

CLINICAL SUMMARIZATION REPORT:

ELECTROCEUTICAL TREATMENT AND REFLEX SYMPATHETIC DYSTROPHY (RSD)

175 Patient Summarization Report

R. Sorgnard, Ph.D., Pain Management
F. Savery, M.D., Family Practice
L. Nikolova, M.D., D.Sc., Neurology

R. Schwartz, M.D., Physical Medicine
L. Patterson, M.D., Neurology
M. Robertson, M.D., Anesthesiology

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Introduction:

Reflex Sympathetic Dystrophy (RSD) is a multi-symptom complication typically affecting one or more extremities, although it may appear in any part of the body. RSD is defined as a dysfunction of the sympathetic vasoconstrictive tone manifested by pain, edema, disorders of temperature regulation and trophic changes. The sympathetic nervous system innervates and controls the blood and lymph vessel tone and affects all tissue levels - skin subcutaneous fascia muscle, synovium and bone. Although individual patients may experience a variation in symptoms or changes, ***ALL complain of pain.***

Discussion: RSD Provocators

The etiology and underlying pathophysiology of RSD is poorly understood and often unrecognized clinically. Current theories divide RSD provokers into three (3) categories:

1. Peripheral factors: In 90% of reported cases, onset of the syndrome was due to lesions of the bone or soft tissue, i.e. fractures, distortions, frostbite, burns, cuts and severed vascular nerves. Inflammatory processes, such as joint inflammations, osteomyelitis, phlebothrombosis, and scleroderma can also induce RSD.
2. Endogenous factors: These include: patient's age, endocrine metabolic disorders (i.e. hyperthyroidism), tumors and neurological conditions (poliomyelitis).
3. Psychic factors: Increased anxiety, emotional lability, tendency to depression in psychosomatic reactions, i.e., asthma.

The more frequent initial factor is trauma and tissue damage resulting in chronic irritation of the peripheral sensory nerve. Injury to the sensory nerve produces an increase in the number of afferent impulses to the spinal cord and with any painful stimuli, a normal sympathetic nerve reflex arc is set in motion. This results in a temporary vasoconstrictive action of the small vessels. However, if this normal reflex arc fails to shut down at the appropriate time, an abnormal sympathetic reflex may develop

From the neurological point of view; painful stimulus enters the spinal cord via the afferent nerve fibers and stimulates the internuncial pool. Resultant afferent autonomic stimulation then goes to the peripheral tissues producing local circulatory disturbances and muscle spasms adding to the already noxious stimuli – propagating the vicious cycle.

Additional research has demonstrated that intensive stimulation of a sympathetic nerve fiber can facilitate the discharge of a pain evoking neuro-peptide. This substance, named neurokinin, is a mediator of neurogenic vasodilation of skin, and when injected subcutaneously produces a reaction which lowers the pain threshold.

Discussion: Clinical Picture

Symptoms of RSD comprise three stages:

STAGE 1: ACUTE OR EARLY STAGE

- A. Pain, usually limited to the site of trauma, sustained or at intervals, in periods of rest and increased in periods of strain.
- B. Localized edema
- C. Disorders of temperature regulation (hyperthermia or hypothermia).
- D. Muscle spasm
- E. Hyperaesthesia
- F. Stiffness and limited mobility
- G. Hyperhidrosis
- H. Trophic disorders, i.e. glossy skin, increased hair and nail growth.

STATE 2: DYSTROPHY STAGE

- A. Pain (sometimes less than stage 1, sometimes more severe) begins to radiate
- B. Edema spreads
- C. Muscle wasting
- D. Skin gets cold and pale, there is loss of hair, and nails become brittle and cracked
- E. Bone decalcifies (visible on X-ray)
- F. Increased joint thickness

STAGE 3: FINAL STAGE

- A. Individualized pain (some patients have little or no pain, others have intractable pain involving the entire limb)
- B. Skin is cyanotic and extremely thin
- C. Bone deossification is now marked and diffuse, coarse structure of spongy bone substance
- D. Stiff joints and false position of joints
- E. Muscle atrophy

Treatment: Materials and Methods

Electroceutical (bioelectric) treatment of RSD with an 80%+ patient success rate was described and documented by Nikolova in the 1960's. Our efforts indicate that patient response directly correlates to early diagnosis and treatment. While 80%+ success accompanies STAGE 1, and some STAGE 2 patients, STAGE 3 patient success is often as low as 50%-60%.

A total of 175 diagnosed RSD patients were treated with bioelectric energy (electroceutical medicine) via a new treatment modality produced in Europe and tested and approved by the Clinical Electromedical Research Academy (CERA). The medical instrument, offered by MATRIX Electromedical, Inc., was approved for sale by the FDA in mid-1993. Initial clinical patient treatment data, retrieved from a number of geographically different facilities participating in the on-going research program, indicate real promise in the favorable influence of Reflex Sympathetic Dystrophy and other vasospastic disorders.

It appears that the primary physiological mechanisms of action produced by clinical electroceutical treatments are:

1. Sympathetic fatigue causing vasodilation of the blood and lymph vessels.
2. Central pain relief - as a result of counter-irritation, which interrupts the vicious cycle: pain disorder of the regulation of the functional range of the sympathetic nervous system - disorder of micro-circulation - edema - disorder of nutrition tissue acidosis - pain.
3. Facilitation of diffusion processes - balancing the differences of electrical and chemical concentration, important for the mitigation of tissue acidosis and for the normalization of the chemical (pH) balance in the tissue.
4. Increased levels of second-messenger, (cyclic AMP) - important for the activation of cell specific activity in the treatment of RSD and for pathologically altered (sub-optimal) cells.
5. Stimulation of neuro-modulator release (endorphins, etc.) - long-term relief, i.e. raising the pain threshold.

A wide variety of treatment approaches, with different effectiveness and side effects, have been recommended for the treatment of RSD. This patient summarization study report demonstrates that electroceutical treatment is a non-traumatic, low cost, safe and effective alternative for RSD and is comparable in benefit to other documented conservative treatments.

These 175 RSD patients had averaged 2.8 years of pain when initiating the treatment regimen. Treatment areas included the affected anatomical areas plus the stellate ganglia, lumbar ganglia, and brachial plexus. Each patient received 20 minutes of electroceutical treatment during each session. Depending on the particular disease-Stage of the patient, specific bioelectric parameters were applied. If subjective pain reduction was not realized within 5 treatments, another parameter was implemented. Before and after starting the treatment program, each patient described the intensity and region of the pain.

Summarized Results and Conclusions:

175 patients entered the program. 142 (81%) of these patients experienced complete or partial relief of pain (Table I). 33 (19%) of the patients reported no benefit from the treatment. 73% of the patients have returned to gainful employment (Fig. I). In addition to pain relief, other improvements were noted. These included increased active movements of extremities, restored sensory function of affected extremities, improved sleep, relief from muscle spasms, overall improved health, mental status, and quality of life.

Our data indicates that specific-parameter bioelectric treatment (electroceutical medicine) is an effective modality for eliminating permanent partial disability and enhancing quality of life and well being. Compared to other means of treatment for RSD (Table II, III, IV, and V), it is a treatment alternative with few adverse reactions, good compliance, and low-cost characteristics - all of which makes this approach valuable.

Reference Bibliography

- Adrian, B. D.: "The mechanism of nervous action. Electrical studies of the neuron." Oxford Press, London, 1932
- Cauthen, I. C. and Renner, F. J.: Transcutaneous anti peripheral nerve stimulation for chronic pain states. *Surg Neurol* 1975; 4:102-105.
- Cousins, M. J.: "Neural Blockage," J. B. Lippincott Co., 1988
- Cronin, K. D., KJirsner, R. L. C.: Diagnosis of reflex sympathetic dysfunction. Use of skin potential response, *Anaes* 37:848-852, 1982.
- Davis, R. M. and Thomas, A. F.: Reflex sympathetic dystrophy syndrome, RSDS.
- Ficat, P., Hungerford, D.: Reflex sympathetic dystrophy, Disorders of Patello Femoral Joint, Reflex sympathetic dystrophy Chapter 9, Copyright 1977 Ed. Reprint 1979
- Fields, H. L. and Basbaum, A. I.: Anatomy and physiology of a descending pain control system, in "Advances in Pain Research and Therapy," vol.3. S. S. Bonica et al (Eds), Raven Press, New York 1979, pp.427-440.
- Foreman, R. D., et al: Effects of dorsal column stimulation on primate spinothalamic tract neurons. *S Neurophysiol* 1976; 39:534-546.
- Hansjürgens, A. and May, H.U.: "Traditional and Modern Aspects of Electrotherapy," 2nd ed, 1984.
- Hendler, N., Iematsu, S., Long, D.: Thermographic validation of physical complaints in "psychogenic pain" patients, *J of Acad of Psychosomatic Med* 23 No.3: March 1982.
- Hunter, Schneider, Mackin, Callahan: Reflex sympathetic dystrophy, Rehabilitation of the Hand 2nd Ed: Lankford, L., Reflex sympathetic dystrophy, Chapter 47, 1983, C. V. Mosby Co.
- Jenker, F. L.: "Transcutaneous Electric Nerve Block," Springer Verlag, 1986.
- Jenker, F. L.: Basic considerations. *Evidence for blocking. Schmerz/Pain/Douleur* 1988; 9:157.
- Kozin, F., McCarty, D. J., Sirtns, I., Cenant, H.: The reflex sympathetic dystrophy syndrome, I. Clinical and histologic. studies: Evidence for bilaterality, response to corticosteroids and articular involvement, *AMA Med* 60:321-331, March 1976.
- Kremer, B.: Measurement of pain: Patient preference does not confound pain measurement. *Pain* 1981; 10:241-248.
- Lampe, C. N.: Introduction to the use of transcutaneous electrical nerve stimulation devices, in "Transcutaneous Electric Nerve Stimulation." American Physical Therapy Assoc., Washington, 1979, pp. 14-18.
- Long D. M., Campbell, J. N., and Cuzer, C.: Transcutaneous electrical stimulation for relief of chronic pain, in "Advances in Pain Research and Therapy," vol.3. S. S. Bonica et al (Eds) Raven Press, New York, 1979, pp.569-585.
- Malament, I.B., Glick, J.B.: Sudek's Atrophy, the clinical syndrome, *A of AM pod Assoc.* 73 No.7:362-368. July 1983.
- Mayer, D. J.: Endogenous analgesia Systems: Neural and behavioral mechanism, in "Advances in Pain Research and Therapy," vol.3.5.5 Bonica et al (Eds), Raven Press, New York, 1979, pp.569-585.
- Melzak, R., and Wall, P.D.: Pain mechanisms: A new theory. *Science* 1965; 150:971-979
- Morel, F., in "The Role of Adenyl Cyclase and Cyclic Adenosine 3', 5' - Monophosphate in Biological Systems." T.W. Rodbell and P.C. Condliffe (Eds), in press. Government Printing Office, Washington D.C., 1970.
- Nikolova, L, and Davidov, M.: The effect of interferential currents on the activity of ferments in nerve lesions (translated from Russian). *Journal Voprosy Kurotol, Fisioterapi Lecebn Fiscic Kultura* 1978; 43:54-57.
- Poplawski, Z.A., Wiley, A.M., Murray, J.F.: Post-traumatic dystrophy of the extremities, *J of Bone and Joint Surg.* 642-655, 1983.
- Rusking, A.P.: "Current Therapy in Physiatry." W.B. Saunders Co., 1984
- Savery, F., M.D., EDiT and Endosan treatment for isehemic diseases, H.C.I. "Advances in Therapy," Vol. 8, No.2, 1991.
- Savery, F., M.D., Clinical applications of EDiT and Endosan treatment on diabetic neuropathy with gangrene. "Advances in Therapy." Vol, No.5, 1990.
- Schaltdt, M., Grass, H., and Brock, M.: "Aktuelle Problem der Neuropsychiatric," Springer Verlag, Berlin, 1978.
- Schoeler, H.: Physical block of the sympathetic chain. *Technikin Der Medizin* 1972; 1:16-18.
- Schwartz, Robert, M.D.: Electric sympathetic block, a review of electromedical physics, "Advances in Therapy," Vol.8, No. 1, 1991.

Spebar, M.J., Rosenthal, D., Collins, G.J., Harstfer, B.S., Walters, M. J.: Changing trends in causalgia, *Am J Surg.* 142:744-746, 1981,

Sorgnard, Schwartz et al Bioelectric Non-invasive Neuron Blockade: 223 Patient Comparative study.

Teeple, E., Ghia, J. N.: Considerations in treatment of causalgia, *Anes* 58:294, 1983.

Thomas, Audrey F., R. N., Davis, Francis J. & Rosalyn M., Reflex Sympathetic Dystrophy Syndrome Association (RSDSA).

Zimmerman, M.: Peripheral and central nervous mechanisms of nociception, pain and pain therapy, in "Advances in Pain Research and Therapy," Vol.3. J. J. Bonica et a (Eds), Raven Press, New York, 1979, pp.3-32.