Reflex Sympathetic Dystrophy Since Livingston

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•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 Wier 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, René 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

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