

Reflex Sympathetic Dystrophy

A Review

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● Reflex sympathetic dystrophy is a syndrome of burning pain, hyperesthesia, swelling, hyperhidrosis, and trophic changes in the skin and bone of the affected extremity. It is precipitated by a wide variety of factors in addition to nerve injury. It occurs outside of dermatomal distributions and can spread to involve other extremities without new injury. The diagnosis is primarily clinical, but roentgenography, scintigraphy, and sympathetic blockade can help to confirm the diagnosis. The most successful therapies are directed toward blocking the sympathetic innervation to the affected extremity, in conjunction with physical therapy. The theories proposed to explain the pathophysiology of reflex sympathetic dystrophy include "reverberating circuits" in the spinal cord that are triggered by intense pain, ephaptic transmission between sympathetic efferents and sensory afferents, and the presence of ectopic pacemakers in an injured nerve.

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In 1864, Mitchell et al,¹ and Mitchell,² in 1872, observed that soldiers with gunshot-wound injuries of peripheral nerves sometimes had persistent burning pain and progressive trophic changes in the affected limb. He called this syndrome *causalgia* because of the burning pain. Since that time, several similar clinical syn-

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dromes have been given different designations because of a predominant clinical feature or the precipitating insult (Table 1).^{1,9} All of these conditions have sympathetic hyperactivity associated with persistent pain, and respond to sympathetic denervation. Today the term *reflex sympathetic dystrophy* (RSD) is commonly used to encompass all of these variants.⁹⁻¹¹ Reflex sympathetic dystrophy is a syndrome of pain, hyperesthesia, vasomotor disturbances, and dystrophic changes that usually improves with sympathetic denervation.⁴

CLINICAL DESCRIPTION AND COURSE

Reflex sympathetic dystrophy is associated with a wide variety of precipitating factors (Table 2). The means by which all of these events

cause the same clinical syndrome is not yet known, but the common mechanism may be injury to either central or peripheral neural tissue, including peripheral nerve twigs.¹² The symptoms may begin gradually, days or weeks after the injury, or may manifest within a few hours.^{1,3,13,14} The patient suffers greatly and protects the affected area.^{2,9,15} This disorder progresses in stages, each of which were originally described as being from three to six months in duration.⁹ However, the actual length of each stage can vary considerably, lasting anywhere from weeks to years.

Stages

Stage I (Acute).—The pain is more than that usually caused by the initial injury, has a burning or aching quality, and is increased by dependency of

Table 1.—Clinical Variant of Reflex Sympathetic Dystrophy (RSD)

Condition	Description
Causalgia	RSD symptom complex that occurs after peripheral nerve injury ^{1,2}
Minor causalgia	RSD symptoms with prominent hyperesthesia that occur after insult which does not cause demonstrable nerve injury ³
Major causalgia	RSD symptom complex that occurs after peripheral nerve injury ³
Mimo-causalgia	RSD symptom complex that occurs after insult to central nervous system ⁴
Sudek's atrophy of bone	RSD symptom complex that occurs after soft-tissue trauma with bony atrophy as predominant finding ⁵
Algoneurodystrophy	RSD symptom complex that occurs after minor trauma ⁶
Shoulder-hand syndrome	RSD symptom complex with "frozen shoulder" that occurs after myocardial infarction, cardiovascular accident, or cervical radiculopathy ⁷
Reflex dystrophy	RSD symptom complex ⁸
Reflex neurovascular dystrophy	RSD symptom complex ⁹

Table 2.—Precipitating Factors and Diseases Associated With Reflex Sympathetic Dystrophy

Peripheral
Soft-tissue injury ^{9,15-17,21,26,34,40,124,125}
Arthritides ^{9,124}
Infection ^{9,15,17,25,26}
Fasciitis, tendonitis, bursitis ^{9,16,22,40,124,126}
Venous or arterial thrombosis ^{16,21,26,124,127,128}
Fractures, sprains, dislocations ^{7,15,17,22,26,40,124}
Operative procedures ^{15-17,24,26,40,47,124,125,129-131}
Malignancy ^{21,132}
Aortic injury ¹³³
Myelography, spinal anesthesia ^{16,134}
Paravertebral alcohol injection ²¹
Postherpetic ^{9,127}
Brachial plexopathy, scalenus anticus syndrome ^{21,124}
Radiculopathy ^{9,13,16,17,124,135-137}
Immobilization with cast or splint ^{35,47,49,56-58,138}
Vasculitis ^{9,127}
Myocardial infarction ^{9,11,17,34,36,40,127}
Weber-Christian disease ^{9,127}
Polymyalgia rheumatica ¹³⁹
Pulmonary fibrosis ¹⁷
Central
Brain tumor ^{9,17,140,141}
Severe head injury ¹⁷
Cerebral infarction ^{9,11,17,21,124,127,142-144}
Subarachnoid hemorrhage ¹⁷
Cervical cord injury ¹⁷
Subacute combined degeneration ²¹
Syringomyelia ^{11,21}
Poliomyelitis ^{21,26,124}
Amyotrophic lateral sclerosis ¹¹
Other
Idiopathic ^{7,9,15,17,23,24,40,127,145}
Prolonged bedrest ¹⁷
Familial ¹⁴⁶

Table 3.—Treatments Used in Reflex Sympathetic Dystrophy

Cold, wet compresses ^{1,2}
Hot wax applications ¹⁴⁷
Anti-inflammatory agents ^{23,126}
Radiation therapy ³⁴
Ultrasound to the stellate ganglion ⁶⁰
Hypnosis ¹⁴⁸
Electroacupuncture ^{149,150}
Immobilization with cast or splint ⁴³
Calcitonin ³⁹
Thalamotomy ¹⁵¹
Physical therapy ^{18,35,46,60,61}
Corticosteroids ^{6,36,53,152,153}
Transcutaneous nerve stimulation ^{21,45,63-65}
Propranolol ⁵⁹
Phenoxybenzamine ⁶⁶
Bier block with guanethidine sulfate ^{69-76,154}
Bier block with reserpine ^{79,83}
Bier block with lidocaine hydrochloride and corticosteroids ¹⁹
Paravertebral sympathetic ganglion block ^{3,22,38,42,44,77,78,84-86}
Continuous paravertebral sympathetic ganglion block ^{88,89,95,155}
Periarterial sympathectomy ^{49,87}
Paravertebral sympathetic ganglionectomy ^{4,15,18,22,30,31,62,87,90-94}

the affected part, physical contact, or emotional upset. Edema, hyperthermia or hypothermia, and increased hair and nail growth occur in the affected part. Bony changes may be present on roentgenograms.

Stage II (Dystrophic).—The edematous tissue becomes indurated and the skin is cool and hyperhidrotic, with livedo reticularis or cyanosis. Hair loss occurs. The nails are ridged, cracked, and brittle. The pain is con-

stant and is increased by any stimulus to the affected part. Roentgenograms may reveal diffuse osteoporosis.

Stage III (Atrophic).—The pain spreads proximally, and irreversible tissue damage occurs. The skin is thin and shiny, and the fingertips are wasted. The fascia becomes thickened, and flexion or Dupuytren's contractures may occur. Roentgenograms show marked bony demineralization and ankylosis.^{9,16}

Mitchell et al¹ and Mitchell² thought that the condition was self-limited, but many cases persist for years.¹⁵⁻¹⁹ The cases that resolve spontaneously tend to recur weeks or months later.^{3,9} Reflex sympathetic dystrophy can exhibit the signs of sympathetic hyperactivity to a minimal degree while still causing severe pain. This usually takes the form of slight or intermittent swelling and mottling in association with the characteristic burning pain. One can also see marked mottling and decreased skin temperature with intermittent burning pain and minimal hyperpathia. These partial forms are more common than the full-blown syndrome, as described in the literature concerning stage II and stage III.^{4,8,15} When RSD is precipitated by a peripheral nerve injury, the symptoms quickly spread outside the distribution of the damaged nerve.²⁰

In patients with RSD of other origins, the symptoms usually start distally and spread proximally. In some cases, additional extremities become involved without the advent of a new injury.^{9,10,17,21-23} One case has been reported in which RSD spread to involve the entire body.²⁴

Patients with RSD seem emotionally unstable, anxious, and socially withdrawn.^{22,25} Chronic invalidism, drug addition, suicide, and psychiatric commitment occur.²¹ The combination of the emotional sequelae of the illness and the disparity between the degree of pain and the physical examination lead many physicians to think that the pain is psychogenic.^{15,26,27} This, in addition to misguided therapeutic efforts and intractable pain, further aggravate the patient's psychologic symptoms.^{15,28} Anxiety increases sympathetic discharge, which exacerbates the pain.^{16,29,30} In patients observed before and after relief of RSD, the emotional disturbances resolved with successful treatment of their condition.^{20,31,32} No significant differences have been found in the personality traits of patients with RSD when compared with patients with nerve injuries without RSD.³³

Multiple studies have addressed the frequency and risk factors for RSD.^{14,16,31,34-38} The frequency of RSD after peripheral nerve injury ranges from 1% to 15%.^{12,37,39} The occurrence of RSD after myocardial infarction has dropped to less than 1%.^{34,36} The frequency of RSD after fractures, sprains, and trivial soft-tissue injuries has not been ascertained, although these are probably the most common precipitating causes. Some authors have reported that RSD

occurs most commonly in women over the age of 50 years.^{9,15,31} In a study of the incidence of RSD in veterans with peripheral nerve injuries, Rothberg et al³⁷ found a rate of 10% to 15% in patients 17 to 34 years old and 47% in patients 35 years of age and older. The series of 140 cases reported by Pak et al⁴⁰ and the series of 61 cases reported by Drucker et al¹⁶ were evenly distributed among all age groups. In both series, the male-to-female ratio was approximately 2:3.

This syndrome also occurs in children and has the same clinical manifestations and therapeutic responses as those that are seen in adults.^{35,38,41-46} The youngest patient reported was a 3½-year-old boy.⁴⁴ Some authors feel that children are more responsive to conservative treatment than are adults.^{35,41,43,45,46} Other authors disagree, and they recommend the early initiation of aggressive therapy.^{38,42,44}

Diagnostic Tests

The diagnosis of RSD is primarily clinical.^{15,47,48}

Roentgenographic studies were the first to confirm this disorder.⁴⁹ The findings include patchy demineralization of the epiphyses and the short bones of the hands or feet.^{49,50} Soft tissues may be swollen and reticulated in appearance. Fine-detail roentgenography reveals subperiosteal bone resorption, striation, and tunneling in the cortices, as well as large excavations and tunneling of the endosteal surface. These changes are not specific for RSD and may also be seen in hyperparathyroidism, thyrotoxicosis, and other conditions associated with increased bone turnover.^{51,52}

Scintigraphy with agents containing technetium Tc 99m demonstrates increased periarticular uptake in the involved extremity.^{48,51-54} Kozin et al⁵³ compared the sensitivity and specificity of roentgenography and scintigraphy in cases of RSD. The specificity of roentgenography was 71%, while that of scintigraphy was 86%. The sensitivity of roentgenography was 69%, and that of scintigraphy was 60%.⁴⁸

The best diagnostic approach to confirm the presence of RSD is the use of differential neural blockade. For the upper extremity, a needle is placed next to the stellate ganglion, and 8 mL of normal saline is infused (placebo). If no pain relief occurs after ten to 15 minutes, 8 mL of 1% procaine hydrochloride is then injected, which blocks only the sympathetic fibers to the arm. If pain relief is still not achieved, the needle is removed and placed into the brachial plexus

sheath, and 20 to 30 mL of 1% procaine hydrochloride is injected. If the pain persists, it must, therefore, be of central origin. For the lower extremities, one can perform epidural spinal blocks with the following solutions injected in sequence at ten-minute intervals: (1) 5 mL of normal saline (placebo), (2) 5 mL of 0.2% procaine hydrochloride (critical sympathetic concentration), (3) 5 mL of 0.5% procaine hydrochloride (critical sensory concentration), and (4) 5 mL of 1% procaine hydrochloride (critical motor concentration). The lowest concentration of procaine that relieves the patient's pain will determine whether the pain is sympathetic, peripheral somatic, or central in origin.⁵⁵

Treatment

Over the past 120 years, a wide variety of therapies have been recommended for the treatment of RSD (Table 3). All therapies that have proved effective are aimed at blocking the effects of sympathetic hyperactivity. While the other treatments listed have isolated reports of success, none have been proved effective in large studies. In particular, we would recommend against the use of casting or immobilization in RSD due to the evidence that this procedure exacerbates the problem.^{35,38,47,49,56-59}

Physical therapy alone has been shown to be effective in the treatment of RSD.^{18,35,47,60,61} The exercises are directed toward improving the mobility of the affected extremity. If the lower extremity is involved, therapy involves gradually increasing the weight-bearing capability of the limb. However, patients are usually in too much pain to participate in physical therapy unless adequate pain relief can be obtained prior to the initiation of activity.^{6,30,33,37}

Transcutaneous nerve stimulation (TNS) is postulated to relieve pain by an artificially generated barrage of nerve impulses in large axons.⁶² One study demonstrated its effect in altering sympathetic tone by raising skin temperature in normal subjects.⁶³ Another study found no alteration in skin temperature, blood flow, or other autonomic functions either in normal subjects or in patients with intractable pain, although some of these patients experienced pain relief.⁶⁴ Two isolated cases of RSD in children have been successfully treated with TNS.^{41,45} A series of eight patients found that TNS provided long-lasting relief in 25% of those observed, transient relief in 50% of those observed, and no relief in 25% of those observed.⁶⁵

The results of treatment with corticosteroids have been examined in several studies. One series of 17 patients received an average prednisone dose of 20 mg/d for ten to 70 weeks (average, 26 weeks). The responses were excellent or very good in 41% of patients, good or fair in 35% of patients, and poor in 24% of patients.⁶ A series of 15 patients treated with 100 to 200 mg of prednisone daily for two weeks, which was gradually tapered, reported good to excellent results in 67% of patients, with no long-term follow-up.¹⁷ In a series of 53 patients treated with 60 to 80 mg/d of prednisone, which was tapered over three to four weeks, 63% of those observed achieved a good to excellent response, and 29% of those observed achieved a poor response. Several patients who achieved good responses later required retreatment.⁵³ These studies indicate that prolonged treatment with high-dose corticosteroids may be beneficial in the treatment of RSD. Therapy with corticosteroids should be considered for patients who refuse or cannot tolerate treatments that directly block sympathetic activity.

Recently, a series of 27 patients with acute RSD (of less than six weeks' duration) have been treated with phenoxybenzamine. These patients received phenoxybenzamine daily (40 to 120 mg for six to eight weeks), which produced total resolution of their symptoms. Three patients required resumption of increased doses during the tapering period because of recurrence of pain. Follow-up ranged from six months to six years, and no recurrences were reported. Orthostatic hypotension was the major side effect reported.⁶⁶

Bier block is a technique utilized for regional anesthesia. The limb is elevated and isolated from the systemic circulation by a tourniquet. An anesthetic or other substance is then injected into the limb intravenously for five to 15 minutes, after which time the tourniquet is removed. Bier blocks with lidocaine hydrochloride block free nerve endings in tissue.^{67,68} Poplawski et al¹⁹ used Bier blocks with lidocaine hydrochloride (Xylocaine hydrochloride) and hydrocortisone sodium succinate (Solu-Medrol) in 28 patients with RSD, 57% of whom improved to the point where only occasional, if any, analgesics were needed, 18% of whom had some benefit, and 25% of whom had no response. All patients whose therapy yielded no benefit had been symptomatic for more than nine months prior to treat-

ment. Most patients required a series of two to three injections for prolonged relief.

Hannington-Kiff⁶⁹⁻⁷² developed a method of regional sympathetic blockade using Bier blocks performed with 10 to 20 mg of guanethidine sulfate. Guanethidine displaces norepinephrine in presynaptic vesicles and prevents its reuptake.^{72,73} Excellent results for pain relief utilizing this technique have been reported by many authors.^{30,73-75} The pain relief usually lasts from 12 to 36 hours, but it can be as long as six months.⁷⁵ This treatment is most effective in patients who have hyperpathia and hyperesthesia as the prominent symptoms.^{70,73,76-78} In a series of 20 patients who were followed-up for three months, guanethidine administered by Bier block was compared with therapy by stellate ganglion block. Guanethidine was equally effective for pain relief and was slightly longer lasting.⁷⁶ In another series of 47 patients, 21% of those observed received no benefit, 51% of those observed had less than 24 hours of pain relief and, in 13% of those observed, the pain relief lasted more than six months. The greatest effect was noted in patients with marked hyperesthesia.⁷⁵ These studies suggest that this mode of therapy can be beneficial to some patients, but it may be less effective than stellate ganglion blocks.

Reserpine injected intra-arterially is effective in the relief of pain and vasospasm associated with Raynaud's phenomenon and frostbite.⁷⁹⁻⁸² Reserpine interferes with the storage of norepinephrine, thereby causing its gradual depletion in nerve endings. Two patients with RSD were successfully treated with Bier block using 1 to 2 mg of reserpine.⁸³ In a series of 21 patients, 76% of those observed had benefited initially from this treatment, and 24% of those observed had no response. Of the patients with a positive response, 25% had recurrence of symptoms within a period of two weeks to three months.⁷⁹

Homans³ was the first to describe the use of paravertebral sympathetic ganglion blockade to treat RSD. Paravertebral sympathetic ganglion block is now the most widely recommended treatment for RSD.^{3,22,25,38,44,77,78,84-86} In a series of 69 patients, serial ganglionic blocks achieved excellent results in 32% of the patients observed, some benefit in 49% of those observed, and no benefit in 19% of those observed. The same study compared another group of 13

patients treated with corticosteroids and a group of 14 patients treated with ganglionic blocks, finding the blocks more effective. Duration of follow-up was not reported.⁸⁵ Another series of 32 patients found that 63% of those observed had definite improvement, although in only a third were the effects permanent.¹⁸ In a series of 26 patients undergoing follow-up for a period of three years, excellent results were noted in 32% of patients, and good results were noted in 50%; two recurrences were reported. Similar results were achieved with corticosteroid therapy.⁹ In a series of 91 patients, 50% reported complete or adequate relief of pain.⁸⁷ Overall, serial sympathetic ganglion blocks lead to definite, if transient, improvement in most patients and are probably more effective than administration of systemic corticosteroids.

Infusions of local anesthetic by an indwelling catheter have been utilized to achieve prolonged paravertebral sympathetic blockade. In a series of 160 patients, 57% were noted to have an excellent response, and 27% were noted to have a good response but, in the more severe cases, therapy by local anesthetic yielded only temporary palliation.⁸⁸ Another series of 25 patients reported improvement in 90% of those observed, with a 25% relapse rate over a period of three years.⁸⁹

Periarterial sympathectomy has been used successfully to treat RSD, but it is less effective than paravertebral ganglionectomy.^{49,87}

Paravertebral sympathetic ganglionectomy is recommended for those patients in whom only transient relief occurs with ganglion blocks.^{22,90} Several major series have examined the results of paravertebral sympathectomy in this situation, and 58% to 100% of the patients observed (average, 87%) had complete relief of symptoms after ganglionectomy. The follow-up period in these series ranged from six months to 17 years.^{4,15,18,30,31,87,91-93} Those patients whose pain is not relieved by ganglionectomy usually have incomplete sympathetic denervation or severe, long-standing disease.^{4,18,26,91,94} Erdemir et al⁹⁴ suggest that in patients with RSD of the lower extremity epidural sensory blocks should be performed to determine the exact level of ganglionectomy that will be required for complete pain relief. Evans⁹² pointed out that in patients with nerve entrapment causing RSD, complete relief could only be obtained by ganglionectomy and release of the entrapped nerve.

The most important factor in the effective treatment of RSD is the early recognition and treatment of the disease. Patients with long-standing disease are less likely to recover.^{4,18,19,22,26,62,84,91,95-97} Early physical therapy facilitates recovery from the physical disability associated with RSD and also plays a role in providing a prolonged response to other modes of therapy.^{18,19,26,62,84,92,96-98}

In our experience, patients with early or partial RSD respond well to serial paravertebral ganglion blocks. If the symptoms recur, another series of blocks can be performed. Paravertebral ganglionectomy should be considered if there are further recurrences. Bier blocks with either lidocaine and corticosteroids or with reserpine (or guanethidine, if available) can be effective when symptoms recur after ganglionectomy. Patients with both lower extremities affected should be treated with epidural sympathetic blocks. For patients with severe or long-standing disease, sympathectomy should be performed early if there is any response to a paravertebral ganglion block. For all patients, physical therapy to mobilize the extremity should be performed as soon as the pain has been sufficiently reduced to allow the patient to cooperate. The patient should also be encouraged to move the extremity as much as possible without increasing the pain.

Pathophysiology

Many hypotheses have been proposed to explain the mechanism responsible for RSD. A proposed mechanism for RSD must account for the following phenomena: (1) spontaneous burning pain, (2) hyperalgesia, (3) hyperpathia, (4) vasomotor disturbances, (5) exacerbation by emotional upset, (6) occurrence either spontaneously or after minor injury, (7) occasional spontaneous resolution, (8) spread to other parts of the body, and (9) relief by sympathetic denervation. No single hypothesis proposed to date explains all of the features of RSD.

In 1943, Livingston⁹⁹ proposed the so-called theory of reverberating circuits in the spinal cord to explain the phenomenon of RSD. He suggested that intense, painful stimuli initiate these reverberating circuits in the internuncial neuron pools of the spinal cord. Once established, these reverberating circuits can also be triggered by normal stimuli and are interpreted centrally as pain. Livingston did not propose a specific mechanism.

In 1944, Doupe et al⁹ proposed that the pain of RSD was caused by activation of sensory fibers by sympathetic efferents. They supported this proposal with several astute clinical observations. In 1947, Nathan¹⁰⁰ presented considerable clinical evidence in support of the hypothesis that, in those patients with RSD after partial nerve injury, abnormal stimulation of somatic sensory axons occurs in the damaged area of the nerve. This stimulation is caused by efferent impulses from postganglionic sympathetic nerves. Nathan suggested that artificial synapses are formed at the site of the lesion and allow ephaptic transmission to occur between efferent and afferent fibers. The pain caused by this process is referred to the distribution of the sensory nerve. He did not address the issue of the spread of symptoms out of a dermatomal distribution.

In 1959, Drucker et al¹⁶ observed that RSD can result from minor soft-tissue injuries as well as clinically demonstrable nerve injury, and that in both cases the pain and vasomotor changes were identical and responded well to sympathectomy. Because of these facts, they proposed that minuscule peripheral-nerve twigs could be damaged in soft-tissue injury and form artificial synapses in the same manner as major nerve trunks. This results in ephaptic transmission between sympathetic efferents and sensory afferents, which, in turn, increases input into the spinal cord and increases the activity of the internuncial neuron pool. They hypothesized that these neurons stimulate anterolateral sympathetic efferents causing a further increase in the activity of the peripheral ephaptic synapse. Thus, a vicious cycle of pain and sympathetic hyperactivity is established. These authors also noted that the activity of the internuncial neuron pool could be inhibited or stimulated by input from the cerebrum or hypothalamus.

In 1965, Melzack and Wall¹⁰¹ developed the *gate-control theory of pain*. In this theory, these authors specifically mentioned causalgia as a phe-

nomenon that must be accounted for by any theory of pain. They proposed that cells in the substantia gelatinosa functioned to modulate sensory input. Impulses from large sensory fibers initially stimulate the second-order neuron but are then quickly inhibited. This exerts a phasic control of the second-order neuron. Input from small pain fibers stimulate the second-order neuron less easily but, once this is accomplished, a positive feedback system is triggered that causes tonic stimulation. Incoming volleys from large sensory fibers also inhibit the tonic small fibers. These systems operate to a greater or lesser degree at all times, and the information that is relayed centrally is a summation of this activity. Descending inhibition also plays a role in modulation of these systems.

In 1971, Melzack¹⁰² proposed the existence of a central biasing mechanism. He stated that a portion of the brain-stem reticular system exerts a tonic inhibitory influence on transmission at all levels of the somatic projection system. A decrease in sensory input after nerve injury or section decreases tonic inhibition and increases the probability of self-sustaining neural activity. He postulated that prolonged pain may leave "memory traces" in the somesthetic system, making an individual more susceptible to recurrent pain.

In 1976, Sunderland¹⁰³ proposed the *turbulence theory* to explain the mechanism of RSD. He suggested that injury to the postganglionic sympathetic efferents might cause both retrograde changes in the sympathetic ganglia and transsynaptic degeneration in the spinal cord. This would impair the function of whole groups of neurons in the spinal cord, which could then form self-sustaining circuits.

In 1983¹² Devor presented the following hypothesis, which he supported with extensive experimental evidence. Any form of injury or inflammation damages Schwann's cells or the axons themselves, which results in local demyelination or sprout outgrowth. The sprout or

demyelinated segment incorporates excessive numbers of sodium and calcium channels as well as α -adrenergic receptors. Gating properties of existing channels may also change. These factors result in the acquisition of ectopic pacemaker capability and chemosensitivity by the demyelinated segment or sprout. This ectopic pacemaker discharges spontaneously as well as in response to any depolarizing stimulus. Circulating catecholamines and those released from sympathetic efferent sprouts activate the ectopic pacemaker and augment the discharge. The hyperalgesia and abnormal chemosensitivity in the skin may reflect similar membrane changes in the cutaneous axon terminals.

Increased firing of the peripheral nerves, their increased sensitivity to electrical and chemical stimulation, and altered receptive fields in the spinal cord after nerve injury have been demonstrated experimentally.^{12,65,104-114} It has also been shown that abnormalities of vasomotor tone are caused by abnormally responsive peripheral sympathetic efferents.^{100,115-121} The role of medullary and cortical centers in modulating pain and vasomotor activity have also been demonstrated experimentally.^{99,122,123} Thus, based on current experimental evidence, it appears that RSD may result from abnormal firing of peripheral nerves due to increased sensitivity, as described by Devor¹² This would account for the spontaneous pain, allodynia, and hyperesthesia seen clinically. This abnormal firing may, in turn, cause altered responses by the neuronal pools in the spinal cord, which then respond abnormally to brain stem and cortical influences, as proposed by Melzack.¹⁰² These spinal and supraspinal mechanisms are likely responsible for the "centralization" of pain in patients with RSD, rendering it unamenable to treatment. These mechanisms may also be responsible for the occurrence of RSD after insults to the central nervous system. The exact contribution of each of these factors in the actual clinical syndrome has not yet been demonstrated.

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