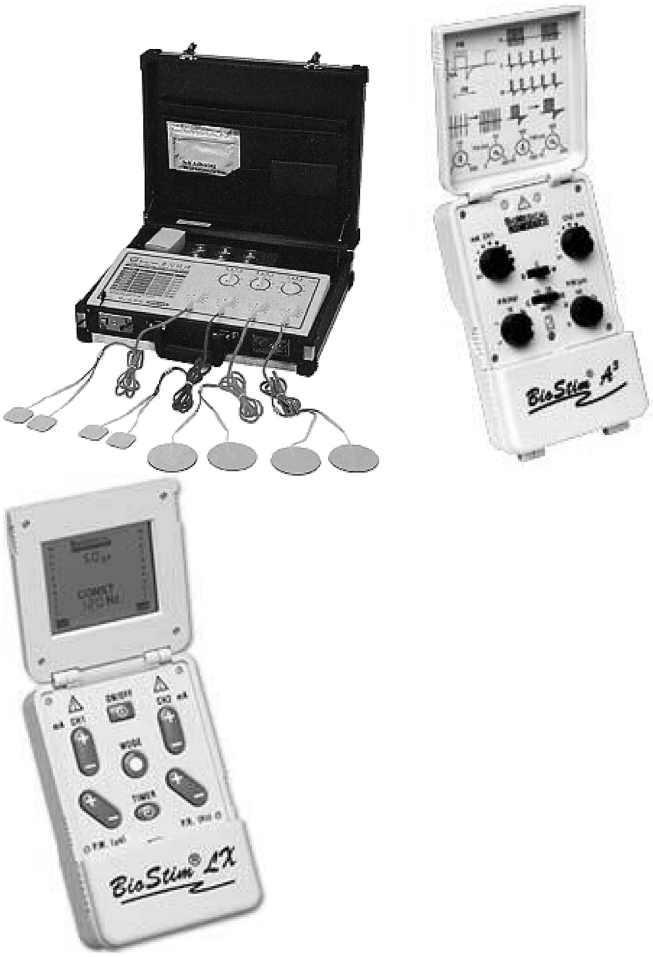

Figure 7. Several commercially available TENS units.

time in greater amounts. While the process of transferring an electrical current from machine to peripheral nerve may seem relatively simple on the outside, several notable problems present ranging from the actual transfer of the current to skin irritation to cost. Several distinct solutions currently are in use, and all present with a variety of tradeoffs (Table 3). Generally speaking, properties of a good electrode for TENS use include low cost/use ratio, good adhesion, comfortable, nonirritating to skin, good electrical conductivity, and easy to use.

Standard EKG or EEG electrodes were initially used for TENS with limited success, as these were designed for much lower amperage and much shorter usage than is needed for effective TENS. It quickly became apparent based on excessive skin irritation and poor adhesion and subsequent nonuniform current distribution that new electrode solutions were needed. One of the most popular current solutions involves silicone impregnated with carbon (Table 3). These carbon silicone electrodes provide the best cost/use ratio of the commercially available electrodes and can often last for months if properly cared for. However, a tradeoff exists in terms of convenience with these carbon silicone pads, as electroconductive gels, rich in suspended ions to facilitate the transfer of electric current from the TENS unit across the epidermis, must be applied

Table 2. Comparisons of Selected Modern TENS UNITS

Manufacturer/unit	Size, cm	Weight, g	Power source	Digital/ Analog	Pulse Width, μs	Stimulus Modes	Burst	Channels	Waveform	Output Current, mA	Output Rate, Hz
Rehabilicare/ ProMax	7.1 × 10.1 × 3.4	122.2	3 AAA Batteries	Digital	50–400	SD, Modulation, Normal, Burst	8 pulses/burst;	2	Asymmetric rectangular biphasic with zero net dc	0–100	2–160
Rehabilicare/ Maxima3	8.4 × 2.5 × 6.3	121	9 V Battery	Analog	50–400	SD, Normal, Burst	8 pulses/burst; Burst at 2 Hz	2	Biphasic, asymmetrical with zero net dc	0–100	2–160
Rehabilicare/ SMP-Plus	9.5 × 6.4 × 2.5	136.2	9 V Battery	Digital	40–300	SMP, Constant, Burst, Modulated Rate, Modulated Width, Multi-modulated	8 pulses/burst; Burst at 2 Hz	2	Biphasic, asymmetrical with zero net dc	0–60	2–125
Empi/Epix VT				Digital	0–400	ELF, Dual Pulse, High Frequency, Ramped Burst, Alternating Ramped Burst, Modulated Amp., Random Modulated, Cycle Burst, Rate Modulation, Multi modulation.	Varies	2	Balanced asymmetrical biphasic	0–60	2–150
Empi/Epix XL				Analog	0–400	Continuous, Burst, Modulated Rate, Multi-modulated	Varies	2	Symmetrical biphasic square zero net charge,	0–60	2–150
BioMedical Life Systems/ BioMed 2000	9.9 × 6.98 × 2.54	132	9 V Battery	Analog	50–250	Constant, Burst, Width modulation,	8 pulses/burst; Burst at 2 Hz	2	Asymmetric rectangular biphasic	0–80	2–150
BioMedical Life Systems/ BioStim A6	9.9 × 6.98 × 2.54	132	9 V Battery	Analog	10–250	Constant, Burst, Width modulation, Rate Modulation, rate/width modulation, cycled burst	Cycled	2	Asymmetric rectangular biphasic	0–100	2–200
BioMedical Life Systems/ BioStim LX	9.5 × 6.3 × 3.2	226	4 AA Batteries	Digital	10–250	Constant, Burst, Burst Programmable, Width modulation, Cycled Burst	Cycled	2	Asymmetric rectangular biphasic	1–98	1–150
BioMedical Life Systems/ BioStim M7	8.2 × 7.0 × 4.5	266	4 AA Batteries	Digital	10–250	Constant, Burst, Burst Programmable, Width modulation, Rate Modulation, rate/ width modulation, cycled burst	Cycled	2	Asymmetric rectangular biphasic	0–98	1–200

Table 2. (*Continued*)

Manufacturer/unit	Size, cm	Weight, g	Power source	Digital/ Analog	Pulse Width, μs	Stimulus Modes	Burst	Channels	Waveform	Output Current, mA	Output Rate, Hz
BioMedical Life Systems/ BioStim Plus	9.9 × 6.98 × 2.54	132	2 AA Batteries	Digital	10–250	Constant, burst, cycled burst, Pulse rate modulation, pulse width modulation	Cycled/ 2 presets	2	Asymmetric biphasic, square wave	0–98	1–150
Vital/TENS Deluxe	9.1 × 6.4 × 2.3	130	9 V Battery	Analog	50–250	Burst, modulation, constant	7 pulses/burst; Burst at 2 Hz	2		variable	2–120
Kingly Star/ KS-168	40.6 × 18.5 × 6.8	1060.5	UM-1 × 6 Battery or 9 V dc adapter	Analog		Constant, Burst, Modulation		4		0–100	2–200
Pain Management Technologies/ Medscope Pro	10.1 × 5.9 × 2.37	140	9 V Battery	Digital	50–260	Constant, Burst, pulse rate modulation, pulse width modulation	7 pulses/burst; Burst at 2 Hz	2	Biphasic square wave with zero net dc	0–80	2–150
Pain Management Technologies/ Bioscope	9.85 × 6.05 × 2.45	134	9 V Battery	Analog	60–250	Constant, Burst, Pulse rate and Pulse width modulation	~7 pulses/burst; Burst at 2 Hz	2	Asymmetric biphasic square	0–15	2–150
ProMed Specialties/ ProM 100	9.1 × 6.4 × 2.4	130	9 V Battery	Analog	40–250	Constant		2	Asymmetric biphasic square with zero net current dc	0–80	2–150
ProMed Specialties/ ProM 200	9.1 × 6.4 × 2.3	130	9 V Battery	Analog	40–260	Burst, Constant, Modulation	9 pulses/burst; Burst at 2 Hz	2	Asymmetric biphasic square with zero net current dc	0–80	2–120
ProMed Specialties/ ProM 300	9.1 × 6.4 × 2.3	130	9 V Battery	Analog	40–260	Burst, Constant, Modulation	9 pulses/burst; Burst at 2 Hz	2	Asymmetric biphasic square with zero net current dc	0–80	2–50
Shining World Health Care Co./ SW 325	12.5 × 6.6 × 2.8	138	9 V Battery	Digital	200	Constant	n/a	2	Biphasic Spiked Wave	0–50	
Body Clock Healthcare/ Profile	10.5 × 6.5 × 2.75	100	2 AA Batteries	Digital	25–250	Constant, Burst, Width modulation, Rate modulation	Variable	2	Symmetric biphasic rectangular	0–100	1–200
Med-Dyne/TA3	9.1 × 6.4 × 2.3	130	9 V Battery	Analog	40–260	Constant, Burst, Modulation	9 pulses/burst; burst at 2 Hz	2	Asymmetric biphasic square with zero net current dc	0–80	2–160

Table 3. Basic Types of Electrodes

General electrode Type	Number of Uses	Typical Retail Cost Electrode (pair)	Typical features			
			Adhesion and Conduction	Composition Materials	Advantages	Disadvantage
Disposable	1 use	Under $3.00	Pressure-sensitive tape surrounding conductive area (wet get in sponge)	Nonwoven Foam	Easiest to use Very good adhesion Comfort	Cost/use Skin irritation Poor electrical performance
Semireusable	3–10 uses	$5.00–10.00	Conductive adhesive over entire surface	Foam Plastic film Carbon silicone	Ease of use Low skin irritation Comfort	Weak adhesion Care and storage Medium cost per use Good electrical performance
Reusable	>100 uses	$4.00[a]	Requires addition of conductive gel and tape adhesion	Carbon silicone	Very good electrical performance (if applied properly) Lowest cost per use	Most preparation to use Skin irritation Messy Requires gel and tape Skill required for optimal performance Poor adhesion Not as flexible in use

prior to each usage. The electrodes must then be affixed with tape to the skin. This process can be laborious if not impossible for the end user to do, depending on electrode location as well as physical disability. While numerous medical tapes exist, care must be taken in their selection. Some users display mild-to-severe allergic reactions to adhesives in various tape products. Likewise, certain adhesive tapes adhere too firmly to the skin and can result in injury with repeated application and removal.

In applications where cost is no object, sterility is needed, or convenience must be maximized, single use adhesive electrodes are used that consist of thin, porous material impregnated with adhesive electroconductive gel covered with cloth or plastic on one side to prevent adhesion to clothing or electrical current escape. These electrodes are used extensively in hospital or rehab facilities where numerous patients are seen and reusing electrodes is neither feasible nor sanitary as well as in individuals who desire or require maximum convenience.

A third option in electrode selection includes so-called "dry gel" electrodes manufactured from a conductive polysaccharide gum, called Karaya or Sterculia gum when taken from the Sterculia Urens tree native to India or a manufactured comparable material. These dry gel electrodes represent a good compromise between the disposable and reusable carbon silicone electrodes, as they are self adhesive, do not require electroconductive gel application, may be reused several times, and represent a significantly lower cost/use ratio than disposable pads.

Patients should be informed of the various pros and cons of the various electrodes as well as counseled regarding proper usage. According to Szeto, several factors should be considered when selecting the proper electrode:

1. How long will each application of the electrodes last, and therefore what is the level of adhesion needed?
2. Is the stimulation site readily accessible or will there be someone to assist? How simple must the application of the electrodes be?
3. What is the patient's skin type (hairiness, oiliness, hyperallergic)? Will special pregelled electrodes be required?
4. Where is the painful are? This factor will help to determine the best electrode size, shape, cosmesis, and number.
5. Does the TENS user lead an active life? If so, a high performance electrode in terms of adhesiveness, pliability, and nonirritability would be needed.
6. Can the user take good care of the electrodes, and what are the financial arrangements? These issues will affect the cost-effectiveness of disposable or semireusable type of electrode (24).

Electrode size is another factor to consider in selection and depends on the location of pain, area required for stimulation, and personal preference. Numerous sizes and shapes of electrode pads are available and suitable for virtually any application.

Sample Electrodes

Electrode Placement

Electrode placement is crucial in maximizing positive outcome with TENS units. Most units employ two or more channels of current, which splits to two electrodes, and it is often advisable for multiple channels to be used to cover maximal areas, as many pain syndromes often do not exhibit pain localized to a specific point source. Numerous books on TENS or manufacturer information as well as various anatomical charts provide users with possible electrode placements. While it is impossible to describe

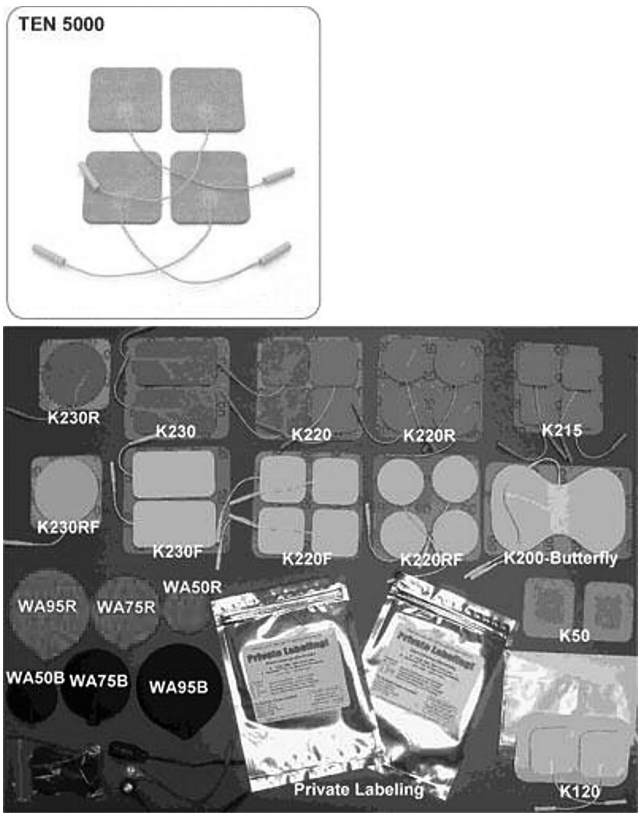

Figure 8. TENS electrodes.

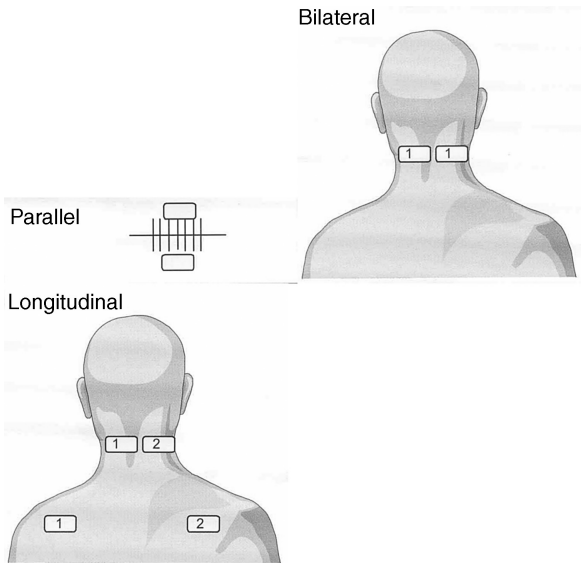

Figure 9. Bilateral pain electrode placement.

proper electrode placement for every pain syndrome, certain electrode arrangements are frequently used.

For the purposes of this discussion, the channels will be referred to as I and II and the negative electrode as "a" and the positive as "b". Parallel placement with one channel is utilized for relatively localized areas of pain, such as point pain or pain from a surgical incision. Electrode Ia is placed on one side of the incision, while Ib is on the other, producing a current that flows between the two with the area of pain in between the electrodes. Bilateral placement is similar, but generally defined as meaning Ia and Ib electrodes are placed on either side of the spine, symmetrically and close together, useful for localized, nonradiating neck or back pain. For radiating neck or back pain, a longitudinal arrangement is often utilized in which electrodes of one channel are on the same side of the spine and placed along the pain pathway. If the pain is bilateral, electrodes of another channel on the can be placed on the opposite side of the spine along the pain pathway (Fig. 9).

A crossed, or interferential, placement is useful for pain localized to large joints, such as shoulder, elbow, or knee. In this pattern, Ia and IIa are placed side by side with IIb below Ia and Ib below IIa, creating a square pattern with electrodes of the same channels diagonally opposite each other with the area of pain in the center of the square. Bracketed placement is generally reserved for treating the dermatomal neuralgia that frequently is associated with shingles, varicella zoster, out breaks. In this arrangement, electrodes Ia and Ib are placed along the dermatome cranial to the neuralgia and electrodes IIa and IIb placed along the dermatome caudal to the neuralgia (Fig. 10).

Occasionally, localized pain is so severe that the user cannot tolerate electrode placement over the affected site, and in this case a contralateral placement in the nonaffected hemisphere over the same anatomical area as on the affected side is utilized. This arrangement will sometimes permit sufficient pain relief for the user to eventually tolerate direct stimulation. While the exact mechanism of analgesia is not known, it is hypothesized the analgesic effect is the result of central inhibitory pathways (24). Certain syndromes, such as Reflex Sympathetic Dystrophy, lend themselves to this placement, and reflex vasodilatory effects may explain contralateral analgesia in these syndromes (25) (Fig. 11).

Certain pain syndromes, such as phantom limb pain, glove–stocking distribution peripheral neuropathy, or acute burns, fractures, lacerations, or other injuries of the hands or feet lend themselves to a placement of the electrodes proximal to the actual source of the pain. In this placement, the electrodes of one channel are simply placed along the dermatome of the pain source, but proximal to the pain (Fig. 12).

The final placement method to be covered is a linear, unilateral, overlapping pattern useful for pain along some, as in myofacial pain, or all, as in radicular pain, of an extremity, and follows a placement procedure outlined by Wolfe (25). After the dermatomal distribution of the pain is elucidated, electrode Ia is placed at the most proximal location where the user experiences pain. Distal to this electrode, the user identifies the site of maximal pain and places IIa here. At the most distal site of pain, electrode IIb is placed, and between IIa and IIb, electrode Ib is placed, being careful to keep all electrodes in the affected dermatome (Fig. 13).

It is important to note with the above placement, the electrical current covers the entire length of the pain the

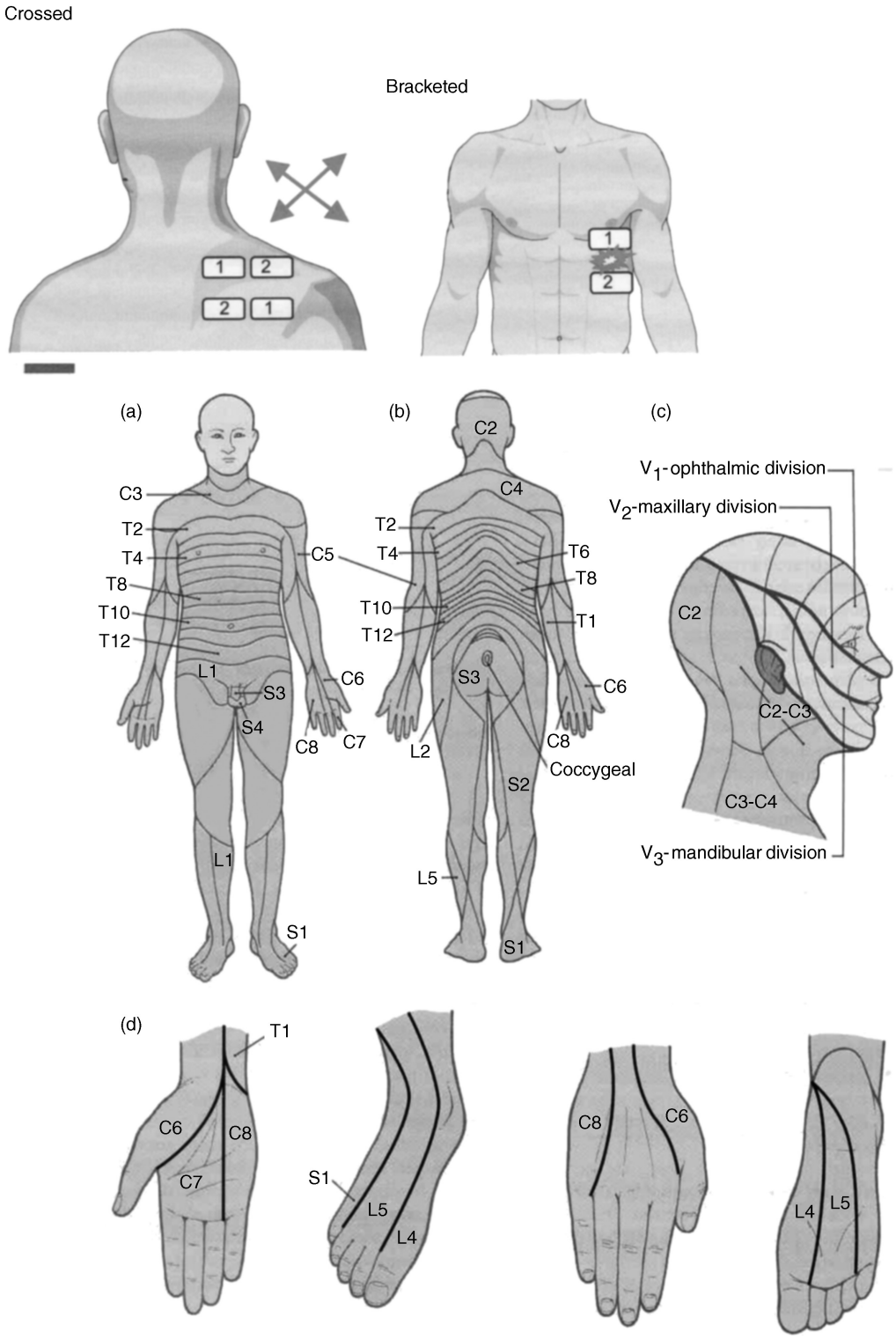

Figure 10. Dermatomal maps of the peripheral distribution of spinal nerves (a and b) and trigeminal nerve (c) d. Details of termatomal maps on anterior and posterior surfaces of the hand and foot.

user experiences. If the user inadvertently places electrodes in a nonoverlapping pattern (i.e., Ia and Ib both proximal to IIa and IIb), the current will not cover the entire pain pathway; instead it will only travel between electrodes of the same channel, leaving the area between Ib and IIa "uncovered". The following should be generally avoided due to poor current coverage area: unilateral, linear (Fig. 14).

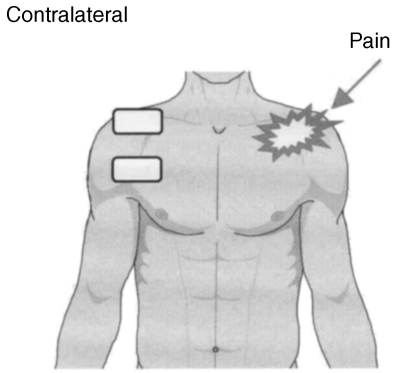

Figure 11. Contralateral electrode placement.

Electric Amplitude–Frequency Selection

Once the proper electrode type is chosen, as well as optimal electrode placement ascertained, the optimal electrical signal must be selected. Generally speaking, the most used currents include "classical" TENS with high intensity–low frequency currents for up to 12 h at a time, low intensity–high frequency currents for up to 45 min several times a day, and intermittent low frequency bursts are used. The varying current intensity–frequency produces analgesia via the different mechanisms as discussed above.

Classical TENS employs high frequency (60–200 pulses per second)/low intensity (15–60 mA) stimulation and produces a distinct "electrical tingling" sensation in the area of electrode pad placement that most users find pleasant. This current is not of significant intensity to produce significant muscular contraction. Pain relief from this form of stimulation is transient, occurring quickly once stimulus is applied and disappearing once current is removed, and the gate control theory likely explains the mechanism of analgesia. The high frequency pulses activate *A*β sensory afferent fibers and inhibit pain transmission in the dorsal column of the spinal cord.

Low frequency (1–5 pulses per s)/high intensity stimulation, producing sustained muscle contractions, results in slower onset pain relief that persists after the stimulus is removed. Numerous studies have demonstrated partial to near total inhibition of analgesic effect via administration of naloxone (13–15). The endorphin and enkephalin theories described previously likely largely account for the mechanism of analgesic activity, especially the endorphin theory and long-term analgesia. While effective at inducing long-term analgesic effects, the low frequency–high intensity method of stimulation is often perceived by many patients as less pleasant than high frequency stimulation.

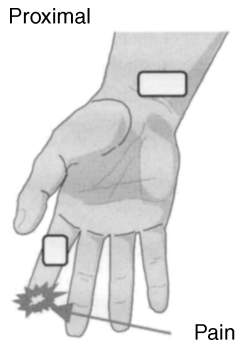

Figure 12. Proximal electrode placement.

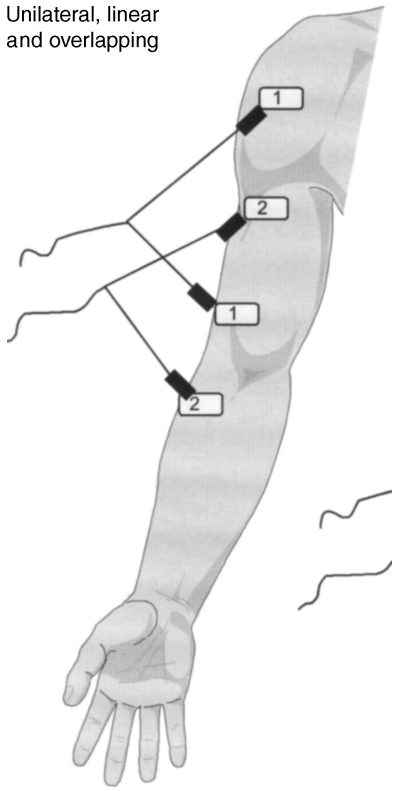

Figure 13. Unilateral, linear, and overlapping electrode placement.

As the long-term effects of low frequency intense stimulation are desirable, manufactures have devised means of producing a more pleasurable sensation while at the same time stimulating muscle contraction enough for long-term analgesia via modulation of the current. To understand the modulation of current in TENS, a brief review of the current waveforms it employs is needed. Briefly, biphasic waveforms, consisting of both a positive and negative phase are used, and these may be either symmetric or

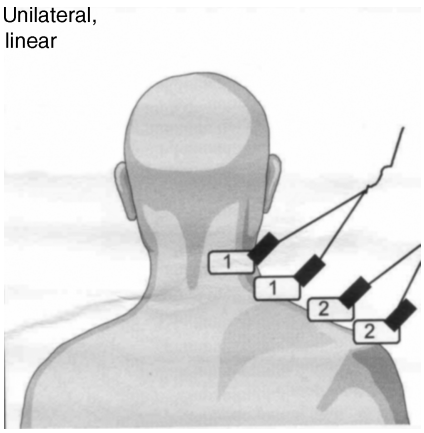

Figure 14. Unilateral, linear electrode placement.

asymmetric. If the current amplitude is equally positive and negative, the current is termed "electrically balanced," also referred to as zero net charge (znc) or no net dc current. While both balanced and unbalanced electrical currents are employed, unbalanced current transmission can result in pH changes in the skin with long-term usage, do to ion exchange, which can result in skin irritation. Additionally, the current employed in TENS is a pulsatile current with interspaced periods of electrical activity and electrical silence. The periods of electrical silence may be either uniform or varying. The frequency of electrical pulses may be given in units of hertz (Hz), cycles per second (cps), or pulses per second (pps) (25). It is important to note that while frequency may be a constant 100 cps, the period between the pulses may be variable.

All of these variables in the current waveform may be adjusted to achieve a net effect that is both pleasant to the patient while sufficient to achieve muscular contraction. For example, the amplitude of the current may be varied over a constant time interval, the duration of the pulse may be varied, the time between pulses may be varied, or a combination of some or all of the previous may be used. As pain severity and character can frequently change, models that allow modulation of electrical current via one or more characteristic offer distinct advantages in patients' individualizing their therapy as well as help prevent physiologic adaptation to the electrical stimulation. While numerous studies have delved into optimizing the electrical waveform (26–28), their conclusions have been varied, and it is likely, there is no optimal waveform. As such, TENS therapy is an individualized one, and patients should have frequent follow ups with their physician to ensure the patient is receiving optimal care for their specific complaint (Table 4).

Table 4. INDICATIONS for Use of TENS

Systemic Pain	
Bursitis	Phantom limb syndrome
Cancer	Raynaud's syndrome
Causalgia	Rheumatoid arthritis
Multiple sclerosis	Synovitis
Neuralgia	Diabetic peripheral
Osteoarthitis	Neuropathy
Fibromyalgia	
Head and Neck Pain	
Cluster headaces	Suboccipital headaches
Dental disorders	TMJ Syndrome
Migraine headaches	Torticollis
Spondylosis	Trigeminal neuralgia
Sprains/strains	Whiplash
Abdominal Pain	
Diverticulosis	Labor
Dysmenorrhea	Postoperative pain
Back Pain	
Facet syndrome	
Intercostals neuralgia	Radiculitis
IVD Syndrome	Sprains/strains
Lumbago	Thoracodynia
Lumbosacral pain	Whole back pain
Lower Extremity Pain	
Ankle pain	Passive stretch pain
Foot pain	Sciatica
Fractures	sprains/strains
Ischialgia	tendonitis
Knee pain	Thrombophlebitis
Upper Extremity Pain	
Epicondylitis	
Frozen shoulder	Sprains/strains
Hand pain	Subdeltoid bursitis
Peripheral nerve	Wrist pain
Injury	

TREATMENT PLANS

Treatment with TENS is an extremely variable and personalized process, and this cannot be underscored enough. It is vital for close healthcare supervision for a user to obtain the maximum therapeutic benefit from tens. TENS may only be used in an acute phase for a short period of time (e.g., incisional pain postsurgery) or for months or years (e.g., those suffering from chronic back pain). For chronic pain sufferers classical TENS may be used for several hours continuously daily. Modulated or low frequency/high intensity may be used for ~30 min three times a day for an indefinite period of time. When using TENS it is important to use as strong or nearly asstrong a current as the user can tolerate to achieve best results.

Fibromyalgia is a poorly understood chronic pain condition that presents unique management challenges not only because it is poorly understood, but also because it is often refractory to traditional treatment modalities. A recent double-blinded study by Cork et al. (29) explored cranial electrotherapy stimulation (CES) as a possible treatment for fibromyalgia. In this study, using electrodes clipped to the participants' ear lobes, the Alpha-Stim CES device, delivered either modified square-wave stimulation at 100 μA and a 50% duty cycle at 0.5 Hz for 1 h daily for 3 weeks or sham therapy (see Fig. 15). While there were no

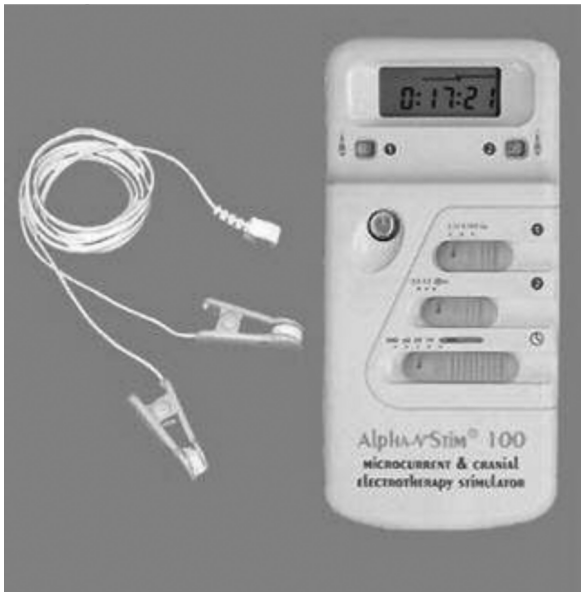

Figure 15. The Alpha-Stim CES device.

Figure 16. Electrode placement for CES.

differences in baseline pain scores of the participants in either group prior to beginning the study, after 3 weeks of CES therapy those in the treatment group displayed significantly lower Pain Intensity Scores, Tenderpoint Scores, and POMS Scores compared to the sham group. After 3 weeks the study was unblinded, and 23 of the 35 participants in the Sham group elected to switch over to active treatment for 3 weeks. Those switching from sham therapy to active CES therapy displayed significant reductions in the aforementioned pain scores as well (29) (Fig. 16).

WARNINGS AND CONTRAINDICATIONS

TENS is contraindicated in individuals with pacemakers, especially those with demand-type pacemakers as the electrical stimulation could cause misfiring or other malfunction of the pacemaker. Electrode placement in areas of sensory or circulatory deficits should be avoided due to the potential for burns or excessive muscular contraction. Electrodes should not be placed over the carotid sinuses to prevent a vasovagal reflex reaction with resultant hypotension. Electrodes should not be placed over the anterior neck due to potential to induce laryngospasm and subsequent asphyxiation. Electrodes should not be placed over the eyes. TENS should be avoided in pregnant women due to the potential to induce contractions and premature labor. Caution should be used in patients with implanted spinal stimulators as well as intrathecal opiate pumps. The unit should not be used with other electrical medical equipment, such as ECGs, EEGs, pulse oximeters, and electrocautery devices.

PRECAUTIONS

Tens has not been proven to have curative value and should be used only under the supervision of a physician. Patient selection is crucial, and not all patients will respond to TENS. The TENS has not been shown to exhibit curative value and should not be used for pain of unknown origin. The unit itself as well as wires and electrodes should be kept out of reach of children and water.

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See also BLADDER DYSFUNCTION, NEUROSTIMULATION OF; ELECTROANALGESIA, SYSTEMIC; ELECTROPHYSIOLOGY; FUNCTIONAL ELECTRICAL STIMULATION.

TRANSPLANTATION, LIVER. See LIVER TRANSPLANTATION.

TRAUMA MANAGEMENT. See CARDIOPULMONARY RESUSCITATION.

TWEEZERS, OPTICAL. See OPTICAL TWEEZERS.