

Electroanalgesia: Its Role in Acute and Chronic Pain Management

Paul F. White, PhD, MD, FANZCA, Shitong Li, MD, and Jen W. Chiu, MB, MMed, DEAA

Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, Texas

The practice of using electrical stimulation for pain control began centuries ago. However, renewed interest in electroanalgesia is related in part to a better understanding of the physiologic basis of pain perception and transmission, as well as to the efforts of researchers interested in finding alternatives to the traditional opioid and nonopioid analgesic drugs. Electrical stimulation has been applied directly to the spinal cord, deep brain centers, peripheral nerves, and to the traditional Chinese acupoints, in an effort to improve the management of acute and chronic pain. This report examines the current scientific evidence supporting the use of electroanalgesia in pain management.

Spinal Cord Stimulation

In 1967, Shealy et al. (1) first reported on the use of an implantable device for direct spinal cord stimulation (SCS). The use of a dorsal column stimulator has been alleged to modulate the perception of afferent nociceptive stimuli. However, because of the paucity of carefully controlled studies, the efficacy of SCS remains controversial even after >30 yr of clinical use. The proposed mechanism of SCS-induced analgesia is based on Melzack and Wall's Gate Control Theory (2). These investigators proposed that modulation of pain perception is ascribed to recruitment of large-diameter A β afferent fibers in the dorsal column of the spinal cord, thereby "closing the gate" and preventing the transmission of pain impulses via small-diameter C fibers. Alternative mechanisms that have been proposed for electroanalgesia include activation of descending pain modulation pathways, inhibition of sympathetic efferents, tonic (use-dependent) blockade, and antidromic conduction (3). Still others have suggested that the mechanistic basis for this form of

electroanalgesia relates to alterations in the levels of endogenous analgesic (and antianalgesic) substances within the central nervous system (4,5).

When implantation of an SCS device is considered, a two-stage procedure is recommended. Trial placement of the electrode(s) and functional testing with an external stimulator should be undertaken before permanent implantation to increase the probability of achieving a successful outcome with SCS (Table 1) (6,7). The stimulating electrodes are usually inserted percutaneously via an epidural needle under fluoroscopic guidance and positioned to obtain optimal parasthetic coverage of the nociceptive areas. However, a recent study concluded that electrodes placed via a thoracic laminectomy were associated with significantly better long-term effectiveness than electrodes placed percutaneously in patients with chronic pain involving the lower back and lower extremities which was refractory to conservative therapy (8). The main complications associated with SCS relate to infection (e.g., wound infection, epidural abscess), bleeding, equipment failure (e.g., electrode migration, lead fractures, circuit leaks, battery failure, fibrosis of the stimulating tip), and the development of tolerance (7,9).

SCS is currently used to treat a wide variety of inoperable and intractable chronic pain syndromes. In patients with failed conservative and surgical treatment of lower-limb ischemia (10), SCS increases skin blood flow, decreases pain, and improves "quality of life." According to recent systematic reviews (7,11), the most favorable results have been observed in patients with peripheral vascular disease, complex regional pain syndrome, and peripheral neuropathy (e.g., diabetic or causalgic origin) (7,12). Of interest, the pain relief achieved with SCS in patients with complex regional pain syndrome is possible without vasodilation (13). The vasodilation found with SCS is attributed to an inhibitory effect on sympathetically maintained vasoconstriction. Diabetic patients with peripheral arterial occlusive disease who present with intractable pain have also been successfully treated with SCS, except those who have severe autonomic neuropathy (14). Recently, SCS has been successfully

Accepted for publication October 10, 2000.

Address correspondence to Paul F. White, PhD, MD, FANZCA, Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, 5323 Harry Hines Blvd., F 2.208, Dallas, TX 75390-9068. Address e-mail to paul.white@utsouthwestern.edu.

Table 1. The Beneficial Effect of a Trial with Percutaneous Electrical Stimulation on the Long-Term Success of SCS in Patients with Chronic Pain Syndrome (7)

Diagnostic category	- Trial (%)	+ Trial (%)
Failed back syndrome	46	51
Peripheral neuropathy/ischemia	49	71
Reflex sympathetic dystrophy	100	100
Spinal cord lesion/cauda equina	13	22
Multiple sclerosis	61	67
Miscellaneous (e.g., rectal, bone, joint, phantom limb)	18	33
Overall success (%)	47	59

SCS = spinal cord stimulation, - trial = implantation of SCS without "trial" test, + trial = implantation of SCS after positive "trial."

used to treat intractable angina pectoris (15) and chronic mesenteric ischemia (16). However, pain caused by long-standing failed back surgery, cauda equina injury, paraplegic pain, and phantom limb pain, do not appear to respond well to SCS (12).

Interestingly, the most common reason for implanting an SCS device is failure of lumbar or cervical surgery (i.e., failed back syndrome). Kumar et al. (7) found that with failed back syndrome, the patients who benefit the most from SCS are those in whom the device was implanted within 3 yr of the initial back operation (Fig. 1). They reported that the success rate of SCS decreased from 93% for patients with less than a 3-yr interval between surgery and implantation to 9% for those exceeding a 12-yr interval. In a prospective, randomized comparison of SCS and reoperation for treatment of failed back surgery syndrome, North et al. (17) found a statistically significant ($P = 0.02$) advantage in favor of SCS over reoperation at the 6-mo cross-over point. At a mean follow-up of 7 yr, 52% of patients who received a permanent SCS reported at least 50% continued pain relief (18). A majority of the patients in this series maintained improvements in activities of daily living and analgesic use. With programmable multichannel devices, significantly fewer clinical and technical failures have been reported.

In a systematic literature review of 39 case reports, Turner et al. (11) analyzed the long-term risks and benefits of SCS for patients with failed back syndrome. After a mean follow-up of 16 mo, only 59% of the patients had >50% pain relief. Burchiel et al. (19) performed a prospective, multicenter study to investigate the efficacy of SCS in patients with chronic back or extremity pain. Of the 219 patients studied, 83% were implanted with permanent stimulating devices. Of the 70 patients who completed a >1-yr follow-up

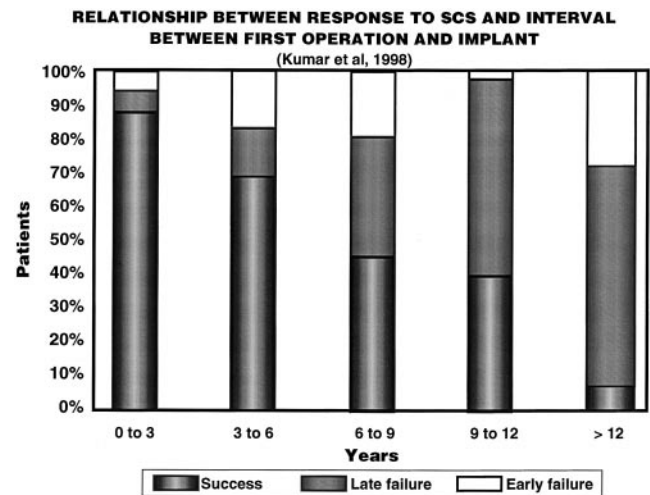


Figure 1. Relationship between long-term (analgesic) response to spinal cord stimulation (SCS) and the time interval between the patient's first back operation and implantation of the SCS device (7).

evaluation, 55% achieved sustained improvements in pain and quality-of-life measures.

Kumar et al. (7) reported their experience in using SCS for chronic pain management in 235 patients who were followed for a mean period of 5.5 yr. Of the 80% who received permanent devices, 59% continued to receive satisfactory pain relief at their last follow-up, and of these, 42% were gainfully employed compared with only 20% before implantation. However, the ability to resume activities of daily living may be a more appropriate endpoint than return to work or alleviation of pain in this patient population.

Although judicious patient selection based on documented pathology and psychological stability has been alleged to be important in achieving success with SCS, North et al. (20) found little evidence to support the value of psychological testing in selecting patients for SCS. Other predictors of long-term success include the following: 1) percutaneous insertion of stimulating electrodes to determine that the stimulation-induced paresthesia encompassed the topographic distribution of the patient's pain, 2) use of multipolar stimulators and dual leads to augment the range of stimulation and paresthesia coverage, and finally, 3) a positive trial stimulation before definitive SCS implantation. In a recent study, North et al. (21) found that an insulated four-contact array implanted via laminectomy provided more effective and sustained pain relief than a percutaneous four-contact electrode of the same design. Although the criteria used to define success with SCS have differed among the various studies, most experts would require a 50% or more decrease in pain compared with pretreatment levels after at least 1 yr of using the device.

Thus, SCS may be a useful electroanalgesic therapy in properly selected patients. By reducing the demand

for medical care, SCS is cost-effective in patients with failed back surgery syndrome when used for an average of 5.5 yr (22). The development of multilead electrodes allows for more extensive neuraxial stimulation. However, SCS is an invasive and expensive analgesic intervention with potentially serious side effects, and therefore should be reserved for the management of severe, intractable chronic pain syndromes. Although SCS may be a reasonable alternative to reoperation for patients with failed back syndrome, the lack of randomized, prospective, controlled trials precludes conclusions concerning its efficacy and cost-effectiveness relative to other less invasive electroanalgesic modalities, or even no specific treatment at all (11).

Deep Brain Stimulation

Pain relief after deep brain stimulation (DBS) was first evaluated in the late 1950s (23). The most common targets of stimulation have been the periventricular and periaqueductal gray matter in the mesencephalic-diencephalic transition zone, ventroposterolateral-medial nucleus of the thalamus, internal capsule, and motor cortex. The proposed mechanisms of DBS relate to decreasing pain transmission along sensory-discriminative pathways and/or the release of endogenous endorphins. A recent study demonstrated that thalamic DBS resulted in activation of the anterior cingulate cortex in patients with chronic pain (24). DBS has mainly been used in the management of debilitating chronic pain syndromes after all other less-invasive therapeutic modalities (including SCS) have failed. The potential for serious intracranial complications (e.g., intracranial hemorrhage, infection, and oculomotor abnormalities) has limited the more widespread application of this highly invasive therapeutic modality.

The use of DBS involves stereotactic implantation of a stimulator and electrode guided by ventriculography, computerized tomography, or magnetic resonance imaging. Electrical stimulation is activated via percutaneously placed leads when the target site has been located, and final electrode placement is contingent upon a favorable response to direct stimulation. The use of a multipolar electrode system allows for a number of bipolar stimulation combinations to be evaluated during the trial period. Analogous to SCS, the stimulator would only be internalized if satisfactory pain relief was achieved during the trial period.

In a 15-yr experience involving the use of DBS for intractable pain syndromes, Kumar et al. (25) reported that 78% of the 68 patients had the stimulating devices internalized, and of these, 79% continued to receive adequate pain relief on follow-up evaluation at the end of 1 yr. Literature reviews (25,26) suggest that

DBS was effective in treating refractory failed back syndrome, trigeminal and peripheral neuropathy, and deafferentation and somatic pain, with 1-yr success rates of 50% to 80%, depending on the etiology and stimulation site. Interestingly, paresthesia-producing (but not periventricular gray) DBS provides effective analgesia in patients with stable neuropathic pain (27). Patients with thalamic pain, spinal cord lesions, and postherpetic neuralgia respond poorly to DBS. Although DBS is a nonablative alternative to SCS and peripheral nerve stimulation, it is still considered investigational and has not yet found a clearly defined role in chronic pain management.

Peripheral Nerve Stimulation

The use of peripheral nerve stimulation (PNS) for the relief of chronic pain states was first reported over 30 yr ago by Wall and Sweet (28). Recent studies have demonstrated that electrical stimulation of peripheral nerves leads to inhibitory input to the pain pathways at the spinal cord level (29). To date, the success rate of PNS has been most favorable in the treatment of neuropathic pain (e.g., posttraumatic neuropathy, diabetic neuropathy) when the nerve lesion is distal to the site of stimulation. For lesions proximal to the stimulation site, in particular lumbar radiculopathy, the results have been much less impressive (30). Before the permanent implantation of a stimulating device (e.g., SCS, DBS), patients should undergo a series of less invasive testing procedures, including diagnostic peripheral nerve blocks, percutaneous electrical stimulation, and/or electrophysiologic analyses.

Percutaneous Electrical Nerve Stimulation

Percutaneous electrostimulation was originally described by North et al. (31) for the treatment of intractable pain associated with chronic low back pain syndrome, cancer, and other disorders. Electrical stimulation of the spinal cord was achieved by using electrodes inserted percutaneously into the epidural space by using a Touhy needle. More recently, the term percutaneous electrical nerve stimulation (PENS) has been used (32-37) to describe a technique involving insertion of 32-gauge (ultra-fine) acupuncture needle probes into the soft tissues or muscles to electrically stimulate peripheral nerve fibers in the sclerotomal, myotomal, or dermatomal distribution corresponding to the patient's pain symptoms. The conceptual basis for PENS is related to both electroacupuncture (which involves electrical stimulation of percutaneously placed needle probes positioned at classical Chinese acupoints) and transcutaneous electrical nerve stimulation (via cutaneous electrodes positioned at the symptomatic dermatomes). Compared with the latter, the use of PENS has the advantage of

“bypassing” the resistance of the skin and delivering the electrical stimulus in closer proximity to the peripheral nerve endings located in the soft tissue, muscle, and/or periosteum in the affected region. For example, electrical stimulation of the nerve endings located in the periosteum appears to be an important factor in achieving PENS-induced analgesia in cancer patients with bony metastases (32).

Clinical experience with PENS in the treatment of acute herpes zoster infections suggested that it was not only effective in ameliorating acute zoster-related pain, but in expediting resolution of the cutaneous lesions (33). This prospective study also demonstrated that PENS therapy compared favorably to a standard antiviral drug regimen in terms of improving the patient's functional status and quality of sleep, as well as in decreasing the severity of postherpetic neuralgic symptoms. However, further studies evaluating long-term outcome are clearly needed.

More recent PENS studies have also demonstrated significant efficacy in the short-term management of back pain (Fig. 2), sciatica, headaches, and diabetic neuropathic pain (34–37). Although the published studies have clearly established the acute pain relieving effects and short-term improvement in physical activity and quality of sleep, the long-term benefits of PENS in patients with chronic pain disorders have not been established. Analogous to transcutaneous electrical nerve stimulation, the response to PENS is influenced by the location (38), frequency (39), and duration (40) of the electrical stimulation. The use of mixed stimulating frequencies (e.g., alternating 15 and 30 Hz) was more effective than either low or high frequencies alone (39). The optimal duration of the electrical stimulus with PENS has been determined to be 30–45 min. Of importance, more prolonged electrical stimulation does not appear to improve the analgesic effects (41).

Percutaneous Neuromodulation Therapy

Because the term PENS has been used in the past to describe a neurosurgical procedure involving implantation of temporary stimulating electrodes before an SCS device (31), White et al. (38) have recently proposed to change the name for the above-mentioned percutaneous electroanalgesic technique to percutaneous neuromodulation therapy (PNT). The term PNT was chosen because it more accurately describes the neurophysiologic basis for PENS-induced analgesia.

In a recent study involving patients with neck pain, White et al. (38) demonstrated that percutaneous electrical stimulation at the local (cervicothoracic) dermatomes was more effective in decreasing pain and improving physical activity and quality of sleep than remote (lumbosacral) dermatomal stimulation. Another recent study (data unreported) has compared

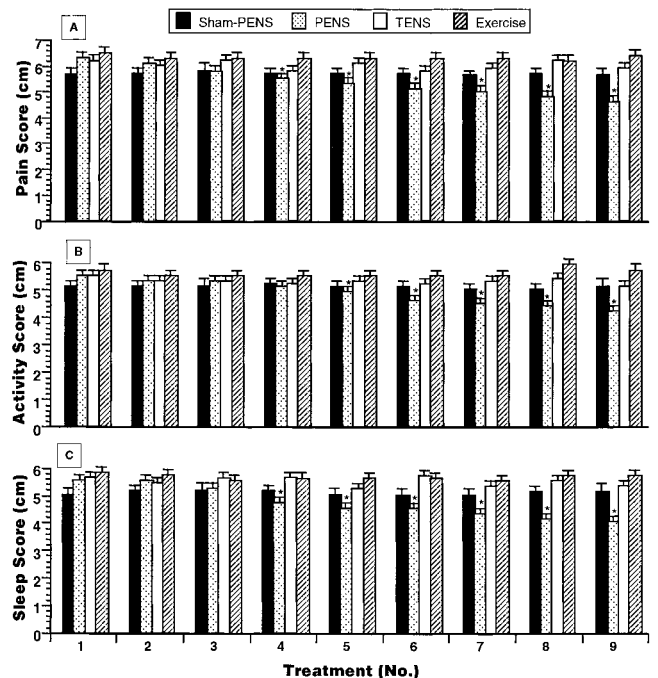


Figure 2. Visual analog scale scores for low back pain (A), physical activity (B), and quality of sleep (C) before each of the nine treatment sessions with the four study modalities. Values are means (\pm SEM). Asterisk indicates that the value is significantly different from the baseline value ($P < 0.03$). PENS = percutaneous electrical nerve stimulation, TENS = transcutaneous electrical nerve stimulation (34).

the acute analgesic effects of PNT in patients with low back pain in which the needles were positioned at differing depths in the lumbar region. The results of this pilot study suggested that a depth of 3 cm was more effective than superficial stimulation at <1 cm. Finally, a unique “sweep” pattern of electrical stimulation involving frequencies from 4 to 50 Hz has been developed in an attempt to slow the development of tolerance (habituation) to the electrical stimulus, and thereby to enhance patient comfort during PNT and potentially improve its efficacy. Because varying the frequency of the electrical stimulus has been alleged to decrease accommodation (i.e., adaptation) (2,3), tolerance to the analgesic effects produced by peripheral electrical stimulation may be reduced. However, controlled clinical studies are needed to validate this unique stimulation pattern when compared with standard fixed and alternating frequency patterns (e.g., 4, 15/30, 100 Hz).

Electroanalgesia

Electroanalgesia (EA) has gained increasing acceptance in Westernized medical practice. Although classical Chinese metaphysical theories invoking “Qi” and “Meridians” have been used to explain the analgesic

effects of EA, more recently it has been suggested that the analgesic effects of EA may be mediated through the central release of endogenous opioids (i.e., endorphins, enkephalins, and dynorphins) by the body's pain modulation system (42). EA has been reported to be more effective than traditional acupuncture (43) and compared favorably with PNS for pain relief (44). With EA, short-term success rates of up to 70% have been reported in patients with chronic low back pain, osteoarthritis, myofascial discomfort, and migraine headaches (45).

Transcutaneous Electrical Nerve Stimulation

Transcutaneous Electrical Nerve Stimulation (TENS) involves the transmission of electrical energy from an external stimulator to the peripheral nervous system via cutaneously placed conductive gel pads. TENS can be subclassified into two variants: 1) low-intensity (1-2 mA), high-frequency (50-100 Hz) TENS; and 2) acupuncture-like high-intensity (15-20 mA), low-frequency (1-5 Hz) or "dense-disperse" TENS (46). The purported mechanism of action of TENS invokes both spinal (i.e., gate-control, frequency-dependent blockade) and supraspinal theories (i.e., release of endogenous neuromediators). It has also been suggested that TENS involves activation of the body's pain modulation system and increases in the release of endogenous opioids within the central nervous system, thereby suppressing the transmission and perception of noxious stimuli from the periphery (46-48). However, more recent studies suggest that at least part of TENS-mediated hypoalgesia is a consequence of direct peripheral effects (49). Low-frequency TENS produces a local increase in cutaneous blood flow (50).

Early clinical studies evaluating the effect of TENS on postoperative analgesic requirements have produced inconsistent results. For many studies, the specific details regarding the stimulation variables were lacking. Carroll et al. (51) undertook a systematic review of 46 studies pertaining to the use of TENS for acute postoperative pain. Of these, only 17 were deemed to be well designed, and they reported no benefit of TENS over a placebo treatment. In studying the efficacy of TENS for acute labor pain, Carroll et al. (52) failed to find convincing evidence of pain reduction by using either primary (e.g., analgesic sparing) or secondary (e.g., preference for TENS for future labor analgesia) outcome measures.

More recent randomized controlled trials of TENS reported beneficial effects with regard to dental (53), lithotripsy (54), arthroscopy (Table 2) (55), hemorrhoidectomy, and hysterectomy pain (56-59). After thoracotomy procedures, 23% of the patients in an

Table 2. Comparative Effects of TENS and Sham TENS on Pain Scores, Oral Analgesic Requirements, and Range of Motion After Knee Arthroscopic Procedures (55)

	Control	Sham	TENS
Group size (<i>n</i>)	30	30	30
Mean pain score (cm)			
POD #1	5.8	5.5	4.8
POD #2	5.8	4.5	4.0
POD #3	4.1	3.1	2.9
Total	15.7	13.1	11.7*
Mean analgesic dosages (<i>n</i>)			
POD #1	3.6	3.3	2.8
POD #2	3.7	2.8	1.8
POD #3	1.9	1.7	1.3
Total	9.2	7.8	5.9*
Range of motion (degree)			
POD #7	103.2	102.0	111.8*
POD #21	119.4	112.4	124.4*
POD #49	119.3	119.6	130.0*

TENS = transcutaneous electrical nerve stimulation, POD = postoperative day.

* Significantly different from control, $P < 0.05$.

active TENS group required no parenteral opioid analgesic medication during the first 24 h postoperatively (60). In contrast, all of the patients in the sham group needed opioid analgesics after thoracic surgery. In another postthoracotomy study, TENS-treated patients had significantly lower pain scores, shorter stays in the recovery room, and better tolerance of chest physical therapy on both the first and second postoperative days (61). Finally, TENS was found to minimize the decrease in respiratory mechanics (Fig. 3) and reduce the incidence of pulmonary complications after upper abdominal surgery (62).

The proportion of patients who benefit from short-term and long-term administration of TENS therapy for chronic pain ranges from 50% to 80% and 6% to 44%, respectively (63). When TENS was used at either 110 or 4 Hz to treat ischemic pain, only the lower frequency demonstrated a significant analgesic effect (64). When TENS was used to treat chronic pain related to musculoskeletal disorders, only a small percentage of patients experienced long-lasting benefits (65). Although chronic pain relief after TENS therapy has been alleged to result from a "placebo" effect (e.g., chronic low back pain) (66), other studies in diabetic patients with peripheral neuropathy have demonstrated analgesic efficacy that clearly exceeded the placebo effect (67). In fact, Chabal et al. (68) reported that the long-term use of TENS in patients with chronic pain was associated with a 55% decrease in analgesic medication and a 69% reduction in the use of physical/occupational therapy. Fishbain et al. (69) also reported that long-term use of TENS improves not only pain but other outcome variables (e.g., return to work, social activities, need for other therapies).

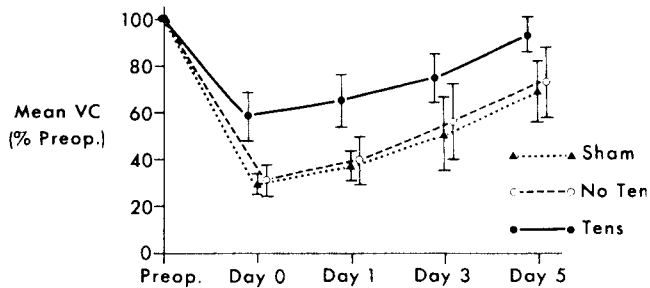


Figure 3. Effect of transcutaneous electrical nerve stimulation (TENS) on postoperative changes in lung vital capacity (VC) as a mean percentage (\pm SD) of the preoperative (baseline) measurements in patients undergoing major abdominal surgery procedures (62).

When TENS is used to treat chronic pain states, the slope of the dose-response curve is very gradual (i.e., the onset of its analgesic effect is slow) (70). The inconsistent analgesic responses to TENS therapy may be related to several factors, including variable stimulation sites (57), frequencies (58,71), intensities (59), and durations of electrical stimulation, as well as the patient's psychological profile (63). Experimental studies by Walsh et al. (72) have confirmed the importance of the TENS variables to the peripheral neurophysiologic effects of this modality and the resultant analgesia.

Transcutaneous Acupoint Electrical Stimulation

Transcutaneous Acupoint Electrical Stimulation (TAES) is a variant of TENS therapy that involves applying cutaneous electrodes at classical Chinese acupoints and stimulating with alternating high- and low-frequency electrical current ("dense-disperse") (59). Acupoint stimulation is as effective as dermatomal stimulation in producing an analgesic-sparing effect after lower abdominal surgery (57). In this study, TENS applied at either the incisional dermatomal level or the Zusanli acupoint conferred better postoperative analgesia than stimulation at a nonacupoint site (Fig. 4). Of interest, simultaneous stimulation at both an acupoint and incisional dermatomes resulted in a $>60\%$ reduction in the opioid analgesic requirement (59). This study also demonstrated that the intensity of electrical stimulation was an important determinant of the analgesic-sparing effect. High-intensity (9–12 mA) TAES was more effective in decreasing the postoperative opioid analgesic requirement than low-intensity (4–5 mA) TAES or sham stimulation.

H-Wave Therapy

H-Wave Therapy (HWT) is a form of electrical stimulation that produces a direct, localized effect on the

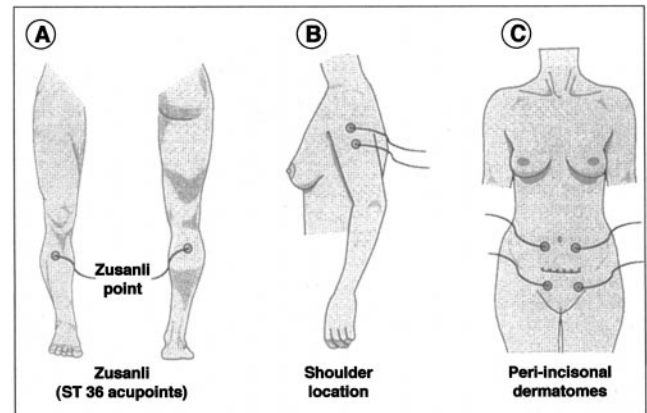


Figure 4. The locations of the transcutaneous electrical nerve stimulation pads in the sham and acupoint groups (A), in the nonacupoint group (B), and in the incisional dermatome group (C) (57).

conduction of underlying peripheral nerves (73). This electroanalgesic modality was originally recommended as an alternative to TENS for dental analgesia. Currently, it is being promoted for the treatment of acute musculoskeletal injuries, postoperative pain, and as a noninvasive form of local analgesia. Julka et al. (74) reported that transcutaneous electrotherapy using an H-wave machine was effective in 76% of diabetic patients with peripheral neuropathic pain. The subjective improvement in their neuropathic symptoms ($44\% \pm 4\%$) was sustained for an average of 1.7 ± 0.3 yr. The effectiveness of HWT may be augmented by concomitant use of antidepressant medication (e.g., amitriptyline) (75). In a recently published randomized, controlled trial involving a mechanical pain model, the analgesic effects of HWT were found to be short-lasting and similar to TENS therapy (76). Moreover, these investigators have previously reported that HWT fails to reduce experimental ischemic pain (77). The stimulating variables for HWT differ from TENS in that its signal comprises a fixed pulse duration of approximately 16 ms (vs 50–200 μ s), a frequency range of 2 to 60 Hz (vs 1–250 Hz), and a biphasic exponentially decaying (versus rectangular or square) waveform. It remains to be determined if these differences in the electrical stimulus produce differences in the analgesic response to HWT compared with conventional TENS therapy.

Interferential Current Therapy

Interferential Current Therapy (ICT) is another variant of TENS that uses the principle of amplitude modulation to decrease the discomfort of stimulating deeper tissues (e.g., muscle) when using transcutaneously applied electrical current. A combination of different stimulation frequencies are used (i.e., one fixed at 4 kHz and another within a variable range) to generate

frequencies between 4 and 250 Hz which are alleged to more effectively penetrate the soft tissues while producing less discomfort at the skin surface (78). With ICT, its postulated mechanism of analgesic action is through direct stimulation of muscle fibers rather than peripheral nerves, allegedly improving muscle blood flow and promoting the healing process. Of interest, neither ICT nor TENS affected either the nociceptive reflex or the H-reflex (79).

Although ICT is used widely in the physiotherapy and rehabilitative medicine settings, there is a dearth of rigorously controlled studies to justify its effectiveness in the management of either acute or chronic pain syndromes. Of interest, two recently published randomized, controlled trials involving ICT failed to demonstrate any additional analgesic effect compared with traditional (conservative) management of shoulder (80) and low back pain (81).

Piezo-Electric Current Therapy

Piezo-Electric Current Therapy (PECT) is an investigative analgesic technique based on the principle that mechanical deformation of a motorized piezoelectric ceramic rod produces a burst of 10 electrical pulses (five positive and five negative), each lasting 2-3 ms. Each electrical burst lasts for 50 to 250 ms (depending on the motor speed set) and generates a current of approximately 25 μ A. The application of PECT to the skin for 2 min produces a tolerable "pricking" pain sensation associated with a neurogenic inflammatory response lasting 3-4 h. This reaction is indicative of stimulation of A δ and C pain fibers, which can lead to depression of nociceptive afferent input via a neuromodulatory mechanism involving diffuse noxious inhibitory controls (82). The extent and duration of this inhibitory process is directly related to the intensity of the applied stimulus and is alleged to be associated with the release of endogenous endorphins (83). In a comparative trial involving conventional and acupuncture-like TENS (or TAES), the more noxious stimuli produced by PECT resulted in a greater analgesic effect in an experimental pain model (82).

Summary

Neuroaugmentation techniques have been used for many years to produce electroanalgesia. The techniques range from noninvasive (e.g., TENS, TAES), to minimally invasive (e.g., PENS/PNT, EA), to highly invasive (e.g., DBS, SCS) (Fig. 5). Recent modifications in the pattern of the electrical stimulus (e.g., sweep, dense-disperse, HWT, ICT, PECT) may further improve the analgesic efficacy of electroanalgesic techniques like TENS and PNT. However, more widespread application of electroanalgesia will depend on the results of well

SPECTRUM OF ELECTROANALGESIA

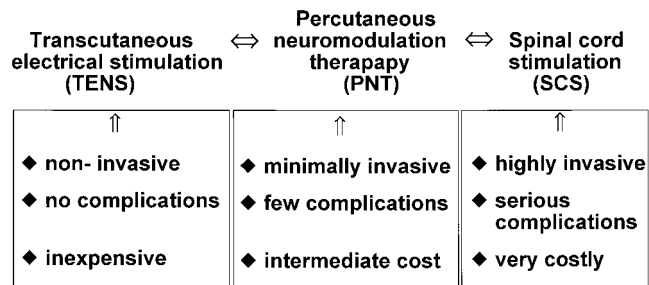


Figure 5. A wide spectrum of electroanalgesic techniques are available to supplement (or "complement") conventional analgesic modalities.

controlled, long-term outcome studies and the availability of Food and Drug Administration-approved stimulating devices. Other nonpharmacologic analgesic therapies (e.g., electromagnets, lasers) produce either nonsignificant or ambivalent analgesic effects in controlled clinical trials (84,85).

A more "in depth" understanding of the effect of different electrical stimulation patterns on the pain response may lead to further long-term benefits with electroanalgesic therapy. Future research into the neural modulatory and neurohumoral mechanisms responsible for electroanalgesia should lead to further improvements in these nonpharmacologic therapeutic modalities. Once the theoretical basis for electroanalgesia has been more clearly elucidated, it will be possible to optimize the efficacy of these "complementary" therapeutic options in the management of acute and chronic pain syndromes. In conclusion, a wide variety of electroanalgesic therapies are available for the management of both acute and chronic pain. Given the increasing interest among patients in unconventional (or "alternative") analgesic therapies (86), it behooves anesthesiologists and pain specialists to be aware of the potential benefits of these complementary therapies.

The authors thank Drs. Tijani Issioui and Margarita Coloma for their assistance in reviewing the manuscript, and Ms. Dolly Tutton for her secretarial assistance in preparing the manuscript.

References

1. Shealy CN, Mortimer JT, Reswick JB. Electrical inhibition of pain by stimulation of the dorsal column. *Anesth Analg* 1967; 46:489-91.
2. Melzack R, Wall PD. Pain mechanism: a new theory. *Science* 1965;150:971-9.
3. Pomerantz BA. Natural mechanisms of acupuncture analgesia. In: Lipton S, ed. *Persistent pain*. New York: Academic Press, 1981:271-5.

4. Han JS, Xie GX. Dynorphin: important mediator for electroacupuncture analgesia in the spinal cord of the rabbit. *Pain* 1984;18:367-76.
5. Tonelli L, Setti T, Falasca A, et al. Investigation on cerebrospinal fluid opioids and neurotransmitters related to spinal cord stimulation. *Appl Neurophysiol* 1988;51:324-32.
6. ten Vaarwerk IA, Staal MJ. Spinal cord stimulation in chronic pain syndromes. *Spinal Cord* 1998;36:671-82.
7. Kumar K, Toth C, Nath RK, Laing P. Epidural spinal cord stimulation for treatment of chronic pain: some predictors of success—a 15-year experience. *Surg Neurol* 1998;50:110-21.
8. Villavicencio AT, Leveque JC, Rubin L, et al. Laminectomy versus percutaneous electrode placement for spinal cord stimulation. *Neurosurgery* 2000;46:399-406.
9. Pineda A. Complications of dorsal column stimulation. *J Neurosurg* 1978;48:64-8.
10. Petrakis IE, Sciacca V. Spinal cord stimulation in critical limb ischemia of the lower extremities: our experience. *J Neurosurg Sci* 1999;43:285-93.
11. Turner JA, Loeser JD, Bell KG. Spinal cord stimulation for chronic low back pain: a systematic literature synthesis. *Neurosurgery* 1995;37:1088-96.
12. Lang P. The treatment of chronic pain by epidural spinal cord stimulation: a 15 year follow up—present status. *Axone* 1997;18:71-3.
13. Kemler MA, Barendse GA, van Kleef M, Egbrink MG. Pain relief in complex regional pain syndrome due to spinal cord stimulation does not depend on vasodilation. *Anesthesiology* 2000;92:1653-6.
14. Petrakis IE, Sciacca V. Does autonomic neuropathy influence spinal cord stimulation therapy success in diabetic patients with critical lower limb ischemia? *Surg Neurol* 2000;53:182-9.
15. Fanciullo GJ, Robb JF, Rose RJ, Sanders JH Jr. Spinal cord stimulation for intractable angina pectoris. *Anesth Analg* 1999;89:305-6.
16. Ceballos A, Cabezudo L, Bovaira M, et al. Spinal cord stimulation: a possible therapeutic alternative for chronic mesenteric ischaemia. *Pain* 2000;87:99-101.
17. North RB, Kidd DH, Piantadosi S. Spinal cord stimulation versus reoperation for failed back surgery syndromed: a prospective, randomized study design. *Acta Neurochir Suppl* 1995;64:106-8.
18. North RB, Kidd DH, Zahurak M, et al. Spinal cord stimulation for chronic, intractable pain: experience over two decades. *Neurosurgery* 1993;32:384-94.
19. Burchiel KJ, Anderson VC, Brown FD, et al. Prospective, multicenter study of spinal cord stimulation for relief of chronic back and extremity pain. *Spine* 1996;21:2786-94.
20. North RB, Kidd DH, Wimberly RL, Edwin D. Prognostic value of psychological testing in patients undergoing spinal cord stimulation: a prospective study. *Neurosurgery* 1996;39:301-10.
21. North RB, Kidd D, Davis C, et al. Spinal cord stimulation electrode design: a prospective randomized, controlled trial comparing percutaneous and laminectomy electrodes. *Stereotact Funct Neurosurg* 1999;73:134.
22. Bell GK, Kidd D, North RB. Cost-effectiveness analysis of spinal cord stimulation in treatment of failed back surgery syndrome. *J Pain Symptom Manage* 1997;13:286-95.
23. Heath RG, Mickle WA. Evaluation of seven years' experience with depth electrode studies in human patients. In: Ramey ER, O'Doherty DS, eds. *Electrical studies on the unanesthetized brain*. New York: Paul B. Hoeber, 1960:214-47.
24. Davis KD, Taub E, Duffner F, et al. Activation of the anterior cingulate cortex by thalamic stimulation in patients with chronic pain: a positron emission tomography study. *J Neurosurg* 2000;92:64-9.
25. Kumar K, Toth C, Nath RK. Deep brain stimulation for intractable pain: a 15-year experience. *Neurosurgery* 1997;40:736-47.
26. Richardson DE. Deep brain stimulation for the relief of chronic pain. *Neurosurg Clin N Am* 1995;6:135-44.
27. Tasker RR, Vilela Filho O. Deep brain stimulation for neuropathic pain. *Stereotact Funct Neurosurg* 1995;65:122-4.
28. Wall PD, Sweet WH. Temporary abolition of pain in man. *Science* 1967;155:108-9.
29. Hanai F. Effect of electrical stimulation of peripheral nerves on neuropathic pain. *Spine* 2000;25:1886-92.
30. Campbell JN, Long DM. Peripheral nerve stimulation in the treatment of intractable pain. *J Neurosurg* 1976;45:692-9.
31. North RB, Fischell TA, Long DM. Chronic stimulation via percutaneously inserted epidural electrodes. *Neurosurgery* 1977;1:215-8.
32. Ahmed HE, Craig WF, White PF, Huber P. Percutaneous electrical nerve stimulation (PENS): a complementary therapy for the management of pain secondary to bony metastasis. *Clin J Pain* 1998;14:320-3.
33. Ahmed HE, Craig WF, White PF, et al. Percutaneous electrical nerve stimulation: an alternative to antiviral drugs for acute herpes zoster. *Anesth Analg* 1998;87:911-4.
34. Ghoname EA, Craig WF, White PF, et al. Percutaneous electrical nerve stimulation for low back pain: a randomized crossover study. *JAMA* 1999;281:818-23.
35. Ghoname EA, White PF, Ahmed HE, et al. Percutaneous electrical nerve stimulation (PENS): an alternative to TENS in the management of sciatica. *Pain* 1999;83:193-9.
36. Ahmed HE, White PF, Craig WF, et al. Use of percutaneous electrical nerve stimulation (PENS) in the short-term management of headaches. *Headache* 2000;40:311-5.
37. Hamza MA, White PF, Craig WF, et al. Percutaneous electrical nerve stimulation (PENS): a novel analgesic therapy for diabetic neuropathic pain. *Diabetes Care* 2000;23:365-70.
38. White PF, Craig WF, Vakharia AS, et al. Percutaneous neuromodulation therapy: does the location of electrical stimulation effect the acute analgesic response? *Anesth Analg* 2000;91:949-54.
39. Ghoname EA, Craig WF, White PF, et al. The effect of stimulus frequency on the analgesic response to percutaneous electrical nerve stimulation in patients with chronic low back pain. *Anesth Analg* 1999;88:841-6.
40. Hamza MA, Ghoname EA, White PF, et al. Effect of the duration of electrical stimulation on the analgesic response in patients with chronic low back pain. *Anesthesiology* 1999;91:1622-7.
41. Andersson SA, Ericson T, Holmgren E, Lindquist G. Electroacupuncture: effects on pain threshold measured with electrical stimulation of teeth. *Brain Res* 1973;63:393-6.
42. Kishioka S, Miyamoto Y, Fukunaga Y, et al. Effects of a mixture of peptidase inhibitors (amastatin, captopril and phosphoramidon) on Met-enkephalin-, beta-endorphin-, dynorphin-(1-13)- and electroacupuncture-induced antinociception in rats. *Jpn J Pharmacol* 1994;66:337-45.
43. Saletu B, Saletu M, Brown M, et al. Hypnosis and acupuncture analgesia: a neurophysiological reality? *Neuropsychobiology* 1975;1:218-42.
44. Wang J, Mao L, Han JS. Antinociceptive effects induced by electroanalgesia and transcutaneous electrical nerve stimulation in the rat. *Int J Neurosci* 1992;65:117-29.
45. Ulett GA, Han SP, Han JS. Electroacupuncture: mechanisms and clinical application. *Biol Psychiatry* 1998;44:129-38.
46. Han JS, Chen XH, Sun SL, et al. Effect of low- and high-frequency TENS on Met-enkephalin-Arg-Phe and dynorphin A immunoreactivity in human lumbar CSF. *Pain* 1991;47:295-8.
47. Facchinetti F, Sandrini G, Petraglia F, et al. Concomitant increase in nociceptive flexion reflex threshold and plasma opioids following transcutaneous nerve stimulation. *Pain* 1984;19:295-303.
48. Gao M, He LF. Increase of mu opioid receptor density in rat central nervous system following formalin nociception and its enhancement by electroacupuncture. *Acta Physiologica Sinica* 1996;48:125-31.
49. Walsh DM, Lowe AS, McCormack K, et al. Transcutaneous electrical nerve stimulation: effect on peripheral nerve conduction, mechanical pain threshold, and tactile threshold in humans. *Arch Phys Med Rehabil* 1998;79:1051-8.

50. Cramp AF, Gilsean C, Lowe AS, Walsh DM. The effect of high- and low-frequency transcutaneous electrical nerve stimulation upon cutaneous blood flow and skin temperature in healthy subjects. *Clin Physiol* 2000;20:150-7.
51. Carroll D, Tramer M, McQuay HJ, et al. Randomization is important in studies with pain outcomes: systematic review of transcutaneous electrical nerve stimulation in acute postoperative pain. *Br J Anaesth* 1996;77:798-803.
52. Carroll D, Tramer M, McQuay HJ, et al. Transcutaneous electrical nerve stimulation in labor pain: a systematic review. *Br J Obstet Gynaecol* 1997;104:169-75.
53. Estafan DJ, Dussetschleger F, Schulman A. Clinical evaluation of cell demodulated targeted electronic anesthesia. *J Clin Dent* 1998;9:34-8.
54. Reichelt O, Zermann DH, Wunderlich H, et al. Effective analgesia for extracorporeal shock wave lithotripsy: transcutaneous electrical nerve stimulation. *Urology* 1999;54:433-6.
55. Jensen JE, Conn RR, Hazelrigg G, Hewett JE. The use of transcutaneous neural stimulation and isokinetic testing in arthroscopic knee surgery. *Am J Sports Med* 1985;13:27-33.
56. Chiu JH, Chen WS, Chen CH, et al. Effect of transcutaneous electrical nerve stimulation for pain relief on patients undergoing hemorrhoidectomy: prospective randomized controlled trial. *Dis Colon Rectum* 1999;42:180-5.
57. Chen L, Tang J, White PF, et al. The effect of location of transcutaneous electrical nerve stimulation on postoperative opioid analgesic requirement: acupoint versus nonacupoint stimulation. *Anesth Analg* 1998;87:1129-34.
58. Hamza MA, White PF, Ahmed HE, Ghoname EA. Effect of the frequency of transcutaneous electrical nerve stimulation on the postoperative opioid analgesic requirement and recovery profile. *Anesthesiology* 1999;91:1232-8.
59. Wang BG, Tang J, White PF, et al. Effect of the intensity of transcutaneous acupoint electrical stimulation on the postoperative analgesic requirement. *Anesth Analg* 1997;85:406-13.
60. Rooney SM, Jain S, Goldiner PL. Effect of transcutaneous nerve stimulation on postoperative pain after thoracotomy. *Anesth Analg* 1983;62:1010-2.
61. Warfield CA, Stein JM, Frank HA. The effect of transcutaneous electrical nerve stimulation on pain after thoracotomy. *Ann Thorac Surg* 1985;39:462-5.
62. Ali J, Yaffe CS, Serrette C. The effect of transcutaneous electric nerve stimulation on postoperative pain and pulmonary function. *Surgery* 1981;89:507-12.
63. Lampl C, Kreczi T, Klingler D. Transcutaneous electrical nerve stimulation in the treatment of chronic pain: predictive factors and evaluation of the method. *Clin J Pain* 1998;14:134-42.
64. Walsh DM, Liggett C, Baxter D, Allen JM. A double-blind investigation of the hypoalgesic effects of transcutaneous electrical nerve stimulation upon experimentally induced ischaemic pain. *Pain* 1995;61:39-45.
65. Robinson AJ. Transcutaneous electrical nerve stimulation for the control of pain in musculoskeletal disorders. *J Orthop Sports Phys Ther* 1996;24:208-26.
66. Deyo RA, Walsh NE, Martin DC, et al. A controlled trial of transcutaneous electrical nerve stimulation (TENS) and exercise for chronic low back pain. *N Engl J Med* 1990;15:323:1423-5.
67. Kumar D, Marshall H. Diabetic peripheral neuropathy: amelioration of pain with transcutaneous electrostimulation. *Diabetes Care* 1997;20:1702-5.
68. Chabal C, Fishbain DA, Weaver M, Heine LW. Long-term transcutaneous electrical nerve stimulation (TENS) use: impact on medication utilization and physical therapy costs. *Clin J Pain* 1998;14:66-73.
69. Fishbain DA, Chabal C, Abbott A, et al. Transcutaneous electrical nerve stimulation (TENS) treatment outcome in long-term users. *Clin J Pain* 1996;12:201-14.
70. McQuay HJ, Moore RA, Eccleston C, et al. Systematic review of outpatient services for chronic pain control. *Health Technol Assess* 1997;1:1-135.
71. Bowman BR, Baker LL. Effects of waveform parameters on comfort during transcutaneous neuromuscular electrical stimulation. *Ann Biomed Eng* 1985;13:59-74.
72. Walsh DM, Foster NE, Baxter GD, Allen JM. Transcutaneous electrical nerve stimulation: relevance of stimulation parameters to neurophysiological and hypoalgesic effects. *Am J Phys Med Rehabil* 1995;74:199-206.
73. McDowell BC, Lowe AS, Walsh DM, et al. The effect of H-wave therapy upon conduction in the human superficial radial nerve *in vivo*. *Exp Physiol* 1996;5:821-32.
74. Julka IS, Alvaro M, Kumar D. Beneficial effects of electrical stimulation on neuropathic symptoms in diabetes patients. *J Foot Ankle Surg* 1998;37:191-4.
75. Kumar D, Alvaro MS, Julka IS, Marshall HJ. Diabetic peripheral neuropathy: effectiveness of electrotherapy and amitriptyline for symptomatic relief. *Diabetes Care* 1998;21:1322-5.
76. McDowell BC, McCormack K, Walsh DM, et al. Comparative analgesic effects of H-wave therapy and transcutaneous electrical nerve stimulation on pain threshold in humans. *Arch Phys Med Rehabil* 1999;80:1001-4.
77. McDowell BC, Lowe AS, Walsh DM, et al. The lack of hypoalgesic efficacy of H-wave therapy on experimental ischaemic pain. *Pain* 1995;61:27-32.
78. Goats GC. Interferential current therapy. *Br J Sports Med* 1990;24:87-92.
79. Cramp FL, Noble G, Lowe AS, et al. A controlled study on the effects of transcutaneous electrical nerve stimulation and interferential therapy upon the RIII nociceptive and H-reflexes in humans. *Arch Phys Med Rehabil* 2000;81:324-33.
80. van der Heijden GJMG, Leffers P, Wolters PJMC, et al. No effect of bipolar interferential electrotherapy and pulsed ultrasound for soft tissue shoulder disorders: a randomized controlled trial. *Ann Rheum Dis* 1999;58:530-40.
81. Werners R, Pynsent PB, Bulstrode CJ. Randomized trial comparing interferential therapy with motorized lumbar traction and massage in the management of low back pain in a primary care setting. *Spine* 1999;24:1579-84.
82. Danziger N, Rozenberg S, Bourgeois P, et al. Depressive effect of segmental and heterotopic application of transcutaneous electrical nerve stimulation and piezo-electric current in lower limb nociceptive flexion reflex in human subjects. *Arch Phys Med Rehabil* 1998;79:191-200.
83. Willer JC, Le Bars D, De Broucker T. Diffuse noxious inhibitory control in man: involvement of an opioidergic link. *Eur J Pharmacol* 1990;182:347-55.
84. Nyholm A, Thorsen H, Lonnberg F. The effect of low-level laser therapy on musculoskeletal pain: a meta-analysis. *Pain* 1993;52:63-6.
85. Gross AR, Aker PD, Goldsmith CH, Peloso P. Physical medicine modalities for mechanical neck disorders. *Cochrane Database Syst Rev* 2000:CD000961.
86. Eisenberg DM, Davis RB, Ettner SL, et al. Trends in alternative medicine use in the United States, 1990-1997: results of a follow-up national survey. *JAMA* 1998;280:1569-75.