From the Book:

Chronic Pain: Reflex Sympathetic Dystrophy Prevention and Management

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Migraine and Ischemia

In regard to the mechanism of the development of migraine, there are two major schools of thought. First is the ischemic school, blaming migraine on vasospasm, and second is the CNS school, which tries to find the cause and mechanism of the development of migraine in the central nervous system.

By following reflex sympathetic dystrophy as a model for the mechanism of development of migraine, everything falls in place, and it becomes obvious that vasospasm in the central as well as peripheral nervous system is an integral part of migraine development.

Like any other type of hyperpathic pain originating from the sympathetic nervous system, the patient's personality, family tendency for low threshold for pain, and peripheral nerve injury in the form of injuries to cervicotrigeminal distribution all play major roles in the development of migraine. In this regard, migraine becomes a multifactorial syndrome that covers the entire spectrum of the clinical manifestations of RSD.

This study of migraine through the RSD theory in not an academic exercise. It helps us approach migraine management in a more comprehensive fashion. Just as RSD should not be a justification for amputation of the extremity, any for of surgical procedure obviously becomes useless for treatment of migraine.

The same medications that temporarily help migraine but aggravate it in the long term (narcotics, barbiturates, benzodiazepines) are also contraindicated in any form of chronic pain including the emotionally laden RSD.

The ischemic school of mechanism of migraine had been best presented by Olesen. In his studies he noticed the slowly spreading oligemia involving the cerebral cortex starting at the occipital pole and gradually moving forward. This slowly progressive oligemia, obviously a function influenced by CNS, is very similar to spreading depression of Leao.

Olesen and Welch emphasized the importance of stress as a precipitating factor in the development of migraine.

In Welch's theoretical model the major pathways are identical to the PSTT (Figure 14). "Activity in this pathway triggers activity in intrinsic noradrenergic system.

The activator for development of migraine according to the above authors may be visual through the spreading depression starting from the cerebral cortex or may be somesthetic, such as injuries of the head, face, or cervical spine.

The spreading depression provides the missing link in the chain of events of development of migraine headache after the origination of vasoconstrictive stimulation in the trigeminal nerve distribution. This vasoconstrictive phenomenon is typical of RSD in any other part of the body, which results in release of substance P and norepinephrine to initiate vasoconstriction as well as vascular permeability, prostaglandin synthesis in the periarterial space, and the development and spread of pain.