ELECTROANALGESIC NERVE BLOCK

Theory and case reports on an advanced generation electroanalgesic medical device (EAD) in reducing or mitigating acute and/or chronic intractable pain conditions.

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Electroanalgesic medical treatment involves the use of computer-modulated electronic signals to imitate, exhaust or block the function of somatic or sympathetic nerve fibers. An electroanalgesic medical device (EAD), utilizing communications-level technology, is used to produce and deliver higher-frequency signal energy in a continually varying sequential and random pattern via specialty electrodes. These electrodes of specific size, shape, and anatomical placement, can be effectively used to obtain pharmaceutical effects.[1,2] Electroanalgesic treatment for accomplishing nerve fiber block procedures typically use very small targeting electrodes (approx. ¾"-1.5" diameter), while electroanalgesic physical medicine treatments tend to use much larger electrodes (4" or more in diameter).

This electronically and digitally generated energy pattern also follows quarter-tone incremental steps with a pause at specific harmonic frequencies selected for their desired effects or mechanisms of action. This selection of specific frequencies effectively increases the initiation of tissue resonance phenomenon in the microstructure and macromolecular range. Some well known and well documented mechanisms of action employed by this harmonic resonance include the imitation of hormone/ligand effects, activation of cellular regeneration, and the facilitation of enzymatic metabolic processes.[3,4,5] The EAD unit used in the subsequent case reports was the Sanexas Neo GeneSys device.

Background

The use of electrical signals for various medical treatments has been mentioned since ancient times with the earliest man-made records (2750 BC) discussing the electrical properties and treatment potential of the Nile catfish, *Malopterurus electricus*.[6] Subsequent writings of Celsius, Oribasius, and other compilers describe medical treatment with electric fish by Hippocrates (420 BC) but little else until about 46 AD, at which time the Roman physician, Scribonus Largus, introduced the electrical capabilities of the fish into clinical medicine as a cure for intractable headache pain, neuralgia, joint inflammation, and gout.

In the 1700s, European physicians documented the use of controlled electrical currents from electrostatic generators for numerous medical problems involving pain and circulatory dysfunction. During that period, Benjamin Franklin also documented pain relief by using electrical currents for a number of ailments including frozen shoulder.

Today, the clinical use of electromedical modalities in both diagnosis and treatment is well documented with basic and physical science replete with references demonstrating the positive effects on patients for a myriad of medical conditions.[7] Transcutaneous Electrical Nerve Stimulation (TENS) treatment is a well-documented, mild form of electroanalgesia that has been shown to provide pain relief by administering small electrical currents through the skin. It is believed that the primary physiological mechanism of action achieved via standard TENS application is due to a direct *counter-irritation* of the central nervous system (CNS); the mechanism of action is consistent with the Gate Control Theory of Pain by Melzak and Wall.[8,9]

Electroanalgesia nerve blocks, both at the stellate ganglion and the lumbar sym-

pathetic region (paravertebral approach) have already been described in the literature. The reader is referred to the seminal paper by Robert Schwartz, MD titled "Electric sympathetic block: current theoretical concepts and clinical results."[1] These blocks have been shown to be up to 75% effective and may be able to decrease a patient's pain and increase functionality virtually without risk.

Advanced Generation Electroanalgesic Medical Devices

A more advanced, communications-level technology medical device, known as an electroanalgesic medical device (EAD), appears to be much more potent in its ability to reduce or mitigate acute and/or chronic intractable pain conditions than conventional TENS technology. The major difference in this new randomly generated higher frequency EAD technology over the older lower frequency TENS technology is that, in addition to the known and accepted TENS effects, the nerve axon transport of pain signals (action impulses) are interrupted (blocked). EAD technology incorporates randomly-generated electronic signal energy with much higher electrical frequencies (<25,000 Hz). This EAD technology is continually varying the 1) carrier frequency, 2) carrier frequency sweep speed, 3) pulse/modulation rate, as well as 4) continually changing the intensity (dosage) of the current to precisely match parameters delivered at the appropriate time.

Standard TENS technology relies on amplitude modulation (AM) of the electrical current being delivered to the body. The newer EAD technology uses a continually varied and randomly generated electrical signal current delivered to the body as amplitude modulated current (AM) and frequency modulated current (FM) combined. This complex electronic signal is manipulated by an on-board EAD computer, which actually combines or mixes both elements of AM and FM simultaneously. The theory is that this complex electrical signaling system is changing so often that the nervous system cannot "learn" or accommodate to the administered signal and that the speed of the electric signal is so high that a complete depolarization of the nerve membrane occurs.

Specific Parameter Electrical Signaling

Specific parameter signaling is defined as selecting certain parameters to achieve two specific ends: 1) to more directly (and indirectly) focus their electro-physiological effects toward specific characteristics of the various nerve fiber types (A-alpha, A-beta, A-delta, C-fibers, etc.); and/or 2) to address the medical indications where certain "therapeutic mechanisms of action" are known to be useful in the treatment success of that particular indication.

These electrical variables include manipulation of the 1) carrier frequencies, 2) movement (sweeping) of the carrier frequencies; and 3) sweeping of the carrier frequencies at different velocities between two border frequencies. With these frequency changes, the specific parameters of dosage (electrical signal energy amplitude) are varied according to the changing frequency parameters. This adjustment is required because, as frequency increases, higher intensity is required for deeper tissue penetration and effect.[1] The increased dosage is tolerated by the tissues without patient discomfort or heat generation because the current perception threshold also rises. Sophisticated computer signaling is required for the rapid adjustments of amplitude as a function of the changing specific parameters.

Electrophysiology of the Neuron

It is well known that electric current energy can be effectively used to relieve pain. Electric pulses of specific intensity and frequency can interfere with a neuron's own electrical impulses, or action potentials, thereby disrupting its ability to transmit painful stimuli.[10] The functional unit involved in the transmission of, and reaction to, painful stimuli is the neuron, or nerve cell. Neurons have a membrane potential difference in the electric charge between the inside and outside of the cell. This membrane potential is expressed as a negative potential because the inside of the cell is negatively charged compared to the outside. Application of specific parameter electrical stimulus makes the membrane potential more positive—a phenomenon called depolarization. Depolarization of the membrane to a certain threshold level induces a rapid firing of an action potential (action impulse). Once an action impulse has fired, a new one cannot occur until the membrane potential is stabilized back to its physiological resting potential. It should be noted that the action potential (impulse) is responsible for ALL transmission of bio-information, including pain signaling.

Once initiated by a distinct stimulus, the action impulse travels along the surface of the nerve axon and propelled by electrical energy generated locally by the depolarizing membrane. Thus, the impulse is both self-sustaining and self-propagating.

The impulse advances along the length of the nerve axon by electrical currents flowing between an active (depolarizing) membrane patch and adjacent resting (polarized) membrane surface. At rest, the interior of the nerve membrane is negatively charged with respect to the exterior. At the height of depolarization, the nerve membrane briefly reverses its polarity, with the interior now being positive relative to the exterior. This initiates a flow of electric current between depolarized and adjoining resting portions of the nerve, which reduces the membrane potential (i.e. depolarizes) ahead of the active region.

As a result of these electrical depolarizing local current flows, sodium channels activate and sodium ions begin to stream inward. Soon, the inward sodium current exceeds the combined outward flows through potassium and leakage channels, the firing threshold is crossed, depolarization ensues, and an action potential is generated in the adjacent segment.[11,12]

Hypothesized Mechanisms of Electromedical Pain Management

Electromedical management of pain occurs primarily by these hypothesized mechanisms of action:[13]

1. Counter-irritation: The gate-control theory (described by Melzak and Wall)[8] explains that repeated exogenously-applied electrical signals perceived by the sensory nerve fibers affect the brain translation of endogenously produced (pain) signals. This is a neuron function-imitation or function-exhaustion effect that causes suppression of the sensation of pain.

2. Release of neuropeptides: This release occurs electrically by a neuron function stimulation effect upon the sympathetic nervous system and dorsal horn. This stimulation activates the release of endogenous pain-suppressing neuromodulators found in the central nervous system, i.e., endorphins, enkephalin, GABA, etc.[9,14]

3. Nerve fiber block: Multiple signals of transcutaneously-applied specific parameter electrical frequencies fall within

the absolute refractory period of the cell membrane thus producing a sustained depolarization phenomenon. The traveling pain signal is stopped at the depolarized site (Wedensky Inhibition).[11,12,15]The nerve block is sustained by the post hyperactivity depression (PHD) effect (discussed in a subsequent section).

Cell Membrane Hyperpolarization vs. Depolarization

Unlike a pure chemical nerve block, which occurs because of a sustained *hyperpolarization* of the cell membrane,[16] the regularly structured sustained depolarization of the cell membrane—intermittently produced by the electroanalgesic device—also stops the transmitted propagation of the nerve axon pain signal while allowing all cellular voltage-gated channels to function at optimum levels until their designated equilibrium point.[12] This difference is of paramount importance as the necessary metabolic activity of the cell is continued while the patient's pain suppression objective is facilitated. This normalization of neuron cell activity, partly achieved through increased cAMP second messenger activity, directly reverses pain feedback circuits and promotes healing.[7]

Long-term relief is accomplished by stimulating the body's own chemical messengers within the cells to correct or normalize their function. It is known to the medical community that injury and/or disease may cause the cells to not work efficiently in the necessary elimination of metabolic waste products (metabolites), and can directly prevent the circulation from bringing in necessary cellular oxygen and nutrients. This has a direct affect on the immune system response and the ability to heal (gap-junction response). It is hypothesized that the cells are so overwhelmed by the metabolic chemical imbalance that they cannot self regulate.[12]

A recent report suggests that similar neuropathic pain symptoms may have separate and distinct "pain producing mechanisms [which] are pharmacologically separable."[17] Since all voltage gated channels are affected by the parameter specific current, it is postulated that multiple effects may be seen on these separate pharmacologically responsive receptors, in this case the Na+ channels and NMDA receptor systems.

Critical Role of cAMP

It has been demonstrated that electroanalgesic medical treatment (sustained cellular depolarization) has a direct effect on the increase or normalization of cyclic Adenosine Monophosphate (cAMP)[18] which directs all cell-specific activity. With a normalized level of cAMP, the cells will return to their normal activity thus providing a necessary intra-cellular/extra-cellular relationship.

In cellular physiology, the stimulated sustained depolarization that occurs has a direct effect upon the beta-adrenergic receptors, which are coupled to the stimulatory G protein. The initial response is an electrical conformational change of the cell membrane and activation of adenylyl cyclase, which converts ATP to cAMP. It is well described and documented that cAMP directs all cell-specific activity, including repair of insulted tissue that causes the metabolic cascade (leaking arachidonic acid) and increased level of noxious pain mediators. Electroanalgesic medical treatment, as a pain fiber block procedure, produces signal energy stimulation and subsequent sustained depolarization increases (to normal) intercellular levels of cAMP.[11,12]

Post Hyperactivity Depression (PHD) Effect

Specific-parameter electroanalgesic treatment also produces a prolonged, hypo-excitable state of a nerve that arises from the application of a relatively short duration electric signal combined with a chemical blocking agent. This is referred to as post-hyperactivity depression (PHD effect) and clinical studies have shown that a 20-30 minute procedure may produce pain relief that lasts for hours, days, even weeks.[1]

The C-fiber is more sensitive to the PHD effect than that of the A-fibers. Theories explaining this effect address the larger surface/volume ratio of small fibers compared to large fibers, making them more susceptible to trans-membrane potential effects resulting from extracellular ion concentration changes and known nerve fiber physiology concerning easier fatigue of small nerve fibers vs. large fibers.[1,15]

Effect of Dosage (Intensity) of Current

The ability of an electric stimulus to effectively penetrate body tissues and relieve pain is influenced by the current intensity (dosage), the carrier frequencies used, size and shape of specific electrodes employed, as well as anatomical placement. Increased current intensity (dosage) allows for increased depth of penetration and recruitment of deeper nerve fibers. Body tissue impedance, or resistance to alternating current, decreases with increasing current frequency; therefore, a higher current frequency requires less current intensity to overcome the outer skin and tissue impedance barriers. Since the perception threshold—defined as the lowest current intensity at which a patient reports any sensation at all (mild tingling, warmth)—increases with increasing frequency, a higher intensity current is permitted as frequency increases, thus facilitating the delivery of current to deeper tissues while avoiding pain sensation.[1,19,20]

"Greater, longer lasting patient outcomes appear to be achieved by performing the combination specific-parameter electroanalgesic procedure with a chemical blocking agent regimen."[20]

Combined Electroanalgesic/Chemical Block

Greater, longer lasting patient outcomes appear to be achieved by performing the combination specific-parameter electroanalgesic procedure with a chemical blocking agent regimen.[20] This procedure combines the positive benefits of intermittently generated membrane sustained depolarization, interruption of the pain signal along the axon, normalized cAMP levels, beta-adrenergic response, circulatory vasodilatation, general relaxation effects, and endogenous opiate release with the potent temporary blocking effects of the injected chemical blocking agent.

It appears that the chemical effects have either initially "overriding" hyper-polarization effects that cause complete cessation of the transmitted nerve pain signal, with some surrounding transient-firing nerve endings (even at lower dosage), or are simply better absorbed by the target nerve tissue under the electrical guidance phenomenon of the device-delivered electrical energy.[5]

It could also be hypothesized that the expected chemical block

effects can be potentiated by the forced interaction intracellularly, (even at substantially lower dosage), via the electrical manipulation of the voltage-gated channels by the specific parameter electroanalgesic signal energy delivered to the patient.

Case Reports

Several case reports are presented to illustrate a variety of techniques available with this technology. It is the hope of the authors that these cases will serve to not only demonstrate the power and safety of specific parameter electric treatments, but to stimulate further interest by the pain management community in specific parameter electroanalgesia.

Case Report #1: Successful Electroanalgesia Field Block for Carpal Tunnel Syndrome in a Complex Patient

The patient is a 69-year-old African American male who presented status post motor vehicle accident in May 2004. He was the seat-belted driver involved in a head on collision with an oncoming vehicle while making a left hand turn. The patient spent five days in the hospital under observation for a severe concussion. He does not remember the sequence of events leading up to the time of impact and most of his time spent in the hospital. He still does not fully recall his time in the hospital and events leading up to the admission. He had no apparent broken bones but had an abnormal brain scan and mild, vague mental status changes.

The patient presented with a wide variety of complaints, including head pain, neck pain, bilateral hand numbness, and right leg numbness. He had a past history of lower back pain related to disk pathology and associated left lower extremity radiculopathy. Prior to the accident he had pre-existing left hand pain.

An assessment was made of post concussive syndrome, probably post traumatic stress disorder, cervical pain with radiculitis, possible cervical facet syndrome, discogenic low back pain with a new right leg radiculitis, and probably bilateral carpal tunnel syndrome. The initial concern was for his slowly resolving post concussive syndrome. Initial brain CT scan without contrast, dated 05/26/04, revealed probable air within vessels throughout the calvarium and no evidence of acute mass or hemorrhage. Repeat brain scan a day later revealed stable appearing intraventricular air and no other CT evidence of any acute intracranial process. Moderate scanty pre-ventricular white matter was revealed in a subsequent brain MRI.

Initial treatment focused on the patient's confusion and short term memory loss. Appropriate consultations and studies deemed that the patient's post concussive syndrome, while not resolving, was stable. He initially declined interventional treatment, but eventually treatment was focused on his neck and bilateral wrists. The diagnosis of cervical facet syndrome was made, and the patient was treated with diagnostic bilateral C3, C4, C5 and C6 medial branch blocks and pulsed radiofrequency with eventual resolution of his neck pain.

Shortly thereafter, attention was turned to his wrists. He complained of pain and numbness in the medial nerve distributions, with right greater than the left. The pain awakened him at night despite the resolution of his neck and low back pain. A diagnosis of double crush syndrome with bilateral carpal tunnel syndrome had been made on a clinical basis. PE revealed a positive Phelan's and reverse Phelan's test bilaterally, and Tinel's sign was positive on the right. NCV testing was not done because of the patient's mental status.

Past medical history was significant for coronary artery disease; history of transient ischemic attacks (TIAs); hypertension; gastroesophageal reflux disease (GERD); gout; hypercholesterolemia; and lower back pain with previous lumbar spinal injury. The patient was disabled, admitted to occasional alcohol but denied tobacco. He was married, and, after initial sessions, his wife always accompanied his office visits and all treatments. Medications included Allopurinol 300mg qd; Lovastatin 40mg qd; Neurontin 400mg qid; Nifedipine 90mg qd; Pepcid 1-2 po qd; Percocet 10/325, 2 tabs po prn q day; Soma 350mg tid; Ticlopidine Hydrochloride 250mg bid; Vioxx 25mg qd. He was allergic to penicillin.

Because of the patient's age and medical status, noninvasive treatment with the EAD was initiated. The patient underwent treatment with the six-field endogenous electrodes, first to the right, and then the left wrist. An EAD program utilizing four separate and distinct treatment phases employing specific frequencies continually sweeping between 7333 Hz and 8333 Hz, with simultaneous amplitude modulation between 35 Hz and 200 Hz, was selected first. After three sessions on each side, nocturnal pain was eliminated. After approximately eight treatments on the right and four treatments to the left wrist, the patient was experiencing only occasional pain during the day. Subsequently, the patient was treated four times with two separate EAD treatment phases, utilizing specific frequencies between 3800 Hz and 20,000 Hz and an amplitude modulation sweep between 0.1 Hz and 200 Hz for rehabilition.

The patient underwent a final evaluation about a month after the last electroanalgesic treatment. Examination revealed significant improvements in his wrists and neck in comparison to his initial examinations. The patient estimated a 99% overall improvement, consistent with clinical observations.

The resolution of this patient's bilateral carpal tunnel syndrome (CTS) illustrates the utility of a pure non-invasive electronic signal treatment with the electro-analgesic device—without utilizing any local anesthetic blocks—to treat the often recalcitrant problem of this most common of peripheral neuropathies. The carpal tunnel contains other structures which may also be inflamed, and a strong inference can be made regarding the utility of the device to decrease overall tissue edema and promote healing.

Case Report #2: Combining Electroanalgesia Nerve Blocks with Electroanalgesia Field Blocks in a Patient with Refractory Low Back Pain

This 45-year old male presented with low back pain and bilateral radiculopathy as the result of a high-speed (60 mph) rear-end type motor vehicle accident in March 2003. Prior to his presenting, the patient underwent multiple modalities of treatment including medications, chiropractic care, and epidural steroid injections (4 times). He reported only temporary improvement from the injections. Lumbar MRI revealed posterior annular tear at the L3-4 disc and L4-5 left lateral disc herniation. The patient subsequently underwent lumbar discography, with disc disease at these two levels. He consulted with an orthopedic spine surgeon, who recommended multilevel fusion with hardware. The patient did not want to pursue the spine surgery option. He then underwent spinal decompression treatment with a

slight change in the radicular pattern but no overall significant decrease in his low back pain.

The patient complained of continued low back pain. He reported radiating pain into his right lower extremity—mapping out the L4/L5 nerve distribution. He experienced increased pain with straining, coughing, and sneezing. His pain was aggravated with activities of daily living (ADLs), bending, lifting, twisting, etc., and he required a cane to walk. As a result of this chronic low back pain, the patient was disabled and on Social Security.

Prior medical history was significant for a previous motor vehicle accident in November 1999. The patient fractured two ribs and injured his back. His symptoms resolved with conservative treatment, and he reported no residuals prior to the motor vehicle accident in question.

Review of systems and social history were noncontributory. Physical examination revealed that the patient ambulated with an antalgic gait (to the left). Palpation revealed moderate-to-severe tenderness over the lumbar paraspinous musculature and over the midline at the lumbosacral junction. Triad of Dejerine was reported as positive and Tripod sign was present. Perception of pin prick and light touch were decreased in the right L4 and L5 dermatomes.

Neurodiagnostic testing revealed a left sural sensory neuropathy. An assessment of internal disc disruption, L3-L4 and disc herniation at L4-L5, refractory to Epidural steroid injections (ESIs) and spinal decompression, was confirmed. Although fusion surgery had been recommended, the patient remained adamantly opposed to this option. A course of electroanalgesia field blocks along with specific electroanalgesia nerve blocks was recommended.

Over the course of a three week period, the patient underwent a series of electroanalgesic field blocks (12 times) with an EAD program utilizing frequencies between 7333 Hz and 8333 Hz and simultaneous amplitude modulation sweeps between 35 Hz and 200 Hz. After the tenth visit the patient noted that his leg pain was gone, with "only a few twinges" felt occasionally.

It was then elected to utilize a series of paravertebral electroanalgesia blocks at the L4 and L5 levels. Six electroanalgesia blocks were done over a period as right L5 (two blocks), bilateral L5 (one block), and right L4, L5 (three blocks). An EAD program using middle frequencies of 20,000 Hz, with an amplitude modulation of 50 Hz which selectively blocks the C-fibers, was utilized. The patient's pain scores consistently fell from 4-7/10 to 0/10 post block. The patient suffered a small burn to the paraspinous region as the result of a dry electrode during one of the blocks but the area healed without further problems.

During the course of this treatment regimen, he discontinued use of a cane to aid in his ambulation. The patient reported a substantial decrease in low back and radiating pain into his lower extremities. Overall, his VAS had been reduced to 2/10, and he was very pleased with this treatment.

The patient returned after over two full months of nearly complete pain relief. During that time he did not require any pain medications, and he no longer had any pain radiating down the leg. His pain had returned, to a stated 5/10 level, which was less than when he first presented. The patient's primary site of pain was now the lumbosacral region at about L5-S2 just to the left of midline. Electroanalgesia field blocks and paravertebral nerve blocks were reinstituted, and the patient once again reported improvement to a pain-free condition.

This case illustrates the usefulness of combining field blocks with larger electrodes and electroanalgesia nerve blocks, which, in this case, used ¾ inch to one inch electrodes over the paraspinous region with a large (5" x 8") grounding electrode on the abdomen anteriorly. This patient had already failed a course of nonsurgical spinal decompression therapy. Although the patient underwent an extended number of treatments (19 field blocks and eight electroanalgesia paravertebral blocks), this course was much less invasive, as well as less expensive and potentially more effective than the alternative of spine fusion surgery. Although long term improvement has not yet been fully realized, overall pain reduction has been achieved, there is no longer any radiculopathy, his function has been vastly improved, and his medication usage substantially decreased.

Case Report #3: Electroanalgesia Nerve Block Combined with a Local Anesthetic Block in a Patient with an Isolated Facial Neuropathy

The patient is a 51 year(s) old year-old male engineer with the chief complaint of left-sided supraorbital neuropathy with forehead pain of seven years duration. The diagnosis was neuropathic pain from the left supraorbital nerve. The onset was insidious; specifically the patient had no history of trauma and had not engaged in the sport of boxing. There was occasional crossover of the pain to the right side, but the pain was mostly left-sided in the forehead region above the supraorbital notch. The pain was described as constant and burning and radiated to the forehead and frontal skull. It was helped by medications, which included trazadone 50mg tid; Dilaudid 4mg, about 4/day for breakthrough; Valium 10mg tid; and methadone 20mg po q 12 hrs. With these medications, the VAS typically decreased from 8/10 to 3/10. This pain was aggravated by concentrating and other intellectual stress. The patient had been seen by a wide variety of providers, including two pain management physicians, and treated only conservatively. He had also consulted with a neurosurgeon who indicated that the patient was not a candidate for the Gamma knife. It was suggested that he consult with a pain management specialist who could do a phenol block in the region. The patient stated that, at the time he presented, he was "at the end of [his] rope" and would tolerate any motor consequences (although the supraorbital nerve is sensory only) to effect an elimination of the pain.

"The strategy of alternating the combined block with electroanalgesic blocks promoted healing and substantially decreased the patient's chronic neuropathic pain in about five sessions."

The patient reported no prior motor vehicle accidents, industrial related injuries, or other pain. He had undergone no surgery. Medical illnesses included depression (associated with the chronic pain) and hypertension. He had no allergies, was a nonsmoker, and used only occasional alcohol. He was married, worked as an engineer, and exercised more than three times per week.

On physical examination, the patient was alert and oriented and his mood and affect were appropriate. A head, eyes, ears,

nose, and throat (HEENT) exam revealed that he was normocephalic; Pupils Equal, Round, Reactive to Light and Accommodation (PERRLA); extra-ocular motions intact (EOMI); and tympanic membrane (TMs) were intact. Nasopharynx was clear. No jugular venous distention (JVD). Non-palpable thyroid was noted. Pressure on the left supraorbital notch reproduced the patient's pain. There did not seem to be any supratrochlear nerve involvement. The skin was otherwise clear. Examination of the cervical spine revealed no evidence of ecchymosis, erythema, or surgical scars. Palpation revealed no spasm over the paraspinous musculature and no associated tenderness in the same region. There were no trigger points noted. Cervical range of motion was full in all planes.

The patient had not undergone any diagnostic local anesthetic blocks to the region, nor were there any imaging studies available.

Assessment was a chronic idiopathic peripheral neuropathy of the supraorbital nerve, depression and chronic pain syndrome. A treatment strategy was planned utilizing a series of local anesthetic blocks to the left supraorbital nerve in conjunction with electroanalgesia blocks with the EAD. The patient underwent a series of four supraorbital nerve injections over 12 days. Three cc of 0.25% marcaine with a 25 gauge needle was utilized each time. This procedure was followed immediately by electroanalgesia blocks utilizing specific frequencies continually sweeping between 7333 Hz and 8333 Hz and simultaneous amplitude modulation between 35 Hz and 200 Hz in four separate and distinct treatment phases.

After the six electric nerve block treatments with the four supraorbital chemical nerve blocks, the patient's pain had decreased to 1/10. Four more treatments were done to further ensure a positive outcome, including one more combined local anesthetic/ electroanalgesic block, for a total of ten treatments. Final pain score was 1/10, and the patient's medication usage was decreasing. He was asked to return to the clinic in three weeks if the pain returned, but has not done so. The patient is now eight months s/p this treatment regimen.

The resolution of this patient's neuropathy illustrates the utility of utilizing a combined electroanalgesic block with local anesthetic blocks to treat the very difficult problem of peripheral neuropathies. Neuropathic pain is particularly amenable to treatment with electroanalgesia.[18] Myofascial pain may not be as permanently responsive to electroanalgesia treatment. The strategy of alternating the combined block with electroanalgesic blocks promoted healing and substantially decreased the patient's chronic neuropathic pain in about five sessions, after having been treated unsuccessfully for seven years and suffering major depression and anxiety as a result.

Case Report #4: Electroanalgesia Sympathetic Nerve Blocks Followed by Sympathetic Local Anesthetic Blocks in a Patient with Multi-extremity RSD

The patient was a 63 year old female who presented with a history of reflex sympathetic dystrophy (RSD) of the bilateral upper and lower extremities. She sustained a work related severely twisted left ankle in 1994. She was treated for a left ankle sprain but had substantial difficulties, and the symptoms got progressively worse. In 1995, she underwent a left tarsal tunnel release but her pain progressed. She was treated for RSD at Loma Linda and was diagnosed with chronic regional pain syndrome (CRPS); she underwent a series of sympathetic blocks, but RSD of the left lower extremity persisted. At that time she was judged to be substantially disabled, but her condition worsened. By the late 1990s, the RSD had spread to the left upper extremity and then to the right upper and lower extremities (left upper extremity much worse than the right upper extremity). Her left upper extremity pain included left sided anterior chest pain.

For the past seven years, she had been treated by a pain management specialist with a series of lumbar epidural local anesthetic blocks at L4-5 and L5-S1, alternating with primarily left-sided stellate ganglion blocks, one each every other month. She typically received 50% pain relief from these injections, and these injections increased her functionality and her ability to walk. The patient stated that the pain relief from these alternating blocks typically lasted between one to three weeks normally, but some blocks lasted for more than one month. Apparently, the conclusion was reached that there was not much else that could be done for her at this time.

Attempts were also made to manage her pharmacologically. She had an intrathecal morphine pump implanted in 1997, but she had it removed in 1998 and now depends mainly on the oral medication methadone at this time. She had also been treated by a psychiatrist for associated psychological problems (a major depressive disorder, single episode, chronic, severe without psychotic features).

She had moved to Arizona and it was a hardship because she had to travel 300 miles to see her California pain management doctor, since no doctors in Arizona would accept her California Worker's compensation insurance.

The patient had also been diagnosed with myofascial pain syndrome, left tarsal tunnel syndrome, and bilateral occipital temporal muscle contraction cephalgia secondary to a motor vehicle accident in June 2003. Her left sided shoulder and chest pain had been evaluated several times and cardiac involvement had been ruled out. All cardiac testing inclusive of a chemically induced stress test was normal. The patient has been on multiple medications including sublingual nitroglycerin. She states that the medications do not help to control her left sided chest pain.

A very comprehensive independent medical examination by a qualified medical examiner (QME), dated January 2005, stated that "as most authorities know, there is little that can be done to cure a severe case of RSD as she has." He went on to state that "[I] feel strongly that [the patient's pain management physician] should be allowed to continue these stellate ganglion blocks and lumbar epidural sympathetic blocks on basically an indefinite basis..."

Past medical history included hypertension. Surgeries included an appendectomy, cholecystectomy, IT MS pump implantation and subsequent removal, and nose surgery. Medications included Plavix 75mg q day; diazepam 5-10mg prn; Ultram 100mg bid; doxepin hydrochloride 100mg hs; Catapres 0.1mg qid; Atenolol 25mg q day; nitroglycerin 2.5 mg, 2 tabs q day; methadone hydrochloride 10mg q 2 hrs (8 per day); Celexa 20 mg, 2-3 tabs q day; Zonalon approx 4 tabs q day; omeprazole 1 tab hs; lovastatin 20mg q day. Allergies included penicillin and Vicodin. The patient was married and was accompanied by her husband at each visit. She denied alcohol and tobacco and was still able to exercise more than 3 times per week.

On physical examination, the patient was pleasant and coop-

erative, although she appeared somewhat anxious and was in mild distress secondary to her extremity pain and was very likely the result of seeing a new physician. Mood and affect were appropriate under the circumstances. She was alert and oriented and an excellent historian. No short term memory deficits were noted. The skin revealed no evidence of cyanosis and normal turgor, although the skin of the lower remedies appeared dry and there was decreased hair. Examination of the upper lower extremities was very limited by the patient's allodynia and hyperalgesia. She would not allow any palpation or other examination of her extremities nor permit any range of motion. The patient did move with a left sided antalgic gait, and had difficulty getting on and off the examination table. There was, in general, a quite limited range of motion of the neck, many of her joints, as well as the lumbar spine. The bulk of the musculoskeletal examination had to be deferred on the initial visit.

The assessment included: (1) reflex sympathetic dystrophy (RSD), or chronic regional pain syndrome (CRPS I); 2) chronic pain syndrome; 3) major depressive disorder, single episode, chronic, severe without psychotic features; and 4) possible C6-7 disc disease causing left sided C6 pseudoangina. A treatment strategy was planned utilizing a series of electroanalgesia blocks with the EAD to the bilateral paravertebral region at L3 and L5 along with a series of lumbar epidural injections; and a series of electroanalgesia blocks to the left stellate ganglion at C7 together with a series of chemical stellate ganglion injections. In all cases, the nerve block program utilized frequencies, previously described by Schwartz, addressing C-fiber characteristics, since C-fibers make up the bulk of the sympathetic chains in both lumbar and cervical regions.

Regarding the low back, the initial plan was to utilize two electroanalgesia blocks to L3 and L5 prior to doing the chemical block, which was done several days later. After the chemical block, done at midline L4-5 with 10 cc of 0.125% marcaine, she had a greater than 50% reduction in her pain sustained for several weeks. Her low back pain was eliminated, and the pain in her feet decreased by 60%. She found it was much easier to walk and stated that this was the "most effective lumbar epidural that I have ever had." Given the nature of a lumbar epidural injection and her past seven year history of serial LE injection, this increased benefit was attributed to the prior electroanalgesic blocks.

The patient underwent an electroanalgesia left sided stellate ganglion. She noted that the arm was warm, and that the pain decreased from 9-10/10 to 8/10. She also noted substantially decreased arm pain for several days. More significantly, however, is the fact that she has had no further left-sided anterior chest pain.

The patient received permission from her workers comp carrier for a subsequent series of five electroanalgesia sympathetic blocks as well as several more electroanalgesia stellate ganglion blocks over the course of one week. These were followed by a chemical SGB and lumbar epidural on the Friday of the same week. The patient reported several weeks later that "I never felt better in my life," and "It is fantastic!" Her pain levels in both the left arm and bilateral legs have dropped from 9-10/10 to 5/10. She had previously been bent over with the pain, and she now stated that she had straightened up and was walking much more erect and she no longer required her cane. All of her friends and acquaintances have noted the differences, as well. Her methadone usage has dropped from eight 10 mg tablets per day to about four per day, a 50% reduction.

These beneficial effects lasted well over one month and she has recently returned to the clinic for another series of electroanalgesia blocks to be followed at the end of the week with the left SGB and lumbar epidural chemical blocks. Although her treatment is ongoing, the overall time of benefit from a series of treatments continues to increase, as commonly is seen in the successful treatment of RSD. Given this patient's clinical course, the author (RHO) strongly feels that with several more series of treatments, the pain in both upper and lower extremities will be adequately controlled.

Conclusions

EADs, with computer-assisted electronic frequency generators, are used to produce stimulation activity that not only offer different therapies of physical medicine (therapy for myofascial pain), but also bring about a potent analgesic effect by calming down or blocking nerve pain (neuropathy/neuralgia).

Without piercing the skin, physicians can now administer effective nerve pain treatments to reduce the hyper-irritated state of the nerves. This is accomplished by placing specific surface electrodes on the skin and introducing very specific bioelectric signal impulses to produce an electroanalgesic nerve axon blocking procedure. The strength of this targeted procedural treatment field reduces the ability of the affected nerves to transmit pain signals and, at the same time, promotes healing by means of the depolarization effects on the nerve cells.

"The strength of this targeted procedural treatment field reduces the ability of the affected nerves to transmit pain signals and at the same time promotes healing by means of the depolarization effects on the nerve cells."

While the mechanism of action of the combined block (EAD and chemical) is unclear at this time, this technique has been successfully used and documented for a wide variety of refractory pain management problems, including fibromyalgia and diabetic neuropathy. Long term advantages of this treatment regimen include:

1) avoiding interventions or surgery in a patient for whom every conservative alternative had been exhausted or is a high risk medical patient;

2) avoiding the probability (even with surgery) of chronic pain for this patient for the balance of his life (depending on the outcome of ongoing treatment);

3) dramatic cost savings in both treatment and subsequent (lifelong) medication costs; and

4) potentially returning a disabled patient to the work force

5) allowing the patient to perform activities of daily living with minimal pain.

Patient results indicate that specific parameter electroanalgesic medical treatment is an effective modality for reducing or mitigating acute and chronic intractable pain for enhancing quality of life and well being. There appears to be enough evidence to encourage the use of EAD treatment in pain manage-

ment. It is the authors' belief that this technology has the potential to modify how interventional pain management is practiced and to improve outcomes with little added risk. The purpose of this preliminary communication is to stimulate interest by the pain management community in particular, and the medical community in general, in electroanalgesia nerve blocks and field blocks. ■

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References

1. Schwartz RG. Electric sympathetic block: current theoretical concepts and clinical results. *J. Back & Musculoskeletal Rehab*. 1998. 10: 31-46

2. Woessner J. Blocking out the pain: Electric nerve block treatments for sciatic neuritis. *Practical Pain Management*. 2002. 2(2): 19-26

3. May HU. Simultaneous Modulation of Frequency and Amplitude Modulated Electric Signals. European Journal of Physiology. 2002. 443 (Suppl. 1):53-59. [Physiological Society. 81st Annual Meeting]

4. May HU. Transcutaneous Stimulation of Nerves and Muscles with FM and AM Electric Signals. European Journal of Physiology 2004. 447 (Suppl. 1): 35-41. [Deutsch Physiologische Gesellschatt 83rd Annual Meeting, Leipzig, Germany]

5. Sorgnard R and Odell R. Integrative Block including Electronic Signal Intonation Sanexas/Outcome Medical, manuscript in preparation for submission. 2006.

6. Kellaway Electromedical History and Development, Malopterurus Electricus. 1946

7. Woessner J. Electric Nerve Blocks. Weiner's Pain Management: A Practical Guide for Clinicians. 2006. Boswell M, Cole B, Eds. CRC Press. pp 1233-1242.

8. Melzack, Wall. Pain Mechanisms: A New Theory. Science. 1965. 150:171-179.

9. Burton C. Pain Suppression by Transcutaneous Electronic Stimulation. IEEE Trans Biomed Eng. 1974. BME21 (2): 81-88.

10. McQuay HJ and Moore RA. Transcutaneous Electrical Nerve Stimulation (TENS) in Chronic Pain. An evidence-based resource for pain relief. Oxford University Press 1998. Chapter 25.

11. Alberts B, Bray D, et al (eds). Molecular Biology of the Cell. Garland Publishing. New York. 1994. pp 523-540.

12. Darnell J and Lodish D (eds). Molecular Cell Biology Scientific American Books, New York. 1990. pp 771-777.

13. Sorgnard, R, May HU, and Gramazio M. Differentiated Electromedicine; Physicians Continued-Education Review. St. Luke's Hospital and Medical Center. Manila, Philippines. 1997.

14. Bonica J (ed). The Management of Pain. Lea and Febiger. Philadelphia. 1990. 1:365-1938.

15. Katz B. Experimental Evidence for a Non-Conducted Response of Nerve to Subthreshold Stimulation. Proc R Soc. 1937. 612.816.3:244-276.

16. Cousins MJ and Bridenbaugh PO (eds). Neural blockade: In Clinical Anesthesia and Management of Pain. Third Edition. Lippincott-Raven. Philadelphia. 1998. p 1177.

17. Gottrup H, et al. Differential Effect of Ketamine and Lidocaine on Spontaneous and Mechanical Evoked Pain in Patients with Nerve Injury Pain. Anesthesiology. 2006. 104: 527-36

18. Woessner J. Electric Nerve Blocks. Weiner's Pain Management, Boswell M, Cole E (eds.) 2006. pp 1233-1242.

19. Savery, Silver, Sorgnard, May, et al. Assessment of Electric Differential Treatment for Ovarian Cysts and Concomitant Symptoms. Advances in Therapy. 1991. Vol. 8, No. 5

20. Savery F and Sorgnard R. Clinical Bioelectric Treatment in Endothelial-Related Disease. Clinical Electromedical Research Academy. 1996.

21. Woessner J. A conceptual model of pain: Measurement and diagnosis. Practical Pain Management. 2002. 2(6):27-35.

22. Outcome Medical LLC, Fayetteville, Georgia