CAUSALGIA

From the Book:
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"A syndrome of sustained burning pain after a traumatic nerve injury combined with vasomotor and sudomotor dysfunction and later trophic changes"
Merskey

Causalgias are divided into two forms:

1. Causalgia major involves peripheral nerve injury with electrical "crosstalk" (ephapse) that causes severe hyperactivity of sympathetic system (hyperpathia, vasoconstriction, and movement disorder). The major form is severe, usually caused by injury with high velocity sharp objects (e.g., butcher's knife), vibratory component major trauma (e.g., bullet), or high-voltage nerve lesions (electrocution).

2. Causalgia minor involves the same principle as causalgia major, but milder injury, e.g., injury to the dorsum of hand or foot, nerve root contusion, patient falling from a height on gluteal region resulting in "guillotine" effect, bruising of nerve root caught at the narrowed intervertebral foramen.

The difference between the two categories is a matter of degree and severity. To classify causalgia as an independent illness is artificial, and causalgia is nothing but a sever form of RSD.340, 363-365
In this severe form of RSD, the course of the disease is quite accelerated from stage 1 through 4 in a matter of weeks or months. S. Weir Mitchell in 1872 first reported rapid development of atrophic changes in the skin, nails, and soft tissues of the extremity in a matter of days to weeks.

Whereas in RSD of disuse the extremity is cold, in ephaptic dystrophy the thermography reveals in the distal portion of the extremely cold extremity that there is an isolated hot spot that points to the area of scar formation and ephaptic peripheral nerve dysfunction (Figures 4-6). In this area the vasoconstrictive capability of the sympathetic nerve is paralyzed, and there is a topical hot spot. This hot spot can be appreciated only by thermography.

This type of RSD is quite painful and very difficult to treat. It demands multidisciplinary therapy as well as early diagnosis. The ephaptic form is characterized by increased heat emission at the area of ephaptic lesion (electric short). As the condition becomes chronic, the distal portion of the extremity involved and the contralateral extremity becomes cold, but the ephaptic spot stays hyperalgesic and warm (Figures 4, 5, and 6b).

**Causalgic Pain**

Sunderland in 1978 succinctly defined causalgic pain as follows:

1. Usually pain occurs after the injury to a nerve trunk.

2. The severity of the injury to the soft tissues other than the nerve does not play a role in the severity of the pain.

3. The pain is spontaneous, severe, and quite persistent.

4. There is a markedly lowered threshold for aggravation of pain. This is the case in all RSD patients, but it is more exaggerated in causalgics. So even a breeze over the skin or the touch of a bed sheet or a change of the environment or a family argument and aggravation can markedly aggravate the pain. This feature of emotional aggravation is common to all RSD patients, and it is nothing but the role of the frontal lobe and the limbic system in aggravation of hyperpathic pain.

5. The pain is felt distal to the proximal nerve injury, i.e., in the hand or foot. This is typical but not invariable. The pain does not necessarily have to be a burning type of pain, and can be described in many other hyperpathic forms.

6. Sunderland established the requirement that the pain should be present at least 5 weeks. However, depending on the severity of the injury, the pain can develop in a matter of days or weeks. What happens to an extremity in RSD of disuse in a matter of several months can happen in a matter of a few weeks to a causalgic patient.
In no case of RSD is the pain so severe, so intolerable from burning, seering, aching, tingling, lightening, stabbing, crushing, to a combination of the above, without burning pain in a matter of a few hours to up to 7 to 10 days in close to 90% of the cases. However, it is not unusual to see some patients who develop the pain as late as 3 to 4 weeks after the injury.

Major causalgia is due to scar formation of peripheral nerves but has a component of high-velocity or high-vibration injury in its etiology. This is usually seen after bullet injuries or high-velocity sharp objects such as a butcher knife or surgical instrument injury. This is typically seen in war injuries, but it can also be seen in civilian trauma due to amputation of an extremity or industrial injury to the extremity. It is not uncommon in electrical injuries. Drilling steel against titanium in the aerospace industry causes high-frequency vibration and makes the patient more susceptible to causalgia.

The difference between the minor and major causalgias is a matter of degree and severity. For more detail on causalgia, see Chapters 1 and 12.

**Major Causalgia and Motor Dysfunction**

Major causalgia is the best example of efferent dysfunction secondary to sensory nerve damage and RSD. This efferent dysfunction is quite frequently present among causalgic patients (at least over 50% of the patients) and is in the form of flexion deformity of the extremity, tremor, weakness of the extremity, and dystonic movements.

The management of major causalgia requires a multidisciplinary approach. Trigger point injections should be applied to referred pain areas rather than the area of peripheral nerve damage. Repetitive sympathetic nerve blocks can be quite effective. Sympathectomy should be used only for patients who have failed with every other form of therapy and when the patient has a short life expectancy.

Even among patients who have has such a conservative approach towards sympathectomy, there still may be the necessity for morphine pump after failure of sympathectomy (Table 42). Morphine pump, a last resort in treatment, provides good control of pain. As most causalgic patients in civilian (as opposed to war) practice are involved in protracted litigation (especially worker's compensation cases), by the time they are being evaluated for RSD several months or years have elapsed, and the only effective treatment is morphine pump in this stage IV of the disease. For more detail on causalgia, see Chapters 1 and 12.