



New Technique Combines **Electrical Currents** and **Local Anesthetic** for Pain Management

Combined electrochemical nerve block reduced pain in 80% of patients with neuropathies and 50% of patients with intractable back pain.

Robert H. Odell, Jr., MD, PhD
Interventional Pain Management
Las Vegas, NV

Richard Sorgnard, PhD
Morhea Technologies
Las Vegas, NV

Numerous types of electrical currents are offered in modern electromedicine. The plethora of currents is made possible by varying the frequency, amplitude (intensity), and direction of the current in time. Within these electric current parameters are distinct and varying physiologic and therapeutic effects for the human biosystem.

Typically, therapeutic electric currents are classified according to their frequencies—for example, low frequency (LF; <2,000 Hz), medium frequency (MF; 2,000-100,000 Hz), or high frequency (HF; >100,000 Hz). This therapeutic classification system appears to originate from numerous physiologic investigations made in the last century.¹⁻⁵ In the human biosystem, LF and MF currents are used for therapeutic stimulation of excitable cells (receptors, nerves, and muscles). Depending on the stimulating frequency delivered, physiologic and therapeutic actions may occur

that may include vasodilatation, vasoconstriction, analgesia, activation of regeneration, and facilitation of metabolism.

This article describes a new electromagnetic device and its use in combination with local anesthetic therapy to treat pain problems.

New Advanced Technology

The vast majority of electromedical devices available in the United States employ LF stimulation (eg, transcutaneous electrical nerve stimulation [TENS]). Balanced MF currents have been developed that produce twice the electrical current with no electrical charge. A new type of electrical current technology has been developed to enhance the stimulating lower frequencies and nonstimulating middle frequencies for increased efficacy in clinical practice. The device also combines, and simultaneously delivers,

frequency-modulated (FM) and amplitude-modulated (AM) electric cell currents in the MF range. We refer to this electromedical approach as electronic signal treatment (EST).

This new technology may reach deeper into tissue structures with simultaneous modulation of amplitude and frequency between 2,500 Hz and 33,000 Hz. It is also capable of modulating its MF electric cell-signaling current down into the LF range at available frequency rates between 0.1 and 999 Hz.

In addition, we have combined the new EST with local anesthetic injections (bupivacaine 0.25%) with clinical success. This technique provides a combined (electrical and chemical) nerve block that enhances treatment of a neuropathy or a painful condition (see Tables 1 and 2, page 63). According to the *Gould Medical Dictionary*, a nerve block is defined as “[t]he interruption of the passage of impulses through a nerve, as by chemical, mechanical, or electrical means.” Because nerve blocks occur at voltage-gated channels, all nerve blocks are essentially electrical. According to Szasz, “There is no such thing as a chemical block ... only an electrical block.”⁶ We refer to this as combined electrochemical block (CEB).

Clinical Experiences

It is the experience of the authors that pain is reduced by CEB in about 80% of patients who have neuropathies. As shown in Figure 1 (page 64), 16 patients with neuropathies improved over a course of 20 treatments. The CEB also has worked well in many cases of failed spine fusion syndrome and failed back surgery syndrome. In a small series of patients, more than 50% of those with hardware and intractable pain and proprioception difficulties showed improvement with bilateral transforminal epidural bupivacaine

Table 1. What Do Electrical Currents and Local Anesthetics Accomplish?

Electrical—sustained depolarization
Neuron blockade
• Afferent blocks results in less perceived pain
• Less pain, local muscle relaxation
• Relaxation, more circulation
• More circulation
• More nutrients/enzymes/hormones
• Less toxic metabolites
• Efferent blocks result in local vasodilation
• More circulation
• More nutrients/enzymes/hormones
• Less toxic metabolites
• Less neurogenic inflammation
Chemical—hyperpolarization
Neuron blockade
• Afferent blocks results in less perceived pain
• Less pain, local muscle relaxation
• Relaxation, more circulation
• More circulation
• More nutrients/enzymes/hormones
• Less toxic metabolites
• Efferent blocks result in local vasodilation
• More circulation
• More nutrients/enzymes/hormones
• Less toxic metabolites
• Less neurogenic inflammation

They achieve exactly the same physiologic results!^a

^a Special thanks to James Woessner, MD, PhD, for his help in the creation of this table.

Continued on Page 63 >>

Table 2: Nerve Fiber Types and Nerve Blocking

Fiber Type	Function	Diameter (microns)	Myelination	Conduction Velocity (m/s)	Sensitivity to Nerve Block
Type A					
Alpha (α)	Proprioception, motor	12-20	Heavy	70-120	+
Beta (β)	Touch, pressure	5-12	Heavy	30-70	++
Gamma (γ)	Muscle spindles	3-6	Heavy	15-30	++
Delta (δ)	Pain, temperature	2-5	Heavy	12-30	+++
Type B	Preganglionic autonomic	<3	Light	3-15	++++
Type C					
Dorsal root	Pain	0.4-12	None	0.5-2.3	++++
Sympathetic	Postganglionic	0.3-1.3	None	0.7-2.3	++++

- Pain practitioners block the nerves transmitting pain impulses (Type A- δ , Type C)
- Lower concentrations of local anesthetic will only block the small unmyelinated and lightly myelinated (Type C and Type A- δ) fibers
- Middle-frequency currents (2,000-20,000 Hz) block smaller unmyelinated (Type C) and small myelinated (Type A- δ) fibers
- Larger fibers (Type A- α , β , γ) require high-amplitude currents and are usually spared in electrical, low-dose chemical (eg, labor epidural) blocks

Continued from Page 53 >>

injections along with the application of EST.⁷ In fact, CEB may be considered a less invasive alternative to spinal cord stimulator implantation.

Case Report

A 73-year-old woman had a 15-year history of low back pain with pain radiation down the legs. Diagnostic studies revealed spinal stenosis and facet arthropathy. She received three CEBs and two ESTs weekly for 3 weeks. After a pause of several days, she received two CEBs and three EST treatments over 2 weeks. At the end of this time, her visual analogue scale pain score had dropped from 10 of 10 to 2 of 10, and she was able to leave her house and walk with her husband.

Electromagnetic Physics of the Body

Why does this work? One major difference between the electric cell-signaling currents in EST and older devices is that EST allows greater depth of penetration through the dermal tissue by overall lowering of impedance to higher-frequency currents. This unique multiplex signaling configuration of mixed higher MF with overriding lower stimulatory frequencies (combined FM and AM signals) allows for the optimum voltage necessary to achieve proper depth of penetration while using lowered therapeutic response frequencies to affect the voltage-gated channels and receptors within target tissue (see Table 2).

By continually varying the primary medium frequency or using the frequency-modulated signals in a higher

range, we can overcome the natural resistance to different tissue structures. Examples of these different resistant values are typically measured in ohms. The lowest resistance (impedance) is, in fact, neural tissue (1,000 Ω), so most (>65%) of the current (energy) will be drawn to the nerves.

Stimulatory effects are defined as the physiologic effects that appear from the use of lower electric current frequencies, which produce repeated action potentials (impulses) in excitable cells (LF). The induced membrane depolarization and subsequent repolarization produce a number of mechanisms of action known to be effective in treatment by varying the stimulation frequency. These include analgesia from the principle of counter-irritation; analgesia from

Average Pain Scores Reduction for 16 Patients with Neuropathies

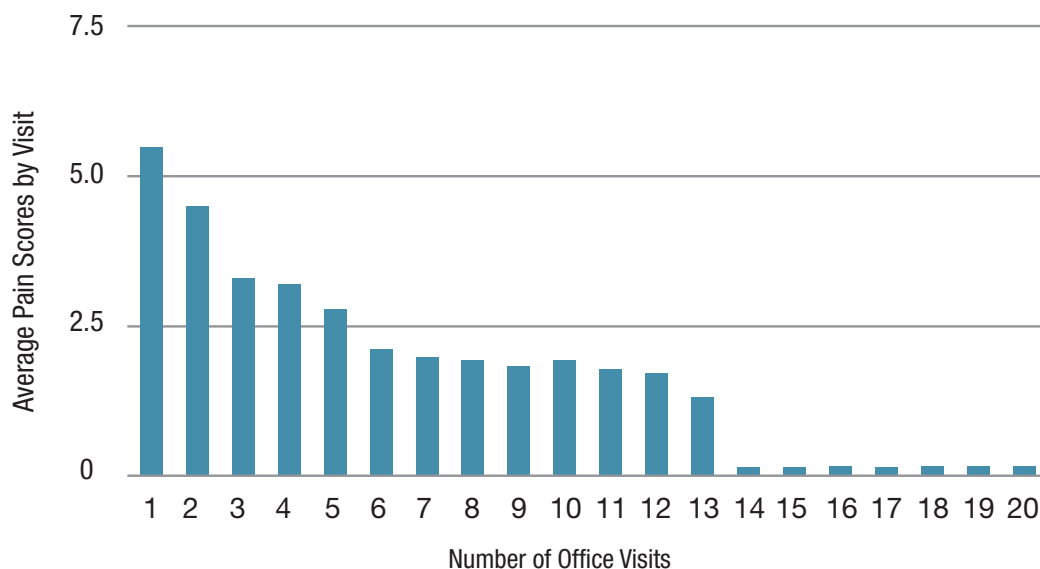


Figure 1. Patients had diabetic or other neuropathies and had combined electric current and local anesthetic treatments. Each patient had a total of 20 clinic visits with varying combinations.

stimulated neuropeptide release (eg, β -endorphin, enkephalin); enhanced circulation; sympathetically mediated vasoconstriction (detumescence effects); sympathetically mediated vasodilatation (antispasmodic effects); muscle activation, training, and strengthening; excitation of sprouting nerve axon processes; and an overall influence on the metabolism.⁸⁻¹²

Facilitatory effects are defined as the varied and multiple physiologic effects (biochemical changes) that appear from the use of higher MF electric currents directly or indirectly, but *not* occurring from the repeated production of any action potentials. There are a number of physiologic changes and biochemical actions that can be seen from the use of these higher MF signals and their resonance components. Examples include analgesia from balancing the metabolite concentration differences (pH); analgesia from second-messenger formation (cyclic AMP [cAMP]-mediated membrane repair processes); analgesia

from nerve (pain) fiber blockade via reactive depolarization; vessel vasoconstriction via contraction of the vessel wall smooth muscle; anti-inflammatory activity by activating filtration/diffusion processes;¹¹ edema management, activation of regeneration and support by second messenger formation (eg, cAMP); immune system activation and support via improved intercellular communication; and the general facilitation of metabolism.¹¹⁻¹⁴

These multiplexed and varied medium frequency signals (MFs) have a direct affect on voltage-dependent gates, and the alteration in the membrane physiology is objectively measurable. A number of scientific citations demonstrate both conformational changes in the G proteins of the cell membrane and subsequent second messenger formation (directing cell-specific activity) within the cell at different voltage gates when exposed to frequency and resonance-specific MF electric signal currents.¹¹⁻¹⁸ By electrically blocking the

severe pain-firing nerves in patients, we can obtain instantaneous pain suppression (electric nerve block) (see Figure 2).

Avoidance of Nerve Accommodation

The definition of nerve accommodation is the ability of nerve tissue to adjust to a constant source and intensity of stimulation, so that some change in either intensity or duration of the stimulus is necessary to elicit a response beyond the initial reaction. Accommodation is probably caused by reduced sodium ion permeability, which results in an increased threshold intensity and subsequent stabilization of the resting membrane potential.^{15,19,20} Nerve accommodation breakdown has been documented as a characteristic of electric nerve blocks.²¹

Unlike other present-day electroanalgesia technologies (eg, TENS, interferential current, MF scanning), it is not possible for the nerve to accommodate during treatment with EST. This is because the specific parameters of the

Electric Nerve Block to the Sciatic Nerve

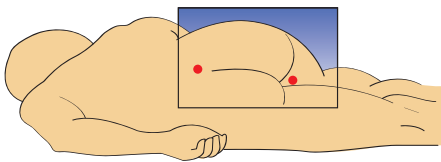
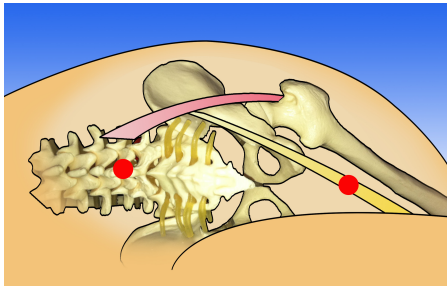


image courtesy of Dr. Odell

Figure 2. Electrical current preferentially follows the parts of least impedance,¹ which integrates distance between electrodes and conductivity of the intervening tissues.

¹ Impedance is resistant to alternating current; physiologically significant values are: nerve—1,000 Ω ; vascular tree—3,500 Ω ; bone—160,000 Ω .

primary MFs, signal lower-modulation frequencies, frequency sweep-step rate, dwell times at each given frequency, intensity of the electric cell signal and harmonic frequencies, and overall type of modulation are constantly changing. The desired parameters to elicit specific mechanisms of physiologic action for treating specific medical conditions are programmed into the treatment algorithms. The constant varying of frequency and intensity are then delivered simultaneously, individually, or alternately to give the body exposure to the greatest number of electric cell-signaling events.

Through the computer-assisted digital manipulation of higher primary EST frequencies at specific intervals, a slower, controlled modulation frequency rate with varied intensities (dosage) is superimposed on the primary frequency. This controlled modulation rate can be varied to match and target types and subsets of ion voltage-gated channels at the cell membrane. It is particularly

useful in assisting the abnormal nerve in returning to more steady-state natural firing frequencies.^{15,22}

Physiologic Effects

There are a number of physiologic and therapeutic actions induced by electric currents in treating various medical conditions (see Table 3, page 66). They are briefly summarized here.

Analgesia: Effects on the Diminution of Pain

There are several mechanisms that explain the mechanism of analgesia:

- Under the influence of rapidly alternating polarity electrical signal energy fields, ion movement is enhanced, and this tends to balance high-concentration differences in metabolites; these effects promote pH normalization and reduction in tissue acidosis;
- Second messenger formation (cAMP) directs all cell-specific activity toward cell membrane

repair, inhibiting arachidonic acid release from insulted membranes and subsequent prostaglandin (pain mediator) cascade;

- Specific electric signal energy parameters produce repeated excitation of afferent nerve fibers, affecting neuronal signaling processes in the central nervous system (CNS) and interfering with local pain perception (gate-control theory);
- Electric cell signaling assists in cell receptor uptake of β -endorphin, enkephalin, and phyllokinin, which modulate or inhibit pain impulses in the CNS; and
- The application of higher-dose, higher-frequency EST electric cell signals fall within the absolute refractory period of the cell membrane, inducing a sustained depolarized state across multiple nodes of Ranvier and inhibition (block) of axon information (pain signal) transport.^{11-15,19,20,23-25}

Table 3: Therapeutic Effects of Simultaneous Variations of Frequency and Amplitude of Electric Currents

Analgesia
Circulatory and Lymphatic Flow
Edema Reduction
Increased Metabolism
Regeneration of Tissue
Muscle Stimulation
Immune Support
Anti-inflammatory Action

Circulatory and Lymphatic Flow

Signaling cAMP leads to the opening of voltage-gated channels in efferent C-fibers of pain neurons and the sympathetic nervous system. Specific parameters of electric cell-signaling energy cause a fatiguing response, which induces sympathetically mediated vasodilatation (after brief vasoconstriction) via the depletion of the synaptic neurotransmitter (norepinephrine). Vessels then vasodilate, which increases local circulation to allow incoming nutrients and the flushing out of metabolic waste products. This cascade will help to eliminate the primary biochemical cause of local pain (peripheral sensitization). In addition, signaling cAMP leads to decreased afferent C-fiber firing, which in turn decreases ephaptic cross firing of afferent A- δ fibers. Use of cAMP by opening voltage-gated channels can likely produce physiologic normalization.^{12-15,26}

Edema Reduction

The multiple mechanisms of action that are induced by varied EST

electric cell signaling energy actively promote management of edema, including edematous tissue repair, enhanced filtration and diffusion processes, and pain and inflammation mediator redistribution. Mechanisms include specific vasoconstrictive EST electric cell-signaling frequencies that enhance centripetal transport of venous blood and lymph via sympathetic stimulation.

Increased Metabolism

EST energy triggers cAMP from increased adenosine triphosphate (ATP) production and improves cell respiration via ion transport (membrane permeability). EST energy produces a hormone-like effect by triggering an electrical conformation change to the cell membrane G protein. This influences adenylate cyclase activity, resulting in the formation of the second messenger cAMP, which is known to direct cell-specific activity, including cellular repair processes. cAMP-induced repair processes are necessary to stabilize (normalize) the cell membrane and

inhibit continued leakage of acids known to trigger pain and inflammation mediators.²⁷ This process may play the most critical role toward normalization of cell function.^{11,13,15,28,29}

Regeneration of Tissue

Specific parameters and dosage of stimulative EST frequencies will produce a response inducing excitation of sprouting axon processes at three to five times the normal regeneration rate of 1 to 3 mm per day via repeated action potential propagation (maximum neuron signaling without neurotransmitter depletion).^{30,31}

Muscle Stimulation, Activation, and Facilitation

Specific MF EST electric cell signals can be employed at higher than the motor firing threshold to activate muscle fibers directly via sustained depolarization with minimal motor neuron involvement.

Specific LF stimulative EST electric cell signals can be employed to activate the oxidative muscle metabolism and enzyme synthesis for better oxidative metabolic adaptation, contractile substance increase, and improved capillary regeneration (neovascularization). Neuromuscular effects include imitative activation, endurance training, thrombosis prevention, strengthening, and relaxation (spasmolysis).

Immune System Support

EST electric cell-signaling energy appears to improve and support the immune system by improving gap-junction intercellular communication. Gap junctions are protein-lined channels that directly link the cytosol of one cell with another adjacent cell, providing a passageway for movement of very small molecules and ions between the cells.^{26,30-32}

EST energy influences the

electrically charged ion movements through gap junctions by increasing the transport through the cell to cell canals and by facilitating intercellular electric and chemical communication and metabolic cooperation.^{11,26,33} EST energy fields contribute to a functional improvement in tissues that are dysfunctional—for example, in the healing phase of injured tissue and in degenerative tissue changes, metabolic conditions, edema, and areas of regional insulted tissue.

Anti-inflammatory Effects

EST energy works through specific biosystems and their controls via multiple mechanisms (listed above) by causing initial inflammation facilitation and then quick resolution of the inflammatory process, preventing it from leading to chronic inflammation and chronic pain.¹¹

Safety

Extensive use over the past 15 to 20 years has established a very low risk profile of treatment with electric currents, even with these complex waveforms. The addition of local anesthetic blocks adds only an incremental risk (for infection). In the personal experience of one of the authors, the only adverse effect noted among a few hundreds patients was slightly increased pain, probably due to over-stimulation or excessive electric signal energy (dosage), easily corrected in subsequent treatments. We also had one minor (first-degree) burn due to excessive application of electric signal energy (power density) to a poorly hydrated adhesive electrode. The burn resolved itself without additional medical intervention. The chances for increased expenses to the patient and third party payers treated with EST because of iatrogenic consequences are minute.

Conclusion

History has clearly shown that electric current devices have treatment merit. In most cases, electrical devices have, unfortunately, been proven to produce temporary patient improvement. It is now evident that complex, painful conditions need more than one treatment modality or an alternation of multiple and different mechanisms of action to sustain long-term patient treatment success.

We believe that longer-lasting outcomes can be achieved by using AM and FM currents at prescribed MF and LF parameters. When combined with a local anesthetic blocking agent, better results can be obtained. This procedure combines the positive benefits of intermittently generated membrane sustained depolarization, interruption of the pain signal along the axon, normalized second messenger

(cAMP) levels, β -adrenergic response, circulatory vasodilatation, general relaxation effects, and endogenous opiate release. The combined use of electric currents and local anesthetic should be more widely investigated in clinical practice. ■

Authors' Bios and Disclosure: *The device employed in this review was the NeoGen-Series available through Sanexas Corporation, Las Vegas, NV.*

Robert H. Odell, Jr., MD, PhD (biomedical engineering), practices interventional pain management and anesthesiology in Las Vegas, NV. Dr. Odell is a diplomat of the American Board of Anesthesiology, the American Board of Pain Medicine, and the American

Academy of Pain Management. He is a fellow of Interventional Pain Practice (FIPP) from the World Institute of Pain.

Over the past 6 to 7 years, Dr. Odell has been working in the clinical implementation of these advanced electro-medical cell-signaling devices, which produce salutary effects for many of the most refractory pain management challenges, especially neuropathy.

Richard Sorgnard, PhD (molecular biology), is Executive Director for Morhea Technologies LLC, which is responsible for the design, development, and engineering of the electric cell-signaling technology used in this device. Dr. Sorgnard continues his life's work in the signal generation and electromedical field, which began with his earlier

association at the National Security Agency (NSA) in Laurel, MD.

Dr. Odell has no financial information to disclose. Dr. Sorgnard has disclosed that he is a consultant for Sanexas International GmbH in Blaustein, Germany. Morhea Technologies LLC is involved in the ongoing research and technical development of electronic signal generation devices for the medical industry on a fee-for-service basis, including Sanexas International, GmbH.

Dr. Sorgnard's wife is currently involved with the management at Sanexas and has stock ownership. Dr. Sorgnard does not own stock nor does he have any direct management involvement with Sanexas International GmbH or its distribution network.

References

- Wyss OA. New principle of electric stimulation: ambipolar stimulation by alternating current, purely sinusoidal, of middle-frequency. *Experientia*. 1962;18:341-342.
- Wyss OA. Nerve stimulation with middle-frequency current pulses. *Helv Physiol Pharmacol Acta*. 1967;25(1):85-102.
- May HU. Simultaneous Modulation of frequency and amplitude modulated electric signals. *European Journal of Physiology*. 2002;443 (suppl. 1):53-59.
- Dalziel C. Effect of frequency on let-go currents. *IEEE Trans Elect Eng*. 1943;62:745-749.
- Chatterjee I. Human body impedance and threshold currents for perception and pain for contact hazard analysis in the VLF-MF Band. *IEEE Trans Bio Eng*. 1986;33(5):486-494.
- Szasz A. Presentation at the Clinical Electromedical Research Academy meeting; 1997; Las Vegas, NV.
- Odell R. The Integrated Nerve Block: Electrical + Chemical; Poster Presentation to the 18th Annual International Spine Intervention Society Meeting; July 23-26, 2010; Las Vegas, NV.
- Melzack W. Pain mechanisms: a new theory. *Science*. 1965;150:171-179.
- Burton C, Maurer DD. Pain suppression by transcutaneous electrical nerve stimulation (TENS) in chronic pain. *IEEE Trans Biomed Eng*. 1974;21(2):81-88.
- Bonica J, ed. *The Management of Pain*. 2nd ed. Philadelphia, PA: Lea and Febiger; 1990.
- Odell R, Sorgnard R. Anti-inflammatory effects of electronic signal treatment. *Pain Physician*. 2008;11(6):891-907.
- Odell R, Sorgnard R, May HU. Electroanalgesic nerve block. *Pract Pain Manag*. 2006;6(3):42-54.
- Savery F, Silver F, Edward R, et al. Assessment of electric differential treatment (EDIT) and Endosan treatment for ovarian cysts and concomitant symptoms. *Adv Ther*. 1991;8(5):243-249.
- Schwartz RG. Electric sympathetic block: current theoretical concepts and clinical results. *J Back Musculoskelet Rehabil*. 1998;10:31-46.
- Schwartz RG. Electric sympathetic block. *Osterr Z Phys Med Rehabil*. 2006;16(1):3-10.
- Blank M. *Electricity and Magnetism in Biology and Medicine*. Berkeley, CA: San Francisco Press; 1992.
- Frey A. *On the Nature of Electromagnetic Field Interactions With Biological Systems*. New York, NY: Springer; 1995:99-126.
- Catarsi S, Scuri R, Brunelli M. Cyclic AMP mediates inhibition of the Na(+)-K(+) electrogenic pump by serotonin in tactile sensory neurons of the leech. *J Physiol*. 1993;462:229-242.
- Bowman B. *Electrical block of peripheral motor activity*. Downey: Rancho Los Amigos Rehabilitation Engineering Center, 1981:89-102.
- Woessner J. Electric nerve block. In: Boswell M, Cole E, eds. *Weiner's Pain Management*. Boca Raton, FL: Taylor & Francis; 2006:1233-1242.
- Kristian H, Arendt-Nielsen L, Andersen O. Breakdown of accommodation in nerve: a possible role for persistent sodium current. *Theor Biol Med Model*. 2005;2:16.
- Liboff A. Signal shapes in electromagnetic therapies: a primer. In: Rosche PJ, ed. *Bioelectromagnetic Medicine*. New York, NY: Marcel Dekker; 2004:17-38.
- Bhadra N, Kilgore KL. High-frequency nerve conduction block. *Conf Proc IEEE Eng Med Biol Soc*. 2004;7:4729-4732.
- Bhadra N, Kilgore KL. Block of mammalian motor nerve conduction using high frequency alternating current. Presented at: 10th Annual International FES Society Conference; July 2005; Montreal, Quebec.
- Tanner JA. Reversible blocking of nerve conduction by alternating current excitation. *Nature*. 1962;195:712-713.
- Lodish H, Berk A, Zipursky SL, Matsudaira P, Baltimore D, Darnell J. Cell-cell adhesion and communication. In: *Molecular Cell Biology*. 4th ed. New York, NY: W. H. Freeman & Co.; 2000:974-975.
- Alberts A, Johnson A, Lewis J, Raff M, Roberts K, Walter P. *Molecular Biology of the Cell*. 4th ed. New York, NY: Garland Science; 2002.
- Farrar J. Introduction to the supplement on ion channels. *J Pain*. 2006;7(1):S3-S12.
- Drevor M. Ion channels as therapeutic targets in neuropathic pain. *J Pain*. 2006;7(1):S3-S12.
- Patel N, Poo MM. Orientation of neurite growth by extracellular electric fields. *J Neurosci*. 1982;2(4):483-496.
- Brushart TM, Hoffman PN, Royall RM, Murinson BB, Witzel C, Gordon T. Electrical stimulation promotes motoneuron regeneration. *J Neurosci*. 2002;22(15):6631-6638.
- Gibson, JR, Beierlein M, Connors BW. Functional properties of electrical synapses between inhibitory interneurons of neocortical layer. *J Neurophysiol*. 2005;93(1):467-480.
- May HU. *High tone power therapy*. Presented at: Sixth Annual International Congress of Egyptian Society of Back Pain; April 19-20, 2006; Cairo, Egypt.