

# **Complex Regional Pain Syndrome (CRPS) and Sympathectomy**

H. Hooshmand, M.D. and Eric M. Phillips

Neurological Associates Pain Management Center

Vero Beach, Florida

**Abstract.** Sympathectomy may provide temporary pain relief, but after a few weeks to months it loses its effect. Sympathectomy and the application of Chemical Sympathectomy (neurolytic agents e.g., phenol, alcohol, etc.) should be limited to patients with life expectancies measured in weeks or months - e.g., cancer patients.

Chemical Sympathectomy (e.g., alcohol, phenol or hypertonic saline nerve blocks) aimed at destroying the nerves are apt to fail, to cause serious complications, and aggravation of the pain - by leaving a large scar behind.

Complex Regional Pain Syndrome (CRPS) patients should not be exposed to aggravation of pain due to sympathectomy, chemical sympathectomy or radiofrequency sympathectomy.

**Keywords.** Complex Regional Pain Syndrome (CRPS), Chemical Sympathectomy, Reflex Sympathetic Dystrophy (RSD), Sympathectomy, and Radiofrequency Sympathectomy.

## **INTRODUCTION**

Sympathectomy has been applied for the treatment of causalgia since 1916 (1). The meta analysis of sympathectomy literature for treatment of Complex Regional Pain Syndrome (CRPS) shows high rates of failure. Long term follow-up of 8.4 years showed 13% success (2). Only young teenage soldiers undergoing sympathectomy and followed up to 26-60 days have very good results (3). The rest of the literature has reported a range of 12% up to 97% success rates. The high percentage group has been wartime soldiers which have been diagnosed early, undergone surgery within a few days, and sent home to be lost to follow-up (4-25). Realizing that children and teenagers (such as soldiers), show a strong plasticity and healing power as compared to adults, and realizing that early diagnosis and treatment is more successful, explain the beneficial, albeit temporary, results of wartime sympathectomy (26-29). In contrast, the sympathectomy done in stage III\* has been reported to show zero percent relief (30) (*Table 1*). Usually, by the time the physician resorts to the sympathectomy procedure, the patient is in advanced stages of the disease. In such late stages, the

nervous system has lost its plasticity and cannot respond properly to surgical sympathectomy (31, 32). More over, the disease has spread, to other parts of the body and a regional sympathectomy will not be of any benefit to the patient (31, 33-37).

## **SYMPATHECTOMY**

To quote Nashold, referring to sympathectomy, "Ill- advised surgery may tend to magnify the entire symptom complex"(38). Sympathectomy is aimed at achieving vasodilation. The neurovascular instability (vacillation and instability of vasoconstrictive function), leads to fluctuation of vasoconstriction alternated with vasodilation in an unstable fashion (39). Following sympathectomy the involved extremity shows regional hyper - and hypothermia in contrast, the blood flow and skin temperature on the non- sympathectomized side are significantly lower after exposure to a cold environment (39). This phenomenon may explain the reason for spread of CRPS. In the first four weeks after sympathectomy, the Laser Doppler flow study shows an increased of blood flow and hyperthermia in the extremity (40). Then, after four weeks, the skin temperature and vascular perfusion slowly decrease and a high amplitude vasomotor constriction develops reversing any beneficial effect of surgery (39). According to Bonica, "about a dozen patients with reflex sympathetic dystrophy (RSD) in whom I have carried out preoperative diagnostic sympathetic block with complete pain relief; sympathectomy produced either partial or no relief (40). Postoperative examination with a sweat test and psychogalvanic reflex revealed residual sympathetic function, and this was confirmed with subsequent sympathetic blocks which produced both sympathetic denervation and pain relief" (40). In the same page, Bonica wrote: "There are other possible explanations for failure of sympathectomy to relieve the pain and causalgia. One is that although sympathectomy relieves burning pain, it may not affect the deep, tearing, stabbing, and bursting pain" (40).

Also, to quote Livingston, referring to Dr. James Evans, report on sympathectomy for reflex sympathetic dystrophy. Dr. Livingston stated that Dr. Evans is correct in stating that when a preliminary procaine block of the sympathetics fails to afford temporary relief it is reasonable to assume that a ganglionectomy will probably fail. I would add the fact that even when the procaine block affords temporary relief the ganglionectomy may fail to confer a satisfactory result. In my opinion there are three reasons why this should be so: (i) The phase of active vasodilation which follows immediately after a block or a ganglionectomy does not persist for long, and although relief may be obtained during this primary phase, the pain may recur when the vessels regain some tone. (ii) Sympathetic nerves have a remarkable ability to regenerate. I have had the disappointing experience of seeing pain return a few weeks after ganglionectomy and have been able to demonstrate that within two to three months sympathetic fibers have successfully bridged a considerable anatomic gap. (iii) These so-called

sympathetic dystrophies begin as a result of irritation at some focal or "trigger point." A sympathetic ganglionectomy does not remove this source of irritation, and not infrequently the pain and many of the associated dystrophic phenomena will recur even in the absence of the sympathetic component (41).

In my opinion the most significant feature of this paper is the fact that 11 of 12 patients treated with procaine block alone were completely cured of their pain. These 11 patients represent almost 20 percent of this whole series. The fact that procaine blocks gives a satisfactory result without the need to resort to a surgical excision indicates to me that the syndrome represents a disturbed physiology which the injection acts in some mysterious manner to correct. I am always in favor of trying repeated procaine blocks of the sympathetics before resorting to the use of alcohol or a surgical excision of the ganglions for three reasons: (i) I hesitate to excise sympathetic ganglions which in their normal state must serve useful functions, some of which functions we may not understand. (ii) When a surgeon excises the sympathetic chain he has removed the point of attack at which the syndrome is most vulnerable. (iii) Since I believe that this is a disturbance of physiology, I would prefer to treat it with physiologic methods rather than surgical excisions. This paper emphasizes two points which are of great importance to the clinician who undertakes to treat painful dystrophies. One of these is that the sympathetic nerves play an important part in sustaining the dystrophy and represent the most logical point of surgical attack. The second point is equally obvious in this paper, that is, a sympathetic ganglionectomy is not a cure-all for painful dystrophies (41).

### **CHEMICAL SYMPATHECTOMY (ALCOHOL BLOCKS)**

Chemical Sympathectomy (Alcohol blocks) which are chemical blocks in the form of phenol, alcohol, etc., are the most dangerous and destructive forms of nerve blocks. They are also called "lytic" blocks which better describe them. The term "lytic" refers to "lysis" which refers to a meltdown of every soft tissue in the target area of the block including nerves, connective tissue, etc. This destruction is not limited to the area of injection-because nothing keeps the alcohol from destroying the "bad nerves", but it also destroys the adjacent perfectly normal nerves. Incidentally, intervention or destructive lytic nerve blocks or sympathectomy are done on damaged nerves. The nerve is nothing but the conveyer of the impulse. In CRPS, the disease originates from microscopic sensory nerves in the wall of the small blood vessels. The large trunk of the nerve fibers that are the target of nerve blocks or sympathectomy, are just the messengers. Destroying the messenger is not going to solve the problem, but it is going to add a new source of pain. Alcohol causes extensive scar formation of the soft tissues including the nerves and such scar formation becomes a new source of severe pain far worse than the original pathology. Alcohol blocks, sympathectomy, or neurectomy (cutting nerve fibers) only adds assault to the injury. Such destructive procedures

relieve the pain for a few weeks to a maximum two months, only for the pain to return with more intensity and in a larger area of the body.

Any destruction of nerve fibers should be definitely avoided. These procedures are all doomed to fail and are dangerous.

On the other hand, performing epidural nerve blocks or paravertebral nerve blocks which flood the nerves in the muscle or in epidural space with a combination of local anesthetic and a small amount of anti-inflammatory medication (such as Depo-Medrol ®, Kenalog, or Celestone) do not destroy the nerves. They simply block the input of painful chemical such as substance P from the extremity into the spinal cord. They don't anatomically destroy any of the nerve fibers and they provide excellent relief lasting anywhere from 2-3 months. In the meantime, during that 2-3 months with proper physical therapy and massage and other measures should preclude the necessity of repeating such nerve blocks in at least 80% of the patients.

Not only should surgical procedures, chemical sympathectomy and neurectomy be avoided, but also application of ice on the extremity by the virtue of destroying the myelin covering of the nerve (the protective sheaths of the nerve) should always be avoided (42).

All the above statements refer to benign, complex, chronic pain. Obviously, if the patient suffers from cancer and has a few months to live, any of these blocks will give the patient a few months of relief and are palliative. In cancer patients any surgical procedure that gives a temporary relief to the patient is justifiable, humane, and should be done. CRPS (RSD) patients do not suffer from cancer. They are quite young. They have 4-5 decades of life ahead of them, and should not be exposed to such destructive procedures which cause more pain than the original disease (4).

## **RADIOFREQUENCY SYMPATHECTOMY**

The most traumatic of all invasive treatments is Radiofrequency Sympathectomy (nerve ablation and block). It is done with a heat generating Radiofrequency electrode causing a boiling hot temperature at the target area which coagulates, destroys and kills the nerve fibers and nerve cells.

Because the Radiofrequency damage causes high temperature in the adjacent areas of the target, it also destroys the adjacent normal nerves causing a much larger lesion and scar formation with spread and aggravation of pain in a permanent fashion.

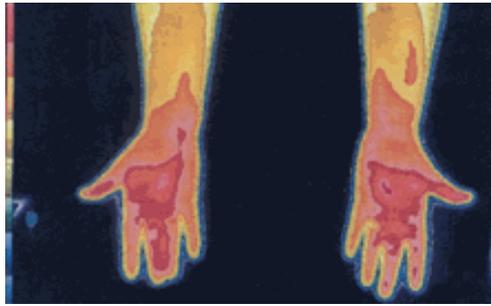
In CRPS the sympathetic system is dysfunctional rather than simply being hyperactive. The longer the disease is left untreated and the more surgical scars,

the more dysfunctional the sympathetic system becomes. This is the reason for practically 100% failure of treating CRPS with sympathectomy, radiofrequency, and chemical sympathectomy with phenol, alcohol, etc.

### **VIRTUAL SYMPATHECTOMY**

The hyperthermia maybe due to iatrogenic injury to the sympathetic ganglia. An example of the therapeutic role of Thermography is identification of Virtual Sympathectomy. After more than a dozen stellate or lumbar, ganglion nerve blocks, the repetitive needle insertion traumatizes the ganglion enough to result in permanent hyperthermia in the extremity ("Virtual Sympathectomy") (33,43) (*Figure 1*). Kozin, in his review of 500 patients treated with sympathetic ganglion blocks, reported "the majority of patients have transient or no significant pain relief" (44). Another meta-analysis of retrospective and prospective randomized controlled trials of 1144 patients revealed the local anaesthetic sympathetic blockade was as ineffective as placebo in treatment of CRPS (45).

Thermography identified the "virtual sympathectomy" phenomenon, and spared the patients from further damage by canceling the procedure (31) (Table 2 and 2A). Repetitive ganglion nerve blocks are routinely applied for diagnosis and treatment of neuropathic pain such as CRPS (46). However, Hogan et al, have reported only 27% of stellate ganglion block achieved the goal of ipsilateral warming to exceed the contralateral skin temperature (47). This 27% success is not worth the traumatic complications of ganglion blockade. Moreover, they noted that cervical paratracheal blocks frequently failed to produce evidence of sympathetic interruption to the arm (48). The sympathetic ganglion blockade done in peripheral occlusive vascular disease or CRPS maybe potentially dangerous and harmful (31, 44, 45, 48) (*Figure 1*).



**Figure. 1**

"Virtual sympathectomy" secondary to repeated stellate ganglion nerve blocks leading to permanent sympathetic nerve damage and hyperthermia (heat leakage) in upper extremities. The use of Thermography spared the patient from further sympathetic nerve blocks.

From: Thermology International. 2001; 11: 2: 53-65. (33)

**Table 1. CRPS Stages**

From: Pain Digest. 1999; 9: 1-24. (31)

**Stage I**

**Dysfunction:** with thermal changes, neuroinflammation, neurovascular instability, neuropathic pain, vasomotor and flexion spasm.

**Stage II**

**Dystrophy:** hair, nail, and skin trophic changes, bouts of hair loss, alopecia, skin rash, spontaneous subcutaneous bleeding, ulcerative lesions, edema, and entrapment neuropathy.

**Stage III\***

**Atrophy:** as well as fluctuating vital signs, visceral neuroinflammation, chest pain, neurovascular instability.

**Table 2.** The influence of treatment on CRPS stages during 2 years or longer follow-up in 824 patients. (Amputation or sympathectomy, deteriorate the disease from stage I to stage III.) From: Pain Digest. 1999; 9: 1-24. (31)

Characteristics of treatment (824 patients)	Stage I **** number of patients	Stage II number of patients	Stage III number of patients
History of Amputation * 11 Patients (1.3%)	0 (0%)	2 (19%)	9 (81%) (P=0.025)
Chemical Sympathectomy 13 Patients (1.5%)	0 (0%)	2 (15.4%)	11(84.6%)
Surgical Sympathectomy 22 Patients (2.6%)	0 (0%)	3 (13.6%)	19 (86.4%)
Surgical Treatment ** 295 Patients (36%)	24 (8%)	106(36%)	165 (56%) (P<0.001)

**Table 2A.** Surgical and Non-Surgical Group. From: Pain Digest. 1999; 9: 1-24. (31)

	Stage I	Stage II	Stage III
Surgical Group 320Patients	24.7% 79 Patients	33.13% 106 Patients	59.8%* 135 Patients
Non - Surgical Group 528 Patients	31% 164 Patients	36% 190 Patients	33% 174 Patients

\* Note high percentage of stage III in the surgical group

(\*) Many patients had more than one treatment modality, which change the total percentage.

(\*\*) Sympathectomy; rotator cuff; thoracic out syndrome; compression neuropathy; exploration; etc.

(\*\*\*) Stage I = Dysfunction; Stage II= Dystrophy; Stage III= Atrophy.

(\*\*\*\*) According to the type of treatment stage III may reverse to stage I and vice-versa.

## **SUMMARY AND CONCLUSION**

The use of Thermography showed failure of sympathectomy to relieve the vascular dysfunction. Thermal imaging done in patients who underwent surgical or chemical sympathectomy showed a high percentage of surgical failure (40, 49).

Early diagnosis in the first six months to maximum two years is the key to successful treatment (50). Surgical procedures have no place in treatment of CRPS. Sympathectomy or removal of a part of the chain of sympathetic ganglia (on the side of the spine) has an extremely high rate of failure. It has been reported to help the war type of CRPS, which is quite different from civil type. In the war type, the soldier is a young teenager who responds favorably to treatment, regardless of the mode of treatment. The war time CRPS is due to high velocity damage to the nerves in the proximal parts of the extremities and sympathectomy, even in these cases, has a high rate of failure in the long run. The civilian type of CRPS is due to a small damage of the sympathetic nerves in the central or peripheral nervous system.

If the patient lives longer than five years, the rate of failure from sympathectomy is over 80% (4). The scar of the surgical procedure becomes a new source of CRPS. Removal of a part of sympathetic ganglia does not prevent spread of the disease in the areas of the body where the sympathetic nerves have been removed. This is due to the fact that the adjacent sympathetic nerves eventually compensate for the lack of sympathetic function due to surgery.

A sympathectomy, be it surgical or chemical, is useless for advanced CRPS. It will cause rapid spread of CRPS to other parts of the body. A chemical sympathectomy is as destructive as a surgical sympathectomy. The surgical sympathectomy is at least clean and circumscribed. The chemical sympathectomy damages the surrounding normal tissues and causes more scarring and pain.

We have to realize that CRPS is not just a hyperactive sympathetic dysfunction but a distorted and pathological sympathetic dysfunction. That's why some patients have warmer extremities and some patients have colder extremities. The damage to the sympathetic ganglia, be it in the form of sympathectomy, chemical sympathectomy, and radiofrequency (all of these sympathectomies are cardinal sins), or repetitive stellate ganglion blocks are damaging (causing "Virtual Sympathectomy") and can complicate the chronic CRPS pain further rather than helping the patient.

## REFERENCES

1. Leriche R: De la causalgie, envisagee comme une nevrite du sympathique et de son traitement Par la denudation et l'excision des plexus nerveus periarteriels. Presse Med. 1916; 24:178-80.
2. Welch E, and Geary J: Current status of thoracic dorsal sympathectomy. J of Vascular Surgery. 1984; 1: 202-14.
3. Jebara VA, and Saade B: Causalgia: A wartime experience-report of twenty treated cases. J of Trauma. 1987; 27:519-24.
4. Hooshmand H: Chronic Pain: Reflex Sympathetic Dystrophy: Prevention and Management. CRC Press, Boca Raton FL. 1993.
5. Payne R: Neuropathic pain syndromes with special reference to causalgia and reflex sympathetic dystrophy. Clin J Pain. 1986; 2: 59-73.
6. Allbritten FF, Maltby GL: Causalgia secondary to injury of major peripheral nerves: treatment by sympathectomy. Surgery. 1946; 19: 407- 14.
7. Verrill P: Sympathetic ganglion lesions. In Wall PD, Melzack R, eds. Textbook of pain. Edinburg: Churchill Livingstone. 1984; 581-9.
8. Sweet WH, Poletti CE: Causalgia and sympathetic dystrophy (Sudeck's Atrophy) In: Aronoff GM, ed Evaluation and treatment of chronic pain. Baltimore: Urban and Schwartenberg. 1985; 149-65.
9. White JC, Heroy WW, Goodman EN: Causalgia following gunshot injuries of nerves. Ann Surg. 1948; 128: 161- 83.
10. Hardy WG, Posh JL, Webster JE, Gurdjian ES: The problem of minor and major causlgias. Am J Surg. 1958; 95: 545-4.
11. Tracy GD, Cockett FB: Pain in the lower limb after sympathectomy. Lancet. 1957;1: 12-4.
12. Berguer R, Smith R: Transaxillary sympathectomy (T2 to T4) for relief of vasospastic / sympathetic pain of the upper extremities. Surgery. 1981; 89: 764-9.
13. Mayfield FH: Causalgia. Springfield: Charles C. Thomas. 1951.
14. Buker RH, Cox WA, Scully TJ, Seitter G, Pauling FW: Causalgia and transthoracic sympathectomy. Am J Surg. 1972; 724-7.

15. Raskin NH, Levinson SA, Hoffman PM, Pickett JB, Fields HL: Post-sympathectomy neuralgia: amelioration with diphenylhydantoin and carbamazepine. *Am J Surg.* 1974; 128: 75-8.
16. Shumacker HB: A personal overview of causalgia and other reflex dystrophies. *Ann Surg.* 1985; 201: 278-89.
17. White JC, Sweet WH: *Pain and the neurosurgeon.* Springfield: Charles C Thomas. 1969.
18. Bonica JJ: *The management of pain.* Philadelphia: Lea and Feibiger. 1953.
19. Evans JA: Reflex sympathetic dystrophy; report on 57 cases. *Ann Intern Med.* 1947; 26; 417-26.
20. Barnes R: The role of sympathectomy in the treatment of causalgia. *J Bone Joint Surg [Br].* 1953; 35B:172-80.
21. Thompson JE, Patman D, Persson AV: Management of post-traumatic pain syndromes (causalgia). *Am Surg.* 1975; 41: 599-2.
22. De Takats G: Reflex dystrophy of the extremities. *Arch Surg.* 1937; 34: 939-56.
23. Wirth FP, Rutherford RB: A civilian experience with causalgia. *Arch Surg.* 1970; 100: 633-8.
24. Shumacker HB, Spiegel IJ, Upjohn RH: Causalgia I. The role of the sympathetic interruption in treatment. *Surg Gynecol Obstet.* 1948; 86: 76-6.
25. Shumacker HB, Spiegel IJ, Upjohn RH: Causalgia II. The signs and symptoms, with particular reference to vasomotor disturbances. *Surg Gynecol Obstet.* 1948; 86: 452-60.
26. Bernstein BH, Singsen BH, Kent JT, Kornreich H, King R Hicks R, et al: Reflex neurovascular dystrophy in childhood. *J Pediatr.* 1978; 93:211-5.
27. Wilder RT, Berde CB, Wolohan M, Vieyra MA, Masek BJ, Micheli LJ: Reflex sympathetic dystrophy in children. *J Bone Joint Surg.* 1992; 74: 910- 9.
28. Poplawski ZJ, Wiley AM, Murray JF: Post traumatic dystrophy of the extremities. *J Bone Joint Surg [Am].* 1983; 65:642-55.
29. Olcott C, IV , Eltherington LG, Wilcosky BR, Shoor PM, Zimmerman JJ, Fogarty TJ: Reflex sympathetic dystrophy- The surgeons role in management. *J Vasc Surg.* 1991; 14: 488-95.

30. AbuRahma AF, Robinson PA, Powell M, Bastug D, Boland JP: Sympathectomy for reflex sympathetic dystrophy: factors affecting outcome. *Ann Vasc Surg.* 1994; 8: 372-9.
31. Hooshmand H, Hashmi H: Complex regional pain syndrome (CRPS, RSDS) diagnosis and therapy. A review of 824 patients. *Pain Digest.* 1999; 9: 1-24.
32. Ochoa JL, Verdugo RJ: Reflex sympathetic dystrophy: A common clinical avenue for somatoform expression. *Neurol Clin.* 1995; 13: 351-63.
33. Hooshmand H, Hashmi M, Phillips EM: Infrared thermal imaging as a tool in pain management- An 11 year study", Part I of II. *Thermology International.* 2001; 11: 2: 53-65.
34. Schiffenbauer J, Fagien M: Reflex sympathetic dystrophy involving multiple extremities. *J Rheumatol.* 1983; 20:165-9.
35. Schwartzman RJ: Reflex sympathetic dystrophy and causalgia. *Neurol Clin.* 1992; 10: 953-73.
36. van der Laan L, ter Laak HJ, Gabreels-Festen A, Gabreels F, Goris RJ: Complex regional pain syndrome type I (RSD). Pathology of skeletal muscle and peripheral nerve. *Neurology.* 1998; 51: 20-5.
37. Radt P: Bilateral reflex neurovascular dystrophy following a neurosurgical procedure. Clinical picture and therapeutic problems of the syndrome. *Confin Neurol.* 1968; 30:341-8.
38. Nashold BS Jr, Friedman H: Neurosurgical relief of pain. 1507-11.
39. Wahren LK, Torebjork HE: Quantitative sensory test in patients with neuralgia 11 to 25 years after injury. *Pain.* 1992; 48:237-44.
40. Bonica JJ: *The Management of Pain.* Lea & Feibger Philadelphia. 1990; Vol. 1: p 229.
41. Evans JA: Sympathectomy for reflex sympathetic dystrophy: A case report of Twenty-Nine cases. *JAMA.* 1946; 132: 11: 620-23.
42. Hooshmand H, Hashmi M, Phillips EM: Cryotherapy can cause permanent nerve damage: A case report. *American Journal of Pain Management.* 2004; 14: 2: 63-70.
43. Hooshmand H: Is Thermal imaging of use in pain management? *Pain Digest.* 1998; 8: 166-70.

44. Kozin F: Reflex sympathetic dystrophy: a review. *Clin Exp Rheumatol*. 1992; 10: 401-9.
45. Carr DB, Cepeda MS, Lau J: What is the evidence for the therapeutic role of local anesthetic sympathetic blockade in RSD or causalgia? An attempted meta-analysis [abstract] Eighth world congress on pain, Vancouver, August 17-22 1966., Seattle: IASP Press . 1996; p 406.
46. Raj PP: Tutorial 16: diagnostic nerve block. *Pain Digest*. 1995; 5:25-34.
47. Hogan QH, Taylor ML, Goldstein M, Stevens R, Kettler R: Success rates in producing sympathetic blockade by paratracheal injection. *Clin J Pain*. 1994; 10: 139-45.
48. Schott GD: Interrupting the sympathetic outflow in causalgia and reflex sympathetic dystrophy. A futile procedure for many patients. *BMJ*. 1998; 316: 792-3.
49. Schwartzman RJ, Liu JE, Smullens SN, Hyslop T, Tahmouh AJ: Long-term outcome following sympathectomy for complex regional pain syndrome type I (RSD). *Journal of Neurological Sciences*. 1997; 150: 149-52.
50. Poplawski ZJ, Wiley AM, Murray JF: Post traumatic dystrophy of the extremities. *J Bone Joint Surg [Am]* 1983; 65:642-55.