

## Role of the Sympathetic Nervous System in Painful Nerve Injury

Oisin R. O'Neill, MD,\* and Kim J. Burchiel, MD†

The existence of chronic pain with associated sympathetic overactivity and possible dystrophic changes consequent to regional injury has been alluded to in the article on Post-traumatic Neuropathic Pain. These states are encountered with relative frequency in clinical practice, although they are often unrecognized. Over the century since they were first described, these syndromes have been subject to classification under a myriad of synonymous titles, which has caused much confusion (Table 1). Although the pathogenesis of these painful disorders remains incompletely understood and no single theory can explain all of the clinical features, much has been learned about the neurophysiology and the mechanisms of pain sensation (nociception) over the last several decades which suggests that the sympathetic nervous system is perhaps causally related to, or at least supportive of, some of the classic features of these disorders. Its role in the dystrophic features of the more common clinical "variants" of causalgia, reflex sympathetic dystrophy, Sudeck's atrophy, and shoulder-hand syndrome is less clear. Roberts<sup>14</sup> has suggested the term *sympathetically maintained pain* (SMP) to encompass these disorders "to avoid reference to only one of this family of

conditions." If extrapolated to the clinical arena, this concept may diminish semantic confusion and facilitate the identification and early treatment of these disorders. Our intention in this article is to alleviate confusion by disposing of obsolete nomenclature and highlighting clinical similarities of pain states related to each other on a spectrum of clinical features under the clinical, not theoretical, umbrella of SMP. We briefly define the clinical conditions and discuss their etiology, pathogenesis, diagnosis, treatment, and prognosis.

### SYMPATHETICALLY MAINTAINED PAIN

Clinically, the SMP syndromes have in common: (1) the appearance of pain completely disproportionate to that expected from the initiating injury, (2) sympathetic dysfunction variably expressed, (3) a propensity for dystrophic change, and (4) delay in functional recovery.<sup>1, 6, 14, 16</sup> Diagnostic criteria for these states have been and remain extremely variable and, indeed, Roberts denies the necessity for dystrophic change or nerve damage and requires response to sympathetic blockade in his cri-

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From the Division of Neurosurgery, Oregon Health Sciences University, Portland, Oregon

\* Resident in Neurosurgery

† Professor and Head

Table 1. Titles Used for SMP Syndromes (Causalgia/Reflex Sympathetic Dystrophy) Based on Predominant Clinical Features or Proposed Etiology

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Acute bone atrophy
Algoneurodystrophy
Causalgia
Chronic traumatic edema
Leriche's post-traumatic pain syndrome
Major causalgia
Minor causalgia
Mimo causalgia
Minor traumatic dystrophy
Major traumatic dystrophy
Post-traumatic pain syndrome
Post-traumatic painful osteoporosis
Post-traumatic vasomotor disorder
Post-traumatic spreading neuralgia
Reflex neurovascular dystrophy
Reflex sympathetic dystrophy
Shoulder-hand syndrome
Sudeck's atrophy
Sympathalgia
Sympathetic overdrive syndrome
Sympathetically maintained pain syndrome
Traumatic vasospasm

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teria for SMP.<sup>1, 6, 14</sup> The characteristic common denominator, pain, may initially be confined to a nerve distribution but, in contrast to neuralgias, might ultimately spread beyond these confines.<sup>1, 16</sup> Classically, a burning persisting pain or dysesthesia exists with the possibilities of associated hyperpathia, hyperalgesia, or allodynia (see Table 2 in the article on Surgical Management of Post-Traumatic Neuropathic Pain.<sup>1, 6, 11, 13, 17</sup> This results in an individual with extreme spontaneous or stimulus-induced pain or both, with the social, psychologic, and physical distress which accompany that pain.

The SMP syndromes were initially described as post-traumatic, particularly, causalgia and reflex sympathetic dystrophy (RSD), although many etiologies exist, including visceral injury and regional anoxic, hypothermic, and electrical injury.<sup>6, 7</sup> The SMP disorders are dynamic syndrome complexes and may present with varying symptoms and at varying stages of progression and, thus, might not exhibit all of the classic features described. What distinguishes the SMP from the RSD syndromes is that, by definition, SMP is an empirical rather than an etiologic or mechanistic

categorization. Thus, if a patient's pain is relieved by pure sympathetic blockade, the pain is considered "sympathetically maintained." In practice, much of what would be called RSD would be called SMP and vice versa. While seeming to add another layer of complexity to an already almost incomprehensible array of disorders, the SMP scheme allows another means to focus on the pathophysiology of the pain and direct treatment.

**Causalgia.** Causalgia is "a syndrome of sustained, burning pain after a traumatic *nerve injury* combined with vasomotor and sudomotor dysfunction and later trophic changes."<sup>11</sup> As initially described by Weir Mitchell<sup>11a</sup> after the American Civil War, causalgia typically followed missile injury to a mixed nerve. Ninety-five percent of cases are associated with incomplete nerve injury, and 80 to 90% of these are proximal to the knee or elbow joint.<sup>1, 13</sup> Severe, spontaneous, and persistent pain is the most pronounced feature. Generally, the pain is in the distal extremity and is burning in character. The pain usually appears within several days of injury, although as many as 43% of patients may experience it immediately or within 24 hours of injury.<sup>1, 13</sup> Paresis and dysesthesias may be evident early on, probably related to direct nerve injury, but these tend to resolve as the causalgic pain worsens. This pain (as is common with all of the SMP variants) is not restricted to nerve or dermatomal distribution and may spread to areas remote from the area of nerve supply. Fifty to seventy-five percent of these patients also experience paroxysms of lancinating pain superimposed on the chronic burning pain as well as allodynia.<sup>1, 13, 16</sup> Significant exacerbation occurs with movement; mechanical or thermal stimuli; venous congestion; emotional states including anger, fear, and mental distress; and, in severe cases, with visual or aural stimuli.<sup>1, 6, 13, 18</sup> The sciatic nerve and its distal branches, the median nerve, and the medial cord of the brachial plexus are affected most frequently, with a reported incidence of 40%, 35%, and 12%, respectively.<sup>1, 13, 18</sup> The majority of postganglionic sympathetic fibers to the extremities are carried in these nerves, and this may be the anatomical basis for their selective vulnerability. The prognosis is unrelated to the degree of initial nerve injury, age of the patient, or associated soft tissue

damage and, although occasionally self-limiting, has been reported to persist for more than 6 months in 85% of cases.<sup>13, 17, 18</sup>

**Reflex Sympathetic Dystrophy.** Debate continues as to whether causalgia is part of the broader RSD category.<sup>1, 6, 9, 11, 13, 14, 16-19</sup> In contrast to causalgia, RSD can be considered to include those patients whose pain syndrome is initiated by soft tissue or bone injury without direct nerve injury. This includes proximal (shoulder, spine) or visceral (heart, brain) injury as predisposing factors as well as many iatrogenic factors including surgical procedures and external fixation. By considering both RSD and causalgia as potential subsets of the SMPs, we can maintain the semantic separation of these entities based on the presence or absence of nerve injury while admitting that there may be common mechanisms in maintaining the pain, sympathetic dysfunction, and, possibly, the dystrophic changes noted in both.

Greater than 50% of *recognized* cases of RSD have been reported following extremity fracture. The cardinal symptoms, as with causalgia, are pain, swelling, discoloration, stiffness, and loss of function.<sup>6, 16</sup> Secondary symptoms include osseous demineralization, vasomotor instability, and sudomotor, trophic, and temperature changes.<sup>6</sup> The course is variable and also potentially self-limiting, although this need not be so. Cases of spontaneous recurrence have been reported after supposed resolution.<sup>1, 17</sup> Reflex sympathetic dystrophy has been divided into three overlapping confluent *stages* (I to III), initially defined as lasting 3 to 6 months but actually varying from weeks to years.<sup>5, 7</sup> The symptom complexes have also been *graded* according to severity: grade 1 being the most severe and similar to classic causalgia and grade 3 being the mildest and perhaps the most common form.<sup>1</sup>

The early or *acute* stage (I) of RSD is heralded by pain onset several days after the injury, although this may be slower and more insidious after visceral injury, and typically lasts several weeks. A predisposing injury is identifiable in approximately 60% of cases, with the remaining patients unaware of any antecedent event.<sup>5, 13</sup> Again, early on the typically burning pain may be confined to a region of nerve supply but later will spread beyond this, as will the related allodynia, hyperesthesia, edema, and ten-

derness. Swelling may be the most common early physical sign manifest as pitting edema.<sup>6</sup> Sympathetic dysfunction evident through sudomotor and vasomotor changes is evident here. Initially, sympathetic hypoactivity is manifest as increased blood flow and regional hyperthermia from 2 to 6°F, with warm, dry, and red skin and regional increased hair and nail growth. Later in this stage, paradoxical sympathetic hyperactivity causes progression to a cold, cyanotic, and sweating state, and bone demineralization begins to be evident.

The *dystrophic* stage (II) begins 3 to 6 months after injury in untreated patients and is characterized by some diminishment in the burning pain at rest but severe exacerbation with motion and a progression of allodynia. The swelling becomes a brawny edema which spreads and contributes, with periarticular thickening and muscle atrophy, to increased limitation in joint movement. The distal extremity appears cool, pale, and shiny, with loss of skin creases, diminished hair growth, and cracked, grooved nails. Roentgenography reveals more patchy osteoporosis, and the patient begins to exhibit signs of the "causalgic personality."

The late or *atrophic* stage (III) is characterized by progression of the dystrophic changes and may proceed to irreversible damage. Normalization of skin temperature and blood flow is evident, and the pain, which may be less problematic during quiescence, may continue to spread. Overt muscle wasting and weakness are evident, and tendon contracture is pronounced. Spread to the entire extremity with loss of function may occur. Bone demineralization and ankylosis are dramatic by this stage.<sup>5, 17</sup> The causalgic personality, secondary to chronic severe pain, is evident, with the patient constantly protecting the affected extremity, seeking seclusion, becoming withdrawn, and exhibiting emotional and behavioral disturbances.<sup>1, 6, 13</sup> The physical devastation associated with SMP is thus accompanied by potentially equally severe emotional and psychologic changes that may tend to amplify and worsen the somatic changes.

Many investigators have attempted to identify the population at risk for causalgia and RSD and the frequency of the classic

features of these disorders in SMP.<sup>1, 6, 15, 17, 18</sup> This has proved difficult as a result of the lack of uniform acceptance of the definitions of RSD and causalgia and the resultant variation in diagnostic criteria. Reported incidences of causalgia in World War II have been from 2 to 5% (range, 1 to 15%) and have questionably decreased since then, possibly as a result of improved wound care techniques.<sup>1, 13, 16</sup> The incidence of reflex sympathetic dystrophy is more difficult to quantify, but it is probably the largest subset of SMP.<sup>1</sup> Its incidence following myocardial infarction is less than 1%.<sup>17</sup>

### Pathophysiology of SMP

No single pathophysiologic mechanism defined over the past century has explained completely all the clinical features of what might now be called SMP. Many theories, most extrapolated from experimental data, have been forwarded and implicate various areas as being responsible. These include peripheral tissue or nerve pathology, spinal cord regional dysfunction, or higher neuraxial dysfunction. Most would acknowledge that the pathophysiology of SMP probably involves dysfunction in many, if not all of these areas.<sup>1</sup>

Proposed peripheral mechanisms for pain include (1) abnormal discharges in sympathetic afferents, (2) sympathetic stimulation of peripheral sensory receptors, and (3) short-circuiting between sympathetic efferents and nociceptive afferents. The central mechanism suggests sensitization of spinal cord nociceptive neurons.<sup>1, 6, 13, 16</sup> Mechanisms by which the sympathetic nervous system might mediate pain include (1) activation of non-nociceptive mechanoreceptors by sympathetic efferents, (2) activation of afferent nociceptors or damaged nociceptor axons by sympathetic efferents, and (3) activation of nociceptor afferents by chemically mediated (sympathetic) release of co-transmitters or neuroactive substances (e.g., prostaglandins).<sup>4, 15</sup>

Lankford<sup>6</sup> suggests that three conditions must be present in the patient in whom RSD/causalgia (SMP) develops: (1) a persistent painful lesion (traumatic or acquired), (2) a diathesis (predisposition), and (3) an

abnormal sympathetic reflex. He suggested, based on a theory proposed by Livingston in 1943, that a persistent painful lesion in an individual with a hyperreactive sympathetic system leads to a maintenance of sympathetic vasoconstriction beyond the normal transient state, thus causing painful local ischemia and setting up a vicious "pain reflex."<sup>6</sup> This might be mediated by increased firing of self-exciting neuron loops in the spinal cord (the "internuncial pool") as a result of the persistent afferent stimulus (pain). One year later, Doupe<sup>1a</sup> suggested that peripheral tissue damage interrupts myelin sheath continuity and results in short-circuiting between the efferent sympathetics and the afferent nociceptors. These artificial synapses, or "ephapses," have since been demonstrated in animal models.<sup>1, 6, 13, 15</sup> A neuroma model has also been suggested as causative. However, ephapses and neuromas are delayed occurrences following trauma and, coupled with the fact that purely sensory nerve or tissue (non-nerve) injury may cause these syndromes, these cannot be sole mechanisms.<sup>13</sup> Another mechanism of increased afferent stimulation is proposed to result from "epileptic" discharges in damaged afferents that may exhibit spontaneous activity and chemical or mechanical hypersensitivity (see article on Pathophysiology of Injured Axons).<sup>1, 4, 13</sup> Melzack, who was responsible for defining the delicate balance in sensory afferent recognition and transmission in his "gate control" theory of pain, suggests that a selective loss of large myelinated fibers allows facilitation of transmission of nociceptive impulse (e.g., the "pain gate" was opened).<sup>1, 6, 10</sup> Melzack also proposed the existence of a "central biasing mechanism" in the reticular-activating system which tonically inhibits rostral transmission of pain at all synaptic levels of the somatic projection system, and that skin sensory input may help maintain this bias. Thus, any decrease in normal sensory input or imbalance of the central biasing mechanism, as might occur with emotional stress, might cause persistent discharge activity with the evolution of "memory traces" in the somesthetic nervous system and a susceptibility to recurrent pain.<sup>1, 17</sup> Roberts<sup>14, 15</sup> has suggested that excitation of peripheral no-

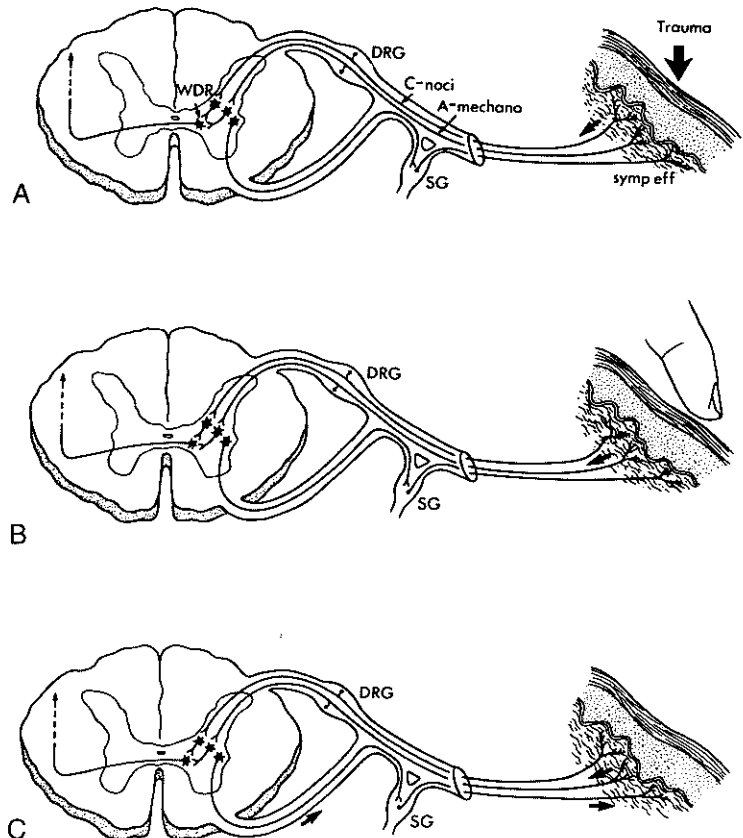
ciceptors or mechanoreceptors mediated by A-delta, C, or C polymodal nociceptors might excite wide dynamic range (WDR) neurons in the dorsal horn of the spinal cord and increase their sensitivity. Normally these neurons are thought to be partly responsible for rostral pain impulse transmission. Further stimulation of sensitized WDR neurons from peripheral stimuli, for example, touch mediated via low-threshold mechanoreceptors and A-beta fibers, might result in central transmission of a "pain signal" (Fig. 1).<sup>1, 12, 14, 15</sup>

Although the role of the different receptors and afferent neurons in normal nociceptive transduction and allodynia is controversial, the dystrophic changes of RSD/causalgia are more difficult to attribute to sympathetic dysfunction directly.<sup>1-3, 12-14, 17</sup> Many investigators believe that they are purely the result of disuse atrophy, and others suggest that they result from diminished regional blood flow as a result of sympa-

thetic dysfunction.<sup>5, 9, 12-15</sup> However, the dystrophic changes may appear earlier and be more significant than one might expect from the initial injury or pure disuse atrophy, prompting the idea that the sympathetic system may play some role, but what this is remains unclear.<sup>13</sup> Bonica<sup>1</sup> stresses that much of the confusion relating to these syndromes stems from "the insistence of writers to explain the entire symptomatology [of causalgia/RSD] on a single physiopathologic process." Therefore, until further delineation of the mechanisms is evident, we should consider sensitization of spinal neurons *and* sympathetic stimulation of afferent neurons as possible causal elements in the pain of the SMPs.

### Diagnosis of SMP

The diagnosis of SMP is made on empirical grounds using the existence of the clinical features and response to sympathetic



**Figure 1.** Mechanism of origin of "sympathetically maintained pain" proposed by Roberts.<sup>14</sup> A, Peripheral or cutaneous trauma produces impulses in nociceptive C-fibers (C-noci), activating wide dynamic range (WDR) neurons in the spinal dorsal horn, which project a "pain" signal centrally in the contralateral anterolateral pathway. B, The WDR neurons begin to respond to input from normally non-nociceptive fibers, such as the A-mechanoreceptor (A-mechano). C, The A-mechanoreceptors develop sensitivity to adrenergic neurotransmitters released peripherally by sympathetic efferents (symp eff). A regenerative cycle is then developed such that activity in A-mechanoreceptors produces pain sensation by activation of sensitized WDRs, producing reflex sympathetic activation in the segment, which produces more A-mechanoreceptor activity. (DRG = dorsal root ganglion, SG = sympathetic ganglion).

blocks already described. As indicated previously, many different sets of diagnostic criteria have been used, but all should incorporate these classic features.<sup>1, 5, 6, 9, 14-16</sup> Many investigators suggest that establishment of the RSD/causalgia diagnosis is dependent on a response to sympathetic blockade and, indeed, some use this as a diagnostic criterion.<sup>1, 13, 14</sup> Roentgenography was first used in the diagnosis of SMP in 1942 and has shown osteoporosis with patchy demineralization and subperiosteal bone resorption in as many as 80% of patients with suspected RSD.<sup>5</sup> However, these changes may be neither highly sensitive nor specific.<sup>9, 17</sup> Bone scintigraphy, particularly three-phase radionuclide scanning, has proved very sensitive and specific and has been reported to be positive in 83 to 96% of patients.<sup>2-5, 9</sup> Assessment of sympathetic tone can be performed by analysis of skin temperature by thermography, skin conductance (moisture) by sympathogalvanic response or sweat test, and blood flow by plethysmography or xenon clearance methods. Again, by definition, sympathetic blockade should be performed as a diagnostic test in patients suspected of having SMP.<sup>1, 7, 13, 15, 17</sup> However, it should be remembered that false-positive and false-negative results are possible as a result of over- or underanesthetizing the region, with loss of somatic nerve function or inadequate sympathetic blockade, respectively.<sup>1, 3, 13</sup> Therefore, one must perform sensory (somatic) testing and sympathetic monitoring (temperature monitoring, Horner's syndrome) before validating the results of the block.<sup>1, 3, 7, 13</sup> This is the most definitive diagnostic test and should be considered early on to facilitate early treatment. Use of sympathetic blocks as well as relevant laboratory studies may help in the differential diagnosis of SMP from arthropathies, scleroderma, de Quervain's disease, carpal tunnel syndrome, and other joint or connective tissue diseases.<sup>6</sup>

### Treatment of SMP

The mainstays of therapy in SMP are interruption of sympathetic efferent activity combined with aggressive physical therapy and rehabilitation. Success rates with

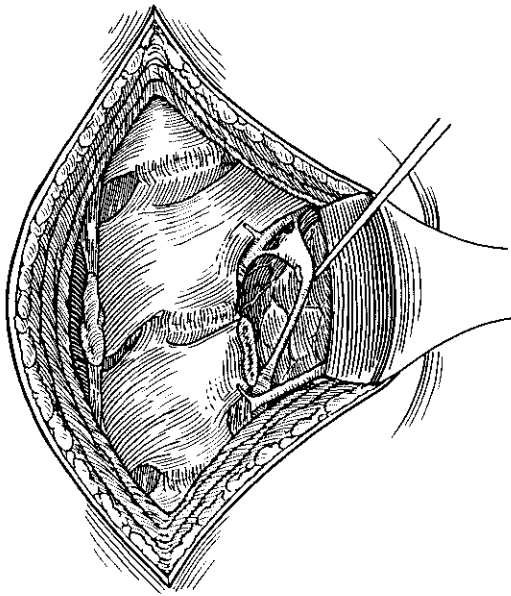
chemical or surgical sympathetic block have ranged from 12 to 90%. This most likely reflects the variance in diagnostic criteria, the effectiveness of sympathectomy, and the natural history of SMP.<sup>1, 13</sup> Some investigators suggest that the burning pain will not be relieved by sympathectomy unless hyperpathia is present.<sup>7</sup> Many investigators believe that sympathetic blockade is effective as a primary treatment of RSD, with relief reported in as many as 95% of cases, whereas rates of success in causalgia have ranged from 50 to 100%.<sup>1, 3, 6, 7, 13, 16</sup> The rate of spontaneous remission of symptoms in RSD/causalgia has been reported to be less than 5%. This fact, coupled with the devastating consequences of treatment delay, highlights the importance of rapid diagnosis and therapy.<sup>1, 13, 16</sup> By the same token, early treatment of SMP, particularly causalgia, may be the single most important variable in determining outcome.<sup>1, 13, 16</sup>

Sympathetic efferent interruption can be accomplished by the following methods: (1) sympathetic ganglion blockade, (2) oral sympatheolytic agents, (3) regional blockade, and (4) surgical sympathectomy.

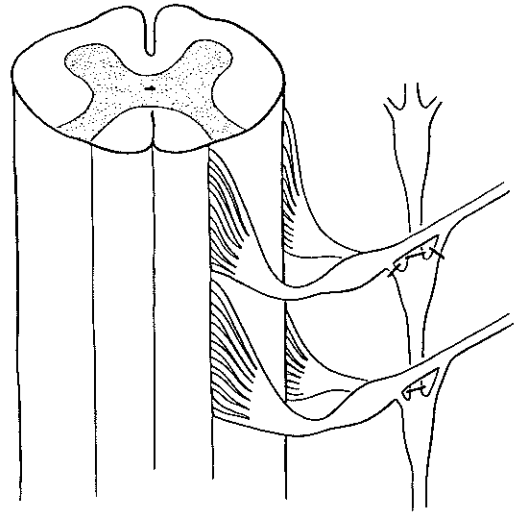
**Sympathetic Ganglion Blockade.** Sympathetic blocks are performed at the stellate ganglion for head, neck, and upper extremity SMP; the lumbar sympathetic ganglia for lower extremity and pelvic region SMP; and the celiac plexus for abdominal SMP.<sup>13, 16</sup> The response should be dramatic, with loss of pain and increased temperature immediately followed, possibly, by diminished swelling and cyanosis with functional improvement over a period of hours to days. The response rate is dependent, in part, on the skill of the physician, the completeness of the block, and the timing of the block.<sup>1</sup> It appears that the earlier the block after the onset of pain, the more effective it is.<sup>13</sup> However, randomized studies have not been performed, and the apparent effects of early blockade may simply reflect the natural history of the disorder. The duration of relief should outlast the local anesthetic effect. If successful, the blocks should be repeated at least 10 to 12 times or, as Long<sup>8</sup> suggests, daily for several weeks to facilitate complete "cure," which has been reported in 18 to 50% of patients.<sup>1, 6, 8</sup>

If anesthetic blocks are successful but of-

fer only transient relief of pain, particularly in patients with stage II or III SMP, more lasting sympathetic interruption by chemical or surgical means should be considered.<sup>2, 19</sup> Chemical sympathectomy is performed in a similar fashion as the blocks using neurolytic phenol (6–10%) or alcohol (50–100%).<sup>6, 13</sup> Surgical sympathectomy, initially performed for pain relief by Spurling in 1930, is most commonly performed by the posterior approach (Smithwick, 1940) (Figs. 2 and 3) or the transaxillary approach (Kleinert, 1965) with removal of the second thoracic ganglion for relief of upper extremity symptoms.<sup>6</sup> Lower thoracic sympathectomy, or splanchnicectomy is performed by resection of the sympathetic chain from T9 through T12 (Figs. 4 and 5). This procedure usually is done for visceral pain related to the heart or abdominal organs, the rationale being that visceral nociceptive afferents travel through the sympathetic chain at this level. However, this operation does produce sympathetic denervation as well, which may, in fact, be the important factor in the



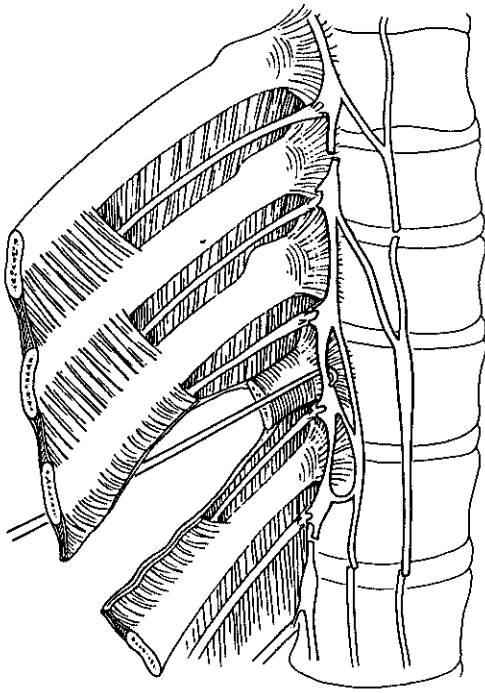
**Figure 2.** Dorsal T2 sympathectomy. The T3 rib head and transverse process are removed, and the nerve root of T2 is identified. Distal to the dorsal ramus of the nerve root, the white and gray rami are identified and followed down to the paravertebral sympathetic ganglion. The ganglion lays on the pleura and is frequently invested in adipose tissue. The sympathetic chain is retracted with a nerve hook.



**Figure 3.** Schematic of the T2 dorsal sympathectomy. The T2 sympathetic ganglion is removed from the chain by division proximal and distal to the ganglionic swelling, with division of the white and gray rami communicantes to T2, which may number two or more.

relief of pain of visceral origin. Lumbar sympathectomy is via the lateral flank extraperitoneal approach with resection of the second through fourth lumbar ganglia (Figs. 6 and 7).<sup>1, 6</sup> Postsympathectomy pain, “sympathalgia,” which is often self-limiting, may occur in 20 to 44% of patients between 10 and 14 days after operation.<sup>3, 13, 15</sup> Failure to respond may indicate incomplete sympathectomy or late-stage disease, and recurrence of pain following sympathectomy has been reported in as many as 30% of patients after as long as 8 years.<sup>1, 13, 17</sup> Recently, radiofrequency lesions have also been used effectively to perform thoracic chain ganglion destruction.<sup>13</sup>

Regional infiltration of an entire extremity, most commonly employing the Bier technique, has been used with local anesthetics, corticosteroids, and pharmacologic sympathetic blocking agents (reserpine, guanethidine, phentolamine) with success rates ranging from 50% for the former to 80% for the later.<sup>2, 7, 13, 17</sup> In SMP the predominant sympathetic receptor mechanism, particularly for vasoconstriction in the extremities, appears to be alpha-adrenergic. This has prompted the use of alpha-



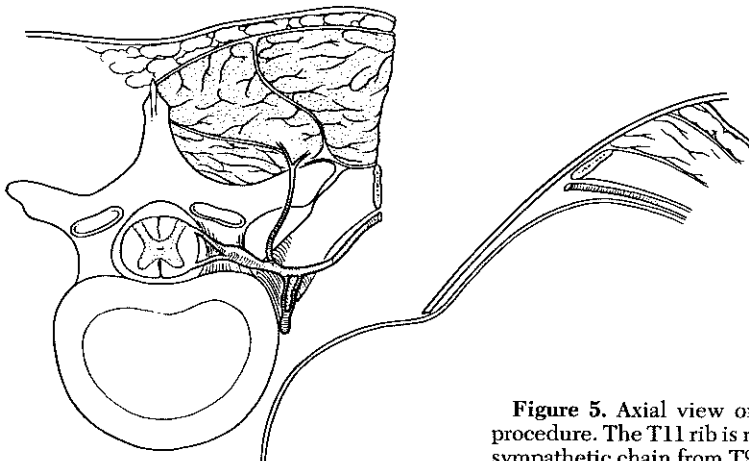
**Figure 4.** A T9 through T12 sympathetic gangliectomy or "splanchnicectomy" for sympathetic denervation of the heart and abdominal viscera. Visceral afferent fibers, which may mediate pain sensation, also are removed in this procedure. The T11 rib is removed, and the sympathetic chain, which may have multiple divisions, is identified and divided from T9 rostrally to T12 distally.

blocking oral sympatholytics (phenoxybenzamine, prazosin), with varying success rates reported.<sup>3, 6, 13</sup>

Other methods of therapy include the use of oral corticosteroids, nonsteroidal anti-

inflammatories, tricyclic antidepressants, anticonvulsants, calcium channel blockers, and narcotic analgesics. All of these, with the exception of narcotic analgesics, may be of benefit in this condition. Systemic corticosteroid therapy has been claimed to afford good response in more than 50% of cases after "failed" sympathectomy.<sup>5, 17</sup> Nifedipine, a calcium channel blocker, has been proposed for local relaxation of vascular smooth muscle and possible stabilization of calcium dependent neuronal "ectopy."<sup>13</sup> Other reports suggest that local percutaneous anesthetic "trigger" block or surgical correction of the primary irritating focus may help abate the vicious pain circle.<sup>1, 6, 9</sup>

Once an adequate pain-reducing or ablating procedure has been performed, aggressive physical therapy should be commenced after early pain relief and continued at home with range of movement and strength exercises. Immobilizing splints and casts should not be used, although dynamic hand splinting may be valuable. Heat therapy with paraffin glove as well as gentle active exercise and massage may help via stimulation of the large myelinated fibers with closing of the pain "gate."<sup>6, 10, 16</sup> This phenomenon, initially described by Mitchell<sup>11a</sup> who noted that his patients had relief of pain when they swathed their legs in cool damp clothes, is also used in contemporary neuromodulation techniques. Transcutaneous electrical nerve stimulation (TENS) may be of significant value as part of the overall therapy for SMP, particularly when used early in stage I, whereas implantable nerve stimulators may



**Figure 5.** Axial view of the T9 through T12 splanchnicectomy procedure. The T11 rib is removed, and the pleura is retracted. The sympathetic chain from T9 to T12 is removed.

