



Reflex Sympathetic Dystrophy (Current Management of Pain)

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Painful disorders following injury of peripheral nerves; bones and other soft tissues have occurred from the earliest times of human existence. Ambroise Pare was called upon to treat the persistent pain experienced by King Charles IX which was caused by a lance wound. The pain was persistent, diffuse and associated with contracture of muscles. The king could neither flex nor extend his arm for a month until the pain finally disappeared. Weir Mitchell, G.R. Moorehouse, and W.W. Keene produced a monumental treatise in 1864 titled "Gunshot Wounds and Other Injuries of Nerves," which contained an account of symptoms and signs of peripheral nerve injuries as observed in Unionist Soldiers. After 1864, however, little mention of this condition was made during peacetime until a spate of articles appeared again after World War One and Two. With the formation of societies such as International Association for the Study of Pain, renewed interest has been shown in understanding the mechanisms and management of pain syndromes. Pain caused by sympathetic disorders has always caught the fancy of clinicians, and yet confusion exists as to the etiology and proper treatment of reflex sympathetic dystrophy. Many new names have been proposed for these syndromes; recent ones include sympathetically or nonsympathetically maintained pain. Taxonomy of The International Association for the Study of Pain lists causalgia and reflex sympathetic syndromes as two distinct entities. All clinicians seem to feel that pain relieved by a diagnostic sympathetic block should be labeled as causalgia or reflex sympathetic dystrophy. Similarly, numerous therapeutic modalities have been proposed. They all center around sympathetic denervation of some sort, pharmacologically, chemically, or surgically. In spite of a great advance in our understanding of pain mechanism in the last quarter century, we are no closer to improving the outcome of patients with severe reflex sympathetic dystrophy. Etiology and incidence is still unclear. Diagnosis is made late and treatment is not standardized. Clinicians who treat causalgia and reflex sympathetic dystrophy have different treatments based upon their background and experience, rather than on the mechanism of the syndrome itself. The time is opportune now to gather some unbiased thoughts on RSD and clem- the air. Our editors, in particular Michael Stanton-Hicks, need to be congratulated for organizing a timely symposium on the subject and inviting international experts to discuss the pathophysiology and treatment of RSD. What follows in this monograph is a clear, concise presentation and discussion of nomenclature, etiology, incidence, mechanism, treatment, and outcome of RSD.

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