REFLEX SYMPATHETIC DYSTROPHY-NEUROSURGICAL APPROACHES

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This review will examine the surgical approaches to the treatment of neural injury pain especially as it relates to the sympathetic system. Difficulties arise when one attempts to fit RSD into this setting. RSD might be used to describe any complex of deafferentation or central pain, including posttraumatic syndrome where the neural injury element may be subclinical, which presents with sympathetic hyperfunction (1). RSD occurs in 5% of all cases of trauma (2). The term is often used interchangeably with Sudeck’s atrophy (3), causalgia, minor causalgia, shoulder-hand syndrome, posttraumatic spreading neuralgia, posttraumatic pain, sympathalgia, chronic traumatic edema, algoneuroadysrophy (1,4). It is precipitated by fracture in 50% of cases but blunt trauma, inflammation, laceration, surgery, soft tissue injury, injection, angina pectoris, vascular disease, myocardial infarct, osteoarthritis, frostbite and burns have all been identified causes. RSD may be diffuse or limited to a single finger; though commoner in adults, it also occurs in the young; it has been described in association with injuries to peripheral nerves, carpal and tarsal tunnel syndrome, lumbar and cervical disc disease, cord lesions, post-herpetic neuralgia and stroke (1,5-18). In a review of 333 consecutive personal cases of central and deafferentation pain (19), the author found evidence of sympathetic hyperfunction in 9 patients. It occurred in 1% of those with pain caused by nerve injury, 1% of the amputation-related group, and 1% of those with neural injury pain associated with disc disease. Two percent of patients with brachial plexus lesions and 4% of those with
posttraumatic syndrome were affected. It was not seen in any of our patients with post-herpetic neuralgia, post-thoracotomy syndrome, peripheral vascular disease or central pain.

Invasive treatment of RSD usually consists of repeated sympathetic blocks and prognosis is generally considered better if the blocks can be done early in the course of the disease. Despite this, permanent sympathectomy is rarely successful in relieving pain (20-31).

Let us now look more closely at the curious group of pain syndromes included under the term neural injury pain: the deafferentation and central pains with which RSD may be associated. In some of these conditions, as Livingston (32) suggested, the neural injury element may be subclinical. This is the case in posttraumatic syndrome and incisional pain where, though small peripheral nerves are doubtless involved, neurological examination reveals no deficit. In other patients the neural element is much more obvious. Neural injury pain is idiosyncratic: that is, not all patients with a given neural lesion develop pain, and it is uncommon in the young. The onset of pain may be delayed after the causative event in 36 to 67% of patients with deafferentation pain, 77 to 86% with central pain. From 98 to 100% of patients suffer from a steady spontaneous pain that is usually burning and tingling in quality. Fourteen to 43% suffer from intermittent spontaneous pain whilst 19 to 88% suffer from evoked pain (hyperpathia, allodynia or hyperesthesia). These ranges reflect the different neural injury pain syndromes reviewed. The steady burning tingling element of RSD pain tends to be temporarily relieved by intravenous doses of sodium thiopental, insufficient to produce sleep. Though rarely relieved by opiates, the effect is not reversed by 0.4 to 0.8 mg of naloxone given intravenously. Proximal or distal local anesthetic somatosensory blockade usually relieves the pain temporarily, but it is important to recognise that permanent surgical denervation at the same site as the block, usually fails. Such burning tingling steady pain behaves as if it were caused by central neural aberrations such as denervation neuronal hypersensitivity caused in turn by the