Brief Report

Reflex Sympathetic Dystrophy Since Livingston

Bernard E. Edwards, M.D.

•The lack of fundamental knowledge and the need for continual research on Reflex Sympathetic Dystrophy is pointed out in this short review. Thermography has developed into the single most useful, valid diagnostic medium for identifying this pathology.

In contemporary literature on reflex sympathetic dystrophy there is seldom an article that does not refer to Weir Mitchell or Mitchell, Morehouse, and Keen,[1] the physicians in charge of Turner Lane Hospital in Philadelphia, the government hospital specializing in the investigation and therapy of peripheral nerve injuries during the Civil War. Their study of "causalgia" and "reflex paralysis"[2] has never been equaled in accuracy or description. John Mitchell [3] reported in 1895, by way of follow-up, that many of his father's patients continued to experience disability and suffering long after discharge from Turner Lane.

Some 75 years later, William Livingston,[4] an examiner of the Oregon State Industrial Accident Commission, and assistant professor of surgery at the University of Oregon, described his experiences and observations relating to phantom limb pain, reflex sympathetic dystrophy, causalgia, and other related pain problems. He lamented the fact that few surgeons had taken the time to read Mitchell's *Injuries of Nerves and Their Consequences*.[5] He affirmed that he intended to carry on the work of Mitchell et al., noting that a large part of his practice was devoted to the study and treatment of pain syndromes.

Livingston's research was guided by works of John Lewis, Rene' LeRiche, George Riddoch, Bailey and Moersch, and many others. He was frustrated by the many questions that arose regarding chronic pain (see below) and the paucity of answers. He pursued answers to these questions in his clinical practice.

His thinking was influenced by Head's *Central Integration of Sensory Impulses* [6] and LeRiche's *Surgery of Pain* [7] and by his own results with periarterial sympathectomy. Livingston's [4] theory of a disturbed internuncial pool [pp. 55-56] (with a "self-exciting system" [p.208]) in the substantia gelatinosa seem plausible; with the neurons arising in the dorsal horn, crossing immediately

to the opposite side, and ascending by way of the lateral spinothalamic tract to the thalamus, the idea appealed to his vision and reason. He supported the concept of an organic basis of chronic pain, in agreement with Reddich,[8] and a psychological cause, as was the theory of Bailey and Moersch.[9] He noted that pain did not always travel in classical nerve pathways; instead it could be referred along vascular pathways, frequently confounding neurologists.

Bonica, following the general work of DeTakats,[10] wrote extensively on his personal experiences with reflex dystrophy during and after World War II, culminating in his 1953 masterpiece, *Management of pain*,[11] a volume that is much in demand 35 years later.

Of particular importance in understanding and diagnosing RSD is the work of Sunderland,[12] who identified the median cord of the brachial plexus, along with the median nerve in the arm and the sciatic nerve in the leg, as being the sites of 40-70% of the sympathetic fibers in the extremities. These fibers closely follow the nerve pathways to the tips of the digits, and this fact could account for many questionable diagnoses of carpal tunnel and tarsal tunnel syndrome in the individual with RSD, resulting in a disappointing low level of therapeutic benefit following carpal tunnel surgery.

In Livingston's heyday low pack pain was, as it is today, a primary object of study and therapy. In *Pain Mechanisms* [4] Livingston relates a personal experience [p.130] that is still as relevant as it was 45 years ago. Drawing upon his vast clinical experience, Livingston described "post-traumatic pain syndrome," which develop following an apparently minor injury but which lack the burning pain of RSD (Livingston called this condition "the causalgic states" [p.114]). Livingston noted that

Sometimes a workman, who has long been accustomed to lifting heavy boxes, happens to lift a relatively light object and while making a turn, experiences a sudden "catch” in his lower back. When the acute pain has passed, a dull ache usually localizes over the original site of pain and may persist.

Instead of disappearing, as the individual expects, the ache persists and the pain becomes more intense and wide spread. It may encompass the whole lower extremity. The extremity to which the pain is referred may be cold and damp with perspiration, becoming the site of areas of numbness or paresthesia. The workman now complains that in addition to the ache extending into the leg, there is feeling of numbness, formication or a sensation "as if water were running down his leg." As the process continues, it is most difficult to predict how the period of disability will continue in such a case and many men have been laid off for months or years as the result of what seemed to be a very, trivial accident [pp.128-130].

We now know that many of these cases represent a complex syndrome - "sympathetic mediated pain" as Roberts has termed it,[13,14] or reflex sympathetic dystrophy.

In his book, *Pain*,[15] Lewis reported experiments relating to muscle ischemia, both intra- and extracellular. His results, combined with the identification of substance P, are provocative possibilities for understanding the etiology of the pain and impairment associated with RSD.

In 1937 DeTakats described the ischemia and dystrophic syndrome occurring after minor trauma and causing severe, uncontrolled pain, with sensory disturbances, a globelike hyperesthesia, and vasomotor and pseudo-motor changes. He noted that Weir Mitchell's classic description of causalgia closely resembled his own observation. While Mitchell's observed numerous cases of "mirror image," [p.305] he did not name the finding as such. He denied seeing any distinct redness or swelling of the opposite side, although he did observe burning and hyperesthesia.

Obviously, bilateral lesions arise from the same level of the cord structure. In his study of 35 cases of mirror image, Livingston [4] observed that the area of the contralateral extension was similar to the original lesion and that it often developed pain and hyperesthesia, rather than vasomotor disturbances, muscle changes, and edema [pp. 12 1 - 125]. Demonstrating his empathy for the pain patient, he wrote

Sometimes when one thing after another that I do to relieve pain has failed, there seems to be a malicious insistency about it. I feel almost that it acquires a personality like a spoiled, stubborn child which fiercely resents interference arid punishment, and deliberately goes ahead seeking means to break one's restraints. I get the feeling that if I had the patience and insight, I might be able to change it

it' sonic fundamental fashion so that it would become tractable. In the treatment of pain, there should be more physiological need for its control than a mere interruption of its communications [pp.26-27].

To put our current knowledge in perspective, a review of Livingston's original questions about RSD is in order.

What is the nature of the organic lesion? Is there really turbulence within the internuncial pool of the cord, producing and perpetrating sensory input? If this is true, how do we explain the total pain relief following a sympathectomy or a sympathetic block?

How can RSD bring about such widespread and disabling signs and symptoms? Much valuable information has contributed to the resolution of this question, such as the tissue change seen with translocation of fluid and the accumulation of gelatinous material traversing into the tissues, the consequences of ischemia in the extremities, and the accumulation of substance P.

By what means does injection therapy modify this organic lesion? Does the beneficial effect from sympathetic block prove that there are sympathetic efferents controlling the size of the blood vessels, or are "sympathetic afferents" blocked, relieving the pain?

How may such a modification act to abolish the extensive disturbances of function? This is a continuing puzzle and remains the basis for extensive ongoing research: Why are the changes reversible in the first 3-6 months but almost never totally abolished if therapy is started a year or more post-injury?

Why does the beneficial effect persist? There seems to be a physiologic holdover after nerve block, as is manifested by the continued increase in circulation and warmth of the extremity.

Like Livingston fifty years ago, we still consider these questions unanswered or only partially answered, While there remains a wide variety of therapeutic regimen's for the treatment of RSD, each being claimed as the treatment of choice by the advocate reporting the modality, there appears to be only one diagnostic procedure, thermography, which is consistently reliable and useful in the differential diagnosis of this syndrome.

The report of the AMA Council on Scientific Affairs, "Thermography in Neuromuscular and Musculoskeletal Conditions",[16] delineates thermography's value in differential diagnosis of reflex sympathetic dystrophy. In a syndrome that characteristically presents a wide range of temperature changes of the extremities (along with the numerous clinical manifestations presented by the patient), thermography stands alone in diagnostic usefulness, demanding, as in every imaging examination, the correlation of the clinical manifestations with the thermographic findings.

Although early cases may present with a temperature elevation, later on one usually finds a 2-5C cooler extremity; both will respond to sympathetic block, properly done, driving the sympathetic system to rectify its aberrant behavior.

Thermography can be used to monitor the progress and treatment coordinating this information with the symptoms and clinical findings. Jose Ochoa, speaking at the IASP meeting in Seattle in 1984, expressed his unequivocal opinion concerning the use of thermography:

We are convinced that thermography has a well defined, useful application in the workup for patients with sensory disorders of primary neurological origin. There are no truly objective clinical signs of the negative symptom (sensory loss) or the positive symptom (pain and paresthesia.) Although electrophysiological testing will detect nerve conduction deficit if the disorder involves hypoactivity in *large*-diameter afferent pathways, the function of the *small*-diameter fibers escapes routine electrophysiology. Moreover, no routine test reliably documents the overactivity that obviously underlies positive sensory phenomena such as paresthesias and pain.[17]

From the Pain Rehabilitation Unit, St, Joseph Medical Center, South Bend, IN

Address single copy reprint request to Bernard E. Edwards, M.D., 720 E.Cedar St., Suite 270, South Bend, IN 46617.

References

1. Mitchell SW, Morehouse GE, Keen WW, Gunshot Wounds and Other Injuries of Nerves. Philadelphia: JB Lippincott Co., 1864: 164.

2. Mitchell SW: Reflex Paralysis. Circular No.6, Surgeon General's Office, Washington, D.C. March 10, 1864.

3. Mitchell JK: Remote Consequences of Injuries and Nerve and Their Treatment. Philadelphia, Lea Brothers & Co, 1895: 245.

4. Livingston WK. Pain Mechanisms. New York: MacMillan, 1943.

5. Mitchell SW. Injuries of Nerves and Their Consequences. Philadelphia:]. B. Lippincott Co., 1872.

6. Head H, Rivers WH, Sherren J. The afferent nervous system from a new aspect. Brain 1905, 28:99-115.

7. LeRiche R. The Surgery of Pain, Baltimore: Williams & Wilkins, 1939; 512.

8. Riddoch G. Central pain. Lancet 1938; 1:1150-1156, 1205-1209.

9. Bailey AA, Moersch FP. Phantom limb pain. Canadian MAJ 1941:45:37-42.

10. DeTakats C. Reflex dystrophy of the extremities. Arch Surg 1937:34:939-956.

11. Bonica. JJ. The Management of Pain, Philadelphia: Lea & Febiger. 1953.

12. Sunderland S: Pain mechanisms in causalgia: J. Neurol Neurosurg Psychiatry 1976; 39:371-380.

13. Roberts WM: A hypothesis on the physiological basis for causalgia and related pains. Pain 1986; 24: 297-311.

14. Roberts WM, Foglesong, ME: A neuronal basis for sympathetic maintained pain. Thermology 1986; 2: 2-6.

15. Lewis 'I': Pain. New York: MacMillian, 1942; 192.

16. AMA Council on Scientific Affairs AMA council reports thermography in neurological and musculoskeletal conditions Thermology 1987; 2: 600- 607.

17. Ochoa J, Fields HL, Dubner R, Cervero F, eds. Advances in Pain Research and Therapy. New York: Raven Press, 1984; 9: 455.