SPINE AND THE AUTONOMIC NERVOUS SYSTEM

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Abstract. In acute noxious injuries to the spine, usually the somatic sensory and motor dysfunctions originate an acute pain which is referred to as acute "nociceptive" or somatic pain (Figure 1). In occasional situations the pain may have a tendency to persist for months or several years after the original injury has healed. This type of persistent pain has been referred to as "neuropathic pain."

Descriptors. autonomic nervous system, complex regional pain syndrome(CRPS), neuropathic pain, parasympathetic nervous system, somatic nervous system, sympathetic nervous system

INTRODUCTION

The autonomic and somatic nervous systems are the integral anatomical and physiological components of the spine. The noxious (painful)stimuli are divided into somesthetic or somatic pain (no neurovascular component) and "neuropathic" pain (associated with neurovascular changes) due to the fact that the majority of the central and peripheral nervous system dysfunctions are somatic in nature, the role of autonomic nervous system in neuropathic type of pain has been over-shadowed and ignored (Table I).

The chronic, neuropathic pain-e.g., nerve root contusion, neuralgia, stump neuroma pain, and sympathetically maintained pain (SMP)- becomes persistent and has a tendency to affect the function of the sympathetic nervous system (SNS). The neuropathic pain has a tendency to cause referred pain, spasm, chronic tissue damage, and inflammation. It may be accompanied by vascular and trophic changes. These chronic autonomic dysfunctions and their relation to the spine will be discussed in this article.

THE AUTONOMIC NERVOUS SYSTEM

The autonomic nervous system (sympathetic and parasympathetic nervous system) is mainly responsible for:

(i). Control of the vital signs (blood pressure, pulse, and respiration)(1,2).

(ii). Control of body temperature by achieving a balanced and stable homeostasis of the internal environment ("Milieu interne")(1,2). This is done through sophisticated modulation of the body temperature, cell membrane exchange, and rate of metabolism.

(iii). Control of the immune system: Any internal or external distress may stimulate the sympathetic system to up-regulate the immune system and T-cell lymphocyte function. This process leads to "neuroinflammation"(2-8).

There are two major autonomic spinal structures: *(i)*. thoracolumbar autonomic system, and *(ii)*. craniocervical autonomic system.

These two divisions are under reciprocal and putative influence of the higher centers of the brain and brain stem on one extreme, and the peripheral neurovascular structure on the other. The neuronal structures in the autonomic nervous system are divided into supraspinal, preganglionic, and postganglionic neurons. The pre- and postganglionic fibers are mainly poorly myelinated small c fibres. The chain of ganglia transmits the impulses vertically and horizontally, contributing to spread of pain, spasm, neuroinflammation, and at times spread of the full-blown disease such as complex regional pain syndrome (CRPS) (1,2,7,9,10) (Figure. 2).

By virtue of the fact that the sympathetic ganglia have a close affinity to the spine, the pathologic spinal or autonomic changes are apt to affect the function of these two different structures (Figure. 2).
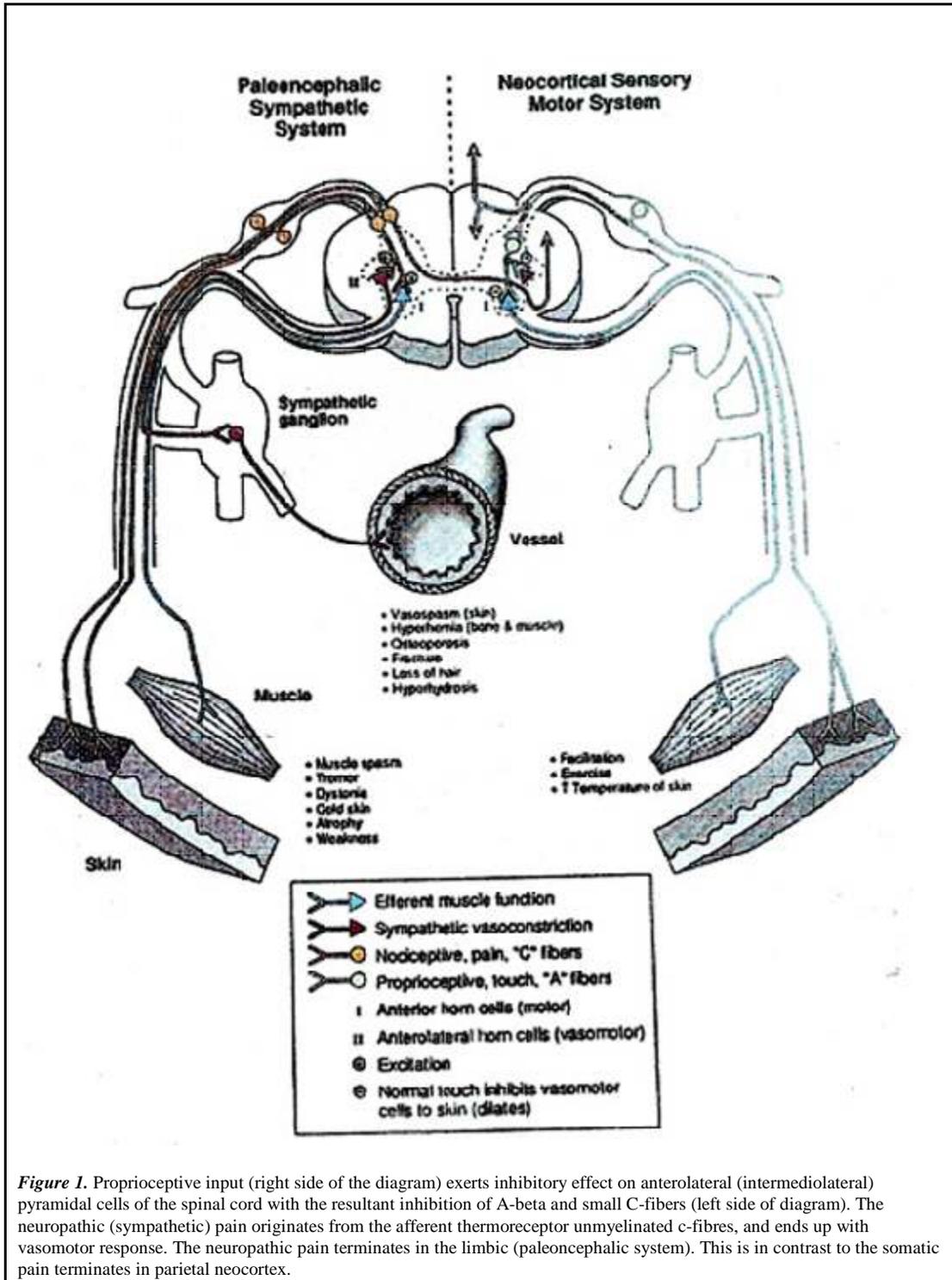

Paleoencephalic Sympathetic System

Neocortical Sensory Motor System

Sympathetic ganglion

Vessel

- Vasospasm (skin)
- Hyperhemia (bone & muscle)
- Osteoporosis
- Faemus
- Loss of hair
- Hyporhydrosis

Muscle

- Muscle spasm
- Tremor
- Dystonia
- Cold skin
- Atrophy
- Weakness

- Facilitation
- Exercise
- ↑ Temperature of skin

Skin

- ⊃—▷ Efferent muscle function
- ⊃—▶ Sympathetic vasoconstriction
- ⊃—◉ Nociceptive, pain, "C" fibers
- ⊃—○ Proprioceptive, touch, "A" fibers
- I Anterior horn cells (motor)
- II Anterolateral horn cells (vasomotor)
- ⊕ Excitation
- ⊖ Normal touch inhibits vasomotor cells to skin (dilates)

Figure 1. Proprioceptive input (right side of the diagram) exerts inhibitory effect on anterolateral (intermediolateral) pyramidal cells of the spinal cord with the resultant inhibition of A-beta and small C-fibers (left side of diagram). The neuropathic (sympathetic) pain originates from the afferent thermoreceptor unmyelinated c-fibres, and ends up with vasomotor response. The neuropathic pain terminates in the limbic (paleoncephalic system). This is in contrast to the somatic pain terminates in parietal neocortex.
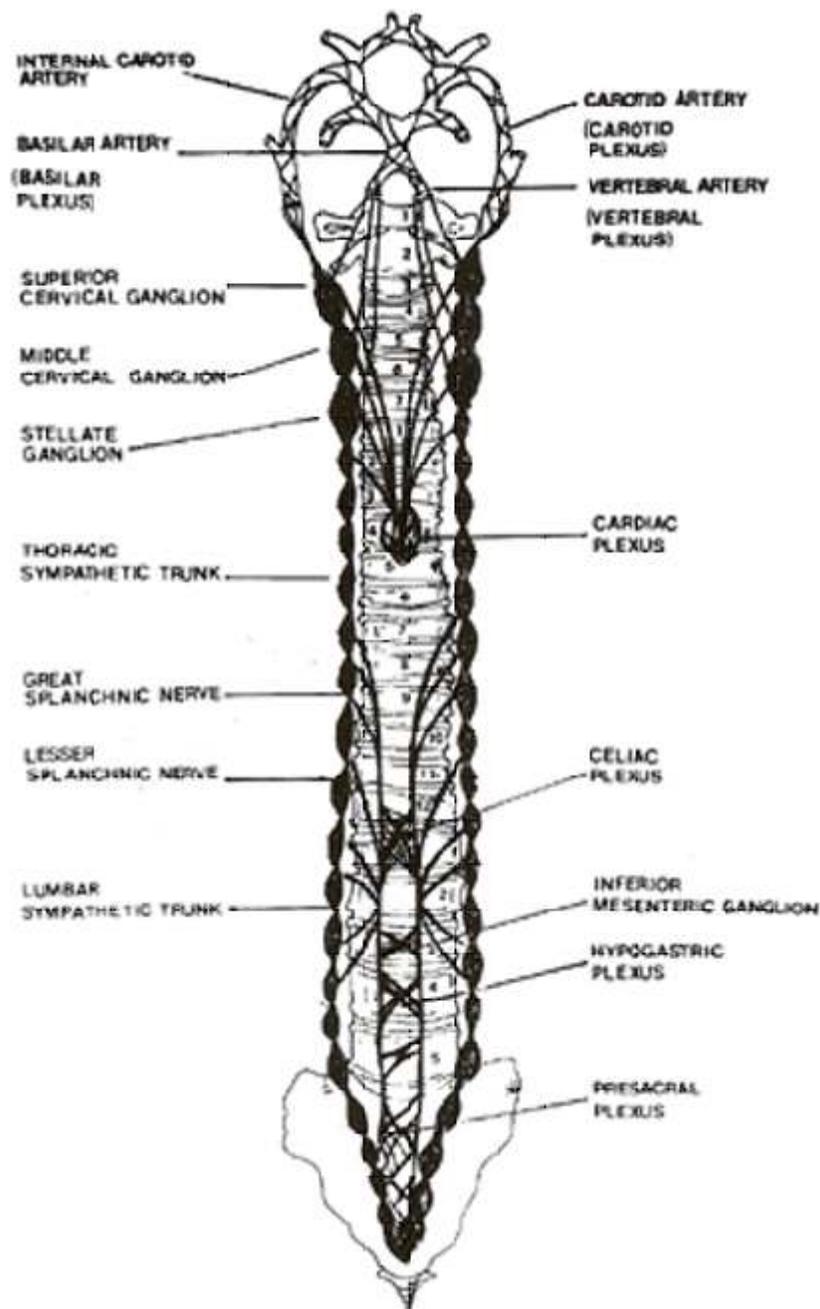
Figure 2. Vertical and midline connections and plexi of sympathetic nervous system. This complex intermingling of SNS fibers renders unilateral sympathectomy ineffective in the long run. The affinity of the sympathetic nervous system to the spine explains the involvement of the SNS in spinal injuries in the form of "neuropathetic pain." "SMP" and in other manifestations such as vascular headaches, vertebral artery insufficiency (dizziness, tinnitus, ataxia, and poor memory, and extremity or visceral inflammation.

The supraspinal efferent nerves influencing the sympathetic system are mainly beta and alpha 2 receptors. They influence the sympathetic preganglionic cells originating alpha T efferent norepinephrine impulses in the brain stem and spinal cord levels. These preganglionic fibers traverse through the cranial nerves and the ventral root of the spinal nerve and synapse at the sympathetic ganglion. These preganglionic fibers synapse with the postganglionic nerves on a 1/20 ratio. The postganglionic sympathetic nerve fibers(alpha 1) end up in the target tissues such as arterioles with resultant peripheral vascular changes (Table I). On the other hand, the parasympathetic preganglionic nerves originate mainly from the brain stem and sacral portion of the spinal cord. The brain stem (cranial) portion of the parasympathetic nerve traverse through the 3rd, 7th, 9th, and 10th cranial nerves with 3/4 of the cranial parasympathetic nerves being represented in the 10th (vagus) nerve. Less than 10% of the sympathetic post ganglionic nerve fibers are cholinergic responsible for sweating, and for vasodilation in the face and neck. Even through the sudomotor (sweating) parasympathetic cholinergic fibres are incorporated in the trunk of sympathetic nerve fibres, they are not truly representative of sympathetic function which is represented by more than 90% norepinephrinergic fibres.

Table I. Commonly used sympathetic blockers

α- Blockers	β- Blockers	Calcium influx blockers[a]	Presynaptic (brain stem) blockers [a]
α_1-Blocker: Phenoxybenzamine (Dibenzyline)[a]	Timolol (Blockadrene)	Nifedipine (Procardia)	Clonidine-hydrochloride[b] (Catapres)
Terazocin [a] (Hytrin)	Propranolol [b] (Inderal)	Diltiazem (Cardizem)	Propranolol [b] (Inderal)
Prozacin [a] (Minipres)	Atenolol (Tenormin)	Verapamil (Calan)	—
α_2-Blocker: Yohimbine [c] Idazoxan (11)	—	Nicardepene (Cardene)	—

a. First choice should be phenoxybenzamine, Terazocin, or Prozacin. If they do not work, the others may be tried. This is an exclusive α_1-adrenergic receptors' supersensitivity (12,13).

b. The disruption of efferent sympathetic modulation results in supersensitivity of sensory end organs to norepinephrin (12). Excessive efferent sympathetic modulation (CRPS) activates the α_1 sensory receptors. So, α_1- blockers are quite effective in control of CRPS. The β- blockers and presynaptic blockers are not helpful.

c. Yohimbine 5.4 mg five times a day is well tolerated and a good adjunct in treatment of CRPS (14-20). It is a presynaptic α_2-blocker.

SYMPATHETIC SYSTEM AND SPINAL INJURY

There has been an excessive emphasis in the role of intervertebral disc protrusion as the cause of chronic neck and back pain. In our experience and as well as the experience of Doctor Rosomoff the intervertebral disc protrusion is responsible for less than 1/3 of chronic neck and back pain (1,21) (Table II). Nerve root contusion, spondylosis, spinal instability, and neuropathic referred pain are some of the common causes of back pain.

Table II. Causes of neck and back pain in 482 patients with detailed studies [a]

Cause of Pain	Number	Percent
Sympathetic mediated pain (SMP)	101[b]	21.0 [c]
Disc protrusion	135	28.0
Nerve root contusion	94	22.0
Conversion reaction	66	15.4
Myofascial injuries	42	9.8
Electrical injuries	32	7.5
Arachnoiditis	12	2.8
Referred pain	6 [d]	1.4
Lumbar stenosis	16	3.7
Spinal cord injury	4	1.0
—	**Total 508 [e]**	**119.8% [e]**

a. EMG, SSEP, Thermography as well as CT or MRI.

b. Sympathetic dystrophy may have been alone or accompanied with other causes of the patient's pain. This accounts for the value of 508 and 119.8%.

c. The SMP was diagnosed with the help of nerve blocks and Thermography or bone scan.

d. Cancer, aortic aneurysm, pelvic injury.

e. Multiple pathologies in a patient caused the total number and total percentages to exceed the actual number of patients.

The craniocervical and thoracolumbar divisions of the sympathetic nervous system involvement in the spinal injuries result in referred pain, and other symptoms mistaken for different illnesses (Table III).

Table III. Symptoms of cervical spondylosis

Symptoms of Spondylosis	Disease Mistaken For
Vertigo secondary to pressure on vertebral artery	Meniere's disease Inner ear disease
CRPS with spasm around the shoulder girdle and secondary bursitis ligament injury	Bursitis Rotator cuff injury Shoulder ligament injury Shoulder-hand syndrome
Pain and CRPS down the hands and elbow	Carpal tunnel syndrome Tardy ulnar palsy and unnecessary surgery
Referred pain to pectoralis trapezius scalene areas	Thoracic outlet syndrome (TOS): thoracic outlet syndrome rarely is symptomatic. Even then, it is usually caused by scalenus anticus spasm due to CRPS and chronic pain
Tremor, dystonia	Familial essential tremor Dystonia
Hemifacial spasm and blepharospasm as referred spasm (rare)	Essential blepharospasm improperly treated with surgery or botulinum toxin injection
Vascular headaches	Migraine
Occipital neuralgia	Idiopathic occipital neuralgia Occipital nerve section has disastrous results
Ophthalmic nerve referred pain to forehead	Trigeminal neuralgia treated with unnecessary surgery
Vertebral basilar artery insufficiency (VBI)	Stroke Bell's palsy Meniere's disease Inner ear disease Idiopathic ataxia, etc.
Chest pain	Coronary artery disease Angina pectoris

Management of Cervical Spondylosis*
Traction both at home and at physical department. The traction should be done on a daily basis at home as well as 2-3 times a week at physical therapy.
Moist heat, muscle relaxants(e.g., Baclofen, Skelaxin), message, ultrasound, and trigger point injections.
*The same treatments and principles apply to chronic cervical spine injury.

Because of the fact that the spinal origination of the neuropathetic pain invariably results in referred symptoms in remote structures, the spinal pathology goes undiagnosed and is mistaken for conditions such as "thoracic outlet syndrome," "TMJ disease," "Meniere's Disease," "Cluster headaches," "inner ear disease," "migraine headaches," "carpal tunnel syndrome," "tarsal tunnel syndrome" and a series of other vague terminologies referring to the end organ symptoms(1,22-24)(Table IV and V).

TABLE IV. CRPS (RSD) MISTAKEN FOR OTHER DISEASES

1. TMJ disease is commonly a manifestation of CRPS (RSD), and infrequently due to temporomandibular joint pathology. TMJ dysfunction can cause CRPS and vice versa.
2. Thoracic outlet syndrome (TOS) is rarely an entity in itself. More commonly what is called TOS is a manifestation of referred pain from cervical spine pathology, or spasm due to CRPS. The same pathology causes pain as well as spasm in the muscles of the thoracic outlet triangle. The muscle spasm produces a classic TOS. TOS surgery obviously will not correct the cervical spine pathology, or CRPS.
3. Carpal tunnel syndrome* and tardy ulnar palsy. Long before CRPS is diagnosed, the patient may undergo multiple operations for misdiagnosed entrapment neuropathies.
4. Rotator cuff injury.
5. Iliotibial band injury.
6. "Myofascial syndrome" is a wastebasket of so-called "soft tissue injuries." It is more likely due to referred pain secondary to nerve injury.
7. "Münchausen's syndrome.

Table V. Carpal tunnel syndrome - somatic vs. sympathetic.

In occasional patients with Carpal tunnel syndrome (CTS), the condition is the result of edema of CRPS (RSD). Surgery in such patients leads to disastrous results. Careful examination can differentiate sympathetic versus somatic types of CRPS.
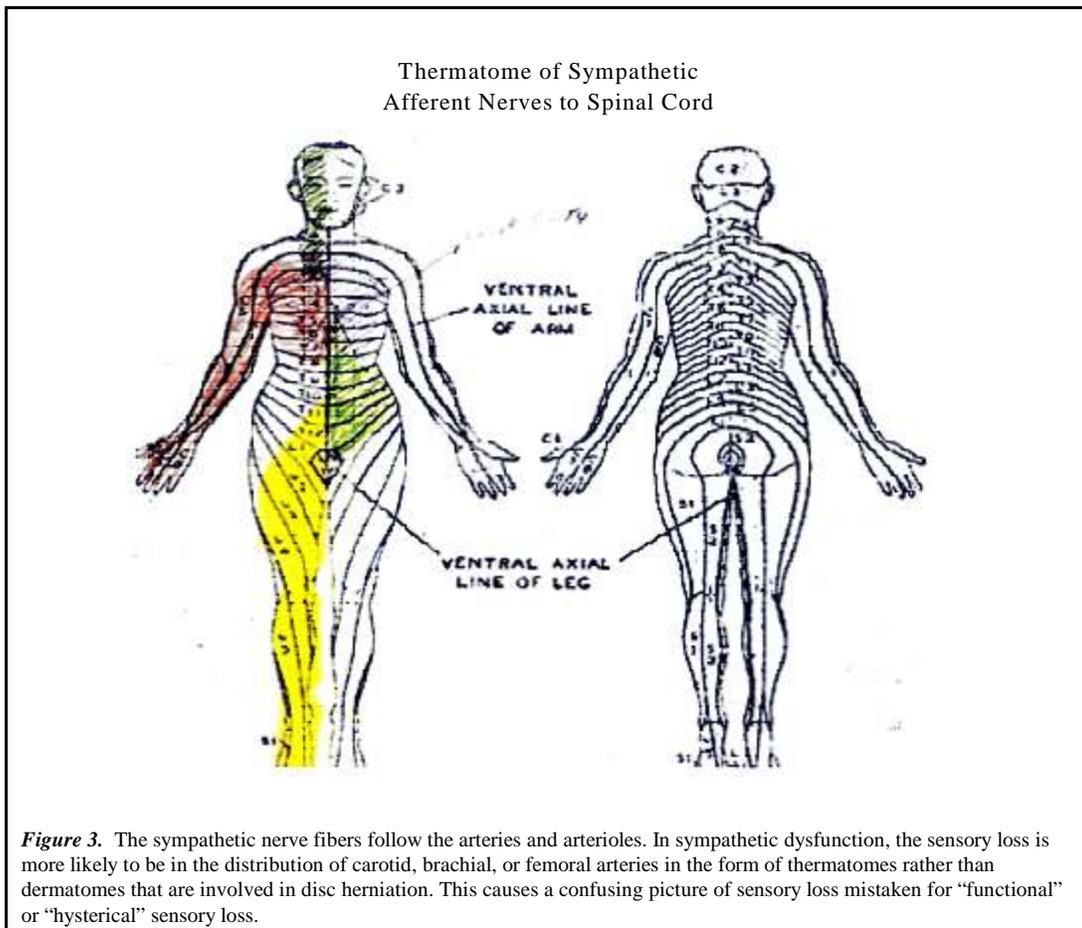
The neuroinflammation due to CRPS may lead to entrapment neuropathies. Surgery may aggravate the CRPS. Somatic entrapment is usually helped with surgery.

	Carpal Tunnel Syndrome (Somatic)	CRPS (Sympathetic)
Pain	Proportionate to impingement	Allodynic and hyperpathic pain- worst in late stages
Trophic changes	Thenar atrophy in late stages	Edema: hair and skin changes; generalized interossei atrophy in late stages
Tinel's sign	Limited to carpal tunnel	Indiscriminative: Entire wrist Hyperpathic
Muscle tonus	Weak, atrophic in advanced cases	Dystonic flexor spasm and deformity of digits
NCV	Delayed sensory and motor distal latency	Normal or borderline sensory delay
Infrared Thermal Imaging (ITI) Changes	Usually no change. In advanced stages with over 6 millisecond distal delay, mild hyperthermia in the first three fingers distribution pointing to vasa neuronum damage**.**	ITI changes are in thermatomal distribution involving the entire hand and wrist, but not limited to median nerve distribution in the first three fingers.
Neurovascular instability: Mottling, color changes: (Chameleon sign)	None	Present off and on
MRI findings	No inflammation	Osteopenia, bone necrosis, and extravasation between bone spaces.
Surgical findings	No inflammation	Moderate edema; osteonecrosis, fragile bones at the wrist.

CRANIOCERVICAL SYMPATHETIC DYSFUNCTION IN SPINAL INJURIES

Cervical spine pathology, usually cervical sprain or cervical spondylosis, is quite frequently accompanied by sympathetic dysfunction resulting in a series of symptoms (Table III). As a result, the patient is exposed to ineffective and at times dangerous treatment such as occipital neurectomy or cryosurgery for treatment of occipital neuralgia, traumatic manipulation of the spine with resultant stroke, and unnecessary surgery and fusion. The spondylosis in and itself is a natural development of osteophytes to stabilize the spine and to minimize damage to the nerve roots. The surgical removal of the osteophytes is throwing off the natural balance and cannot be expected to provide and long-standing relief. Simple physical therapy in the form of traction, moist heat, along with treatment with muscle relaxants and nerve blocks are treatment of choice.

As is the case with the rest if the sympathetic system, the craniocervical sympathetic system follows the craniocervical arteries and arterioles closely to gauge the circulatory and temperature changes (Figure.3). The sympathetic nerves follow a thermatomal pattern-in contrast with somatic radicular dermatomal pattern (7,9,25-27)(Figure.3). This results in erroneous diagnosis of "functional" or "malingering" sensory loss.

Thermatome of Sympathetic
Afferent Nerves to Spinal Cord

Figure 3. The sympathetic nerve fibers follow the arteries and arterioles. In sympathetic dysfunction, the sensory loss is more likely to be in the distribution of carotid, brachial, or femoral arteries in the form of thermatomes rather than dermatomes that are involved in disc herniation. This causes a confusing picture of sensory loss mistaken for "functional" or "hysterical" sensory loss.

The Sherrington-Kerr phenomenon points to the overlapping afferent sensory pools in the cervical spine region(1,28,29) (Figure. 4). The substantia gelatinosa of the upper cervical C1 through C4 nerve root region overlaps the nuclei of the ophthalmic branch of the trigeminal nerve.
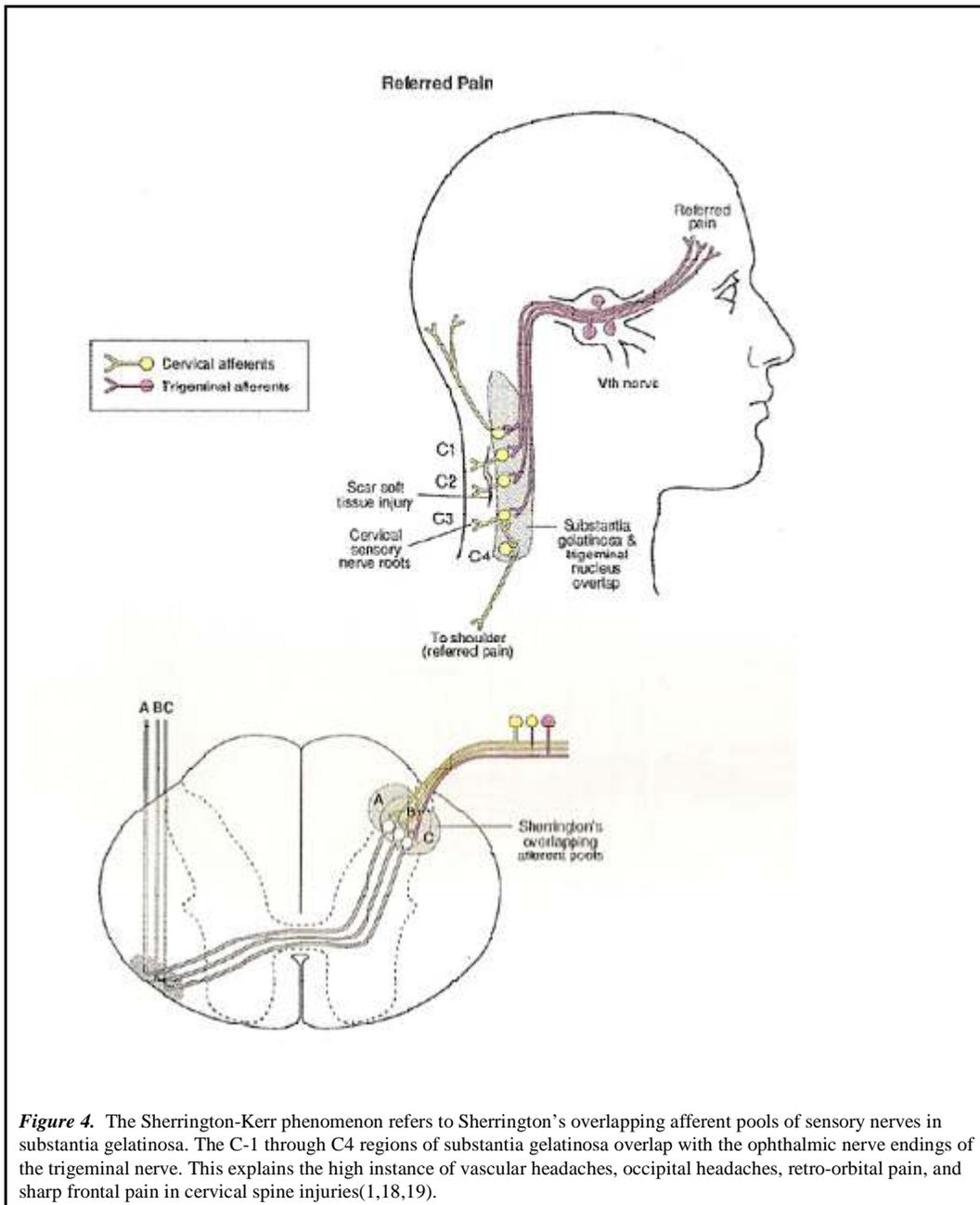
Figure 4. The Sherrington-Kerr phenomenon refers to Sherrington's overlapping afferent pools of sensory nerves in substantia gelatinosa. The C-1 through C4 regions of substantia gelatinosa overlap with the ophthalmic nerve endings of the trigeminal nerve. This explains the high instance of vascular headaches, occipital headaches, retro-orbital pain, and sharp frontal pain in cervical spine injuries(1,18,19).

Injury to the upper cervical sensory nerve roots results in overlapping afferent pool of Sherrington and referred pain to the retro-orbital region, frontal region, as well as to the shoulder and the occipital region(1,28). The end result is frontal, occipital, and retro-orbital vascular headaches (1,30) (Figure. 5). Treatment with traction, moist heat, as well as occipital and paravertebral blocks in most effective (1,30).
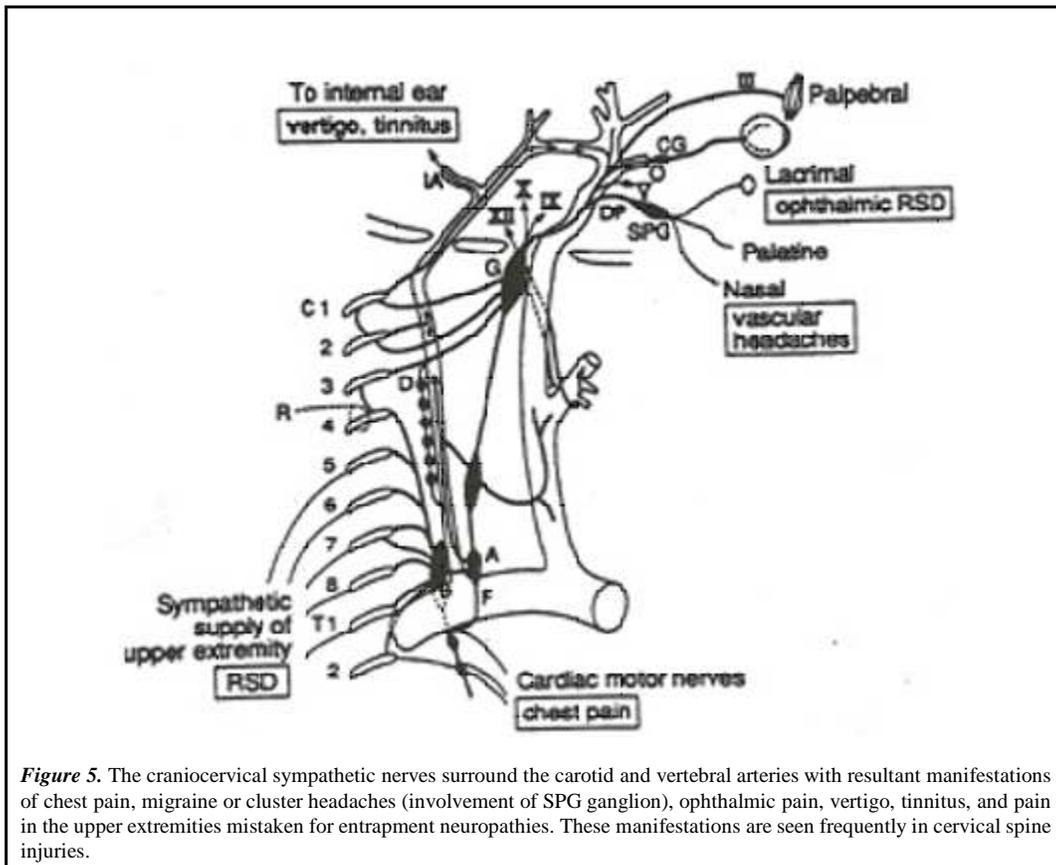
Figure 5. The craniocervical sympathetic nerves surround the carotid and vertebral arteries with resultant manifestations of chest pain, migraine or cluster headaches (involvement of SPG ganglion), ophthalmic pain, vertigo, tinnitus, and pain in the upper extremities mistaken for entrapment neuropathies. These manifestations are seen frequently in cervical spine injuries.

COMPLEX REGIONAL PAIN SYNDROME (CRPS, RSD)
AND CRANIOCERVICAL DYSFUNCTION

Complex regional pain syndrome is a syndrome that may develop as a consequence of any noxious stimulus (trauma, etc.) chronically affecting the sympathetic system in the limbs, craniocervical region, thoracolumbar region, or visceral organs (stroke, heart attack, or cancer). The original injury is usually minor in nature. The lesion may be obvious on the surface or concealed. The syndrome is characterized by:

(i). Allodynic pain (pain induced by a non-noxious stimulus), hyperpathic pain (over-reaction with pain to stimulus which may persist after the stimulation has ceased). The pain is usually practically constant, burning, and / or stabbing in nature. In early stages of the disease the pain is abated with sympathetic nerve blocks (such as I.V. Phentolamine, and Bier block). In late stages of the disease the sympathetic block may not effectively or completely control the pain (the pain is no longer sympathetically maintained pain-or SMP) (1).

(ii). Efferent vascular, motor, and sudomotor response to the pain. This is in the form of vasoconstriction with cold extremity in the area surrounding the lesion, muscle spasms, tremor, dystonia, and abnormal sweating (31-35).

(iii). Edema, inflammation, and dystrophic changes due to the disturbance of sympathetic function which results in the extravasation of fluids, neurodermatitis, cell membrane and immune system disturbance with secondary inflammation(2,6,8,34,36-39).

(iv). Due to the fact that the sympathetic afferent input chronically stimulates the limbic(frontal temporal) system, the patient suffers from insomnia, agitation, depression, poor memory, and poor judgment (1,2)(Figure. 6).
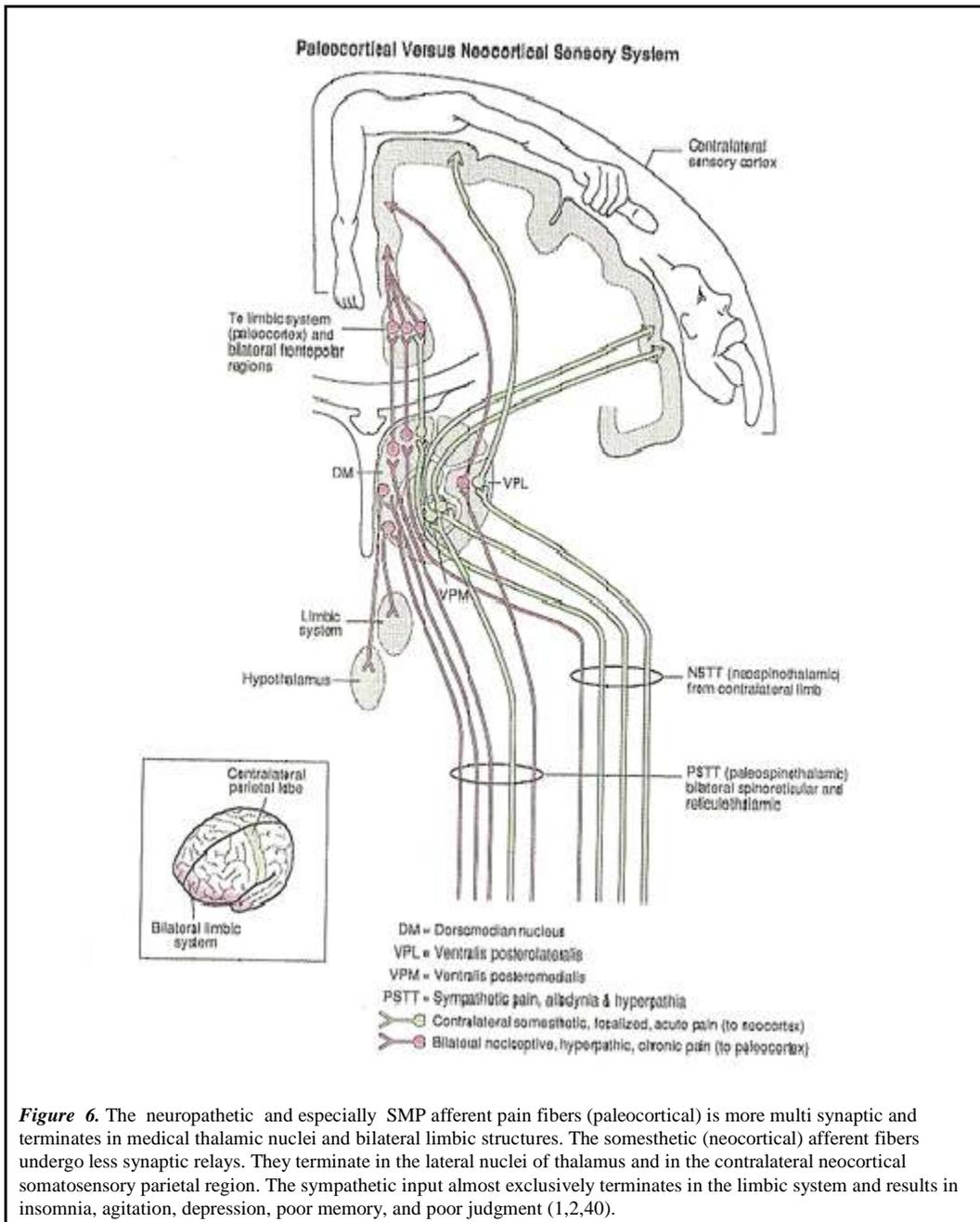
Paleocortical Versus Neocortical Sensory System

Figure 6. The neuropathetic and especially SMP afferent pain fibers (paleocortical) is more multi synaptic and terminates in medical thalamic nuclei and bilateral limbic structures. The somesthetic (neocortical) afferent fibers undergo less synaptic relays. They terminate in the lateral nuclei of thalamus and in the contralateral neocortical somatosensory parietal region. The sympathetic input almost exclusively terminates in the limbic system and results in insomnia, agitation, depression, poor memory, and poor judgment (1,2,40).

In both craniocervical and thoracolumbar CRPS, efferent motor dysfunction quite frequently results in an accordion effect on the spine with secondary disc bulging or even disc herniation. This is an effect rather than a cause.

The treatment should be aimed at treating CRPS rather than surgery for this side effect of the disease.

The same efferent reflex manifestation of CRPS quite frequently results in cervicogenic tremor, dystonia, and even Parkinsonian type of tremor (31,34). The movement disorder responds best to muscle relaxants such as Baclofen, or Skelaxin, and simultaneous treatment with nerve blocks, physical therapy, and ACTH (1,31). The movement disorder is due to the sympathetic dysfunction, and to quote Jankovic, "the negative or positive outcome of litigation rarely influences the course of the movement disorder" (31).

The inflammation of CRPS results in entrapment neuropathies such as carpal tunnel and tarsal tunnel syndrome. Surgical treatment for such entrapment neuropathies invariably fails to relieve the condition, and only accelerates and aggravates the deterioration of CRPS(1,2). Such entrapment neuropathies can be easily differentiated clinically by a careful neurologic examination (Table IV). The main feature that differentiate the conditions are the Tinnel's Sign not being focalized to the area of nerve entrapment, allodynia being present over the entire distal portion of the extremity, and edema especially noted on MRI of the wrists or ankles showing joint extravasation as well as osteopenia in the small bones of the involved area (Table IV).

Treatment of choice for such entrapment neuropathies are nerve blocks, muscle relaxants, epsom salt and warm water, and physical therapy along with the use of newer antidepressants (as the analgesic of choice)(2).
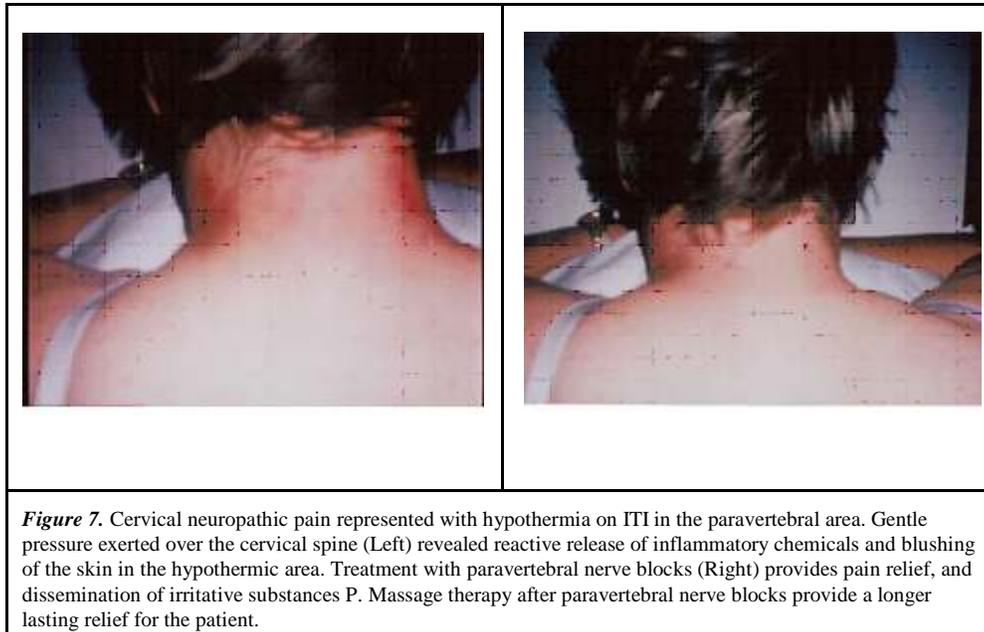
Surgical treatment, the application of ice or assistive devices such as braces or cast can aggravate the condition. The application of a cast is a high risk for the patient to develop cervicogenic Parkinsonian tremor (1,2,21,41-45).

THORACOLUMBAR SYMPATHETIC DIVISION

The diagnostic and therapeutic difficulties in the thoracolumbar division are identical to the craniocervical division. The disc bulging and herniation due to a paravertebral spasm secondary to CRPS are more likely to confuse the diagnosis in the neuropathic pain and the spasm in the thoracolumbar region. Especially women who have had natural child birth, the L-5 / S-1 disc bulging is an incidental painless byproduct of the natural delivery. The neuropathic referred pain from the lower extremity to the lumbar spine is mistaken for the problem of disc bulging and the patient undergoes unnecessary lumbar surgery with disastrous results.

CRPS is not simply a reflection of a hyperactive sympathetic system, but a pathologic sympathetic system. Any operation in the distribution of the nerves involved with CRPS results in a flare-up of the disease and aggravation and spread of the CRPS(1,2,7,9). The surgical procedures for disc bulging or herniation, and for relief of entrapment neuropathy, in CRPS patients can push the patient who is in stage I or II into stage III or IV of CRPS (stage II is the dystrophic stage, and stage III is the atrophic stage and stage IV is addition of complications such as stroke, heart attack, hypertension, suicidal attempts, and immune system dysfunction)(1,2).

The dorsum of the hand, foot, knee, and elbow are the common source of post traumatic CRPS. Secondarily, the disease causes referred pain, spasm, and inflammation spreading to the paraspinal structures(2)(Figure. 7). The involvement of the paraspinal nerve and muscle results in muscle spasm, headache, back pain, dizziness, and movement disorder(1,31,46).

Figure 7. Cervical neuropathic pain represented with hypothermia on ITI in the paravertebral area. Gentle pressure exerted over the cervical spine (Left) revealed reactive release of inflammatory chemicals and blushing of the skin in the hypothermic area. Treatment with paravertebral nerve blocks (Right) provides pain relief, and dissemination of irritative substances P. Massage therapy after paravertebral nerve blocks provide a longer lasting relief for the patient.

CRPS commonly involve the extremities. However, the extremity involvement of CRPS causes referred pain, spasm, and inflammatory changes in the paravertebral nerves and muscles (Figure. 7). This has been mistaken for entrapment neuropathy, or "myofascial syndrome"(1,2,22,23,24,47,48)(Table IV). The pain in myofascial syndrome is truly a neuropathetic and sympathetically driven pain with a tendency for referred pain and inflammation in the trunk of the afferent nerves from the extremity to the paraspinal nerves and vice versa (48). Clinically, the areas of referred pain, nerve and trigger point irritation can be easily identified by careful examination of the paravertebral muscle and joints. In SMP type of pain, pressure in these areas results in a reddish discoloration (Figure. 7).

As the area of nerve and trigger point irritation is identified, this area should be treated with a paravertebral nerve block with 4-5 cc Lidocaine and 40-80mg Triamcinolone or Depo Medrol® (1,30). This type of nerve block results in pain relief and improved circulation to the extremity lasting from a few weeks up to two months. In more acute patients, one set of treatment is usually enough to provide a successful nerve block and permanent pain relief.

The axial sympathetic dysfunction is far less common. In abdominal trauma and especially in post traumatic sympathetic dysfunction secondary to repeated abdominal operations, the patient develops classical neuropathetic pain which can be successfully blocked by coeliac ganglion block, combined paravertebral nerve blocks.

Approximately 1/3 of the patients with failed back syndrome suffer from neuropathetic pain in both referred to the extremity as well as in the paraspinal area. This is especially true if the patient has undergone the so-called exploration for conditions such as disc or nerve root contusion. In some patients a foraminotomy is done to release the pressure on the nerve root. These patients quite frequently suffer from severe neuropathetic pain which has a strong sympathetic component to it (SMP). In such patients, the paravertebral nerve blocks as outline above are quite helpful in alleviating the pain and counteracting the muscle spasm (which works as a vicious circle in aggravating the pain).

Axial sympathetic dysfunction in the thoracic area is quite rare. The usual forms are the intercostal diabetic neuropathy and malignancy involving the intercostal nerve roots. The patient does not develop the full-blown picture of CRPS. In such patients, systemic treatment with anti-viral medications (Zovirax) and sodium channel blockers (Carbamazepine, Phenytoin) or treatment with Neurontin may be helpful. Addition of newer antidepressants (Fluoxetin, or Trazodone) as analgesic of choice in the long run render the best relief. Tricyclics such as Elavil or Tofranil should be avoided due to their side effects of weight gain and aggravation of fatigue.

TREATMENT

Multi-disciplinary treatment is the key to success:

(i). Avoidance of ice application which aggravates the vasoconstriction and accelerates the sympathetic nerve damage. Moist heat and epsom salt are the treatments of choice (2,41-45).

(ii). Avoidance of braces, immobilization, application of casts, Jankovic has noted that 8 of 9 of the cervicogenic Parkinsonian tremors among the patients that were treated with application of cast (31). The reason for avoidance of immobilization is the fact that immobilization deprives the spinal cord from the inhibitory effect of somatic proprioception on the intermediolateral horn cells with resultant aggravation of sympathetic dysfunction(1).

(iii). Nerve blocks: epidural, paravertebral, ganglionic, regional, and systemic such as the use of Clonidine patch or the use of oral alpha blockers (Dibenzyline, Hytrin, Clonidine, or Yohimbine)(1) (Table I).

(iv). Avoidance of surgery, especially exploration of the area of nerve root contusion, exploration of the area of arachnoiditis, or exploration of entrapment neuropathies secondary to inflammation of CRPS (avoidance of surgical procedures for carpal tunnel syndrome, tarsal tunnel syndrome, or "thoracic outlet syndrome")(1).

(v). Discontinuation of opioid agonists and endo-benzodiazepine (Endo BZ) agonists, replacement with opioid antagonists such as Buprenex, Nubain, and Ultram. The use of Elavil causes fatigue and weight gain. Treatments with newer antidepressants such as Trazodone or Desipramine are the treatment of choice for chronic pain.

SUMMARY AND CONCLUSION

The autonomic nervous system concerns itself with preservation and protection of the "Internal Environment." For example, in warm blooded animals the autonomic nervous system keeps the temperature inside the body around 99° Fahrenheit (37 °C).

To protect the internal environment, the autonomic nervous system has two main components: *(i).* The sympathetic system, and *(ii).* The parasympathetic system.

CRPS is a form of disturbance of the function of the autonomic nervous system. Simply having a hyperactive sympathetic nervous system does not make CRPS. The disturbance of the autonomic system comprises several diseases, some acquired, some genetic, some metabolic, some traumatic, etc.

Some examples of dysautonomias "disturbances of the autonomic nervous system" are attacks of hypotension (low blood pressure), congenital absence of sweating, and neuropathic pain syndrome.

The latter category of chronic neuropathic pain syndrome refers to the conditions that are not exactly necessarily CRPS, but have an abnormal sympathetic component to them. They share sympathetically maintained pain (SMP) with CRPS but they are not CRPS.

Examples of such chronic neuropathic pain are postherpectic neuralgia (pain accompanying and following shingles), neuropathic diabetic neuropathy, acute neuropathic pain accompanying bee stings, snake bites or spider venom stings as well as involvement of the sympathetic nerves due to the systemic AIDS infection.

The neuropathic pains are not at all synonymous with CRPS. They do not even in any way resemble CRPS. In some cases, however, they can end up with CRPS.

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