JAOA Review article

# Osteopathic medical considerations of reflex sympathetic dystrophy

KENNETH E. NELSON, DO

**Review of current medical literature reveals little understanding of the physiology underlying the complex signs and symptoms that accompany reflex sympathetic dystrophy (RSD). The author surveyed the osteopathic medical literature and found a significant body of research documenting the physiology of somatic dysfunction. The manifestations of upper thoracic somatic dysfunction are strikingly similar to those of RSD and may offer insight into its heretofore unexplained physiology of this disorder.**

**(Key words: Reflex sympathetic dystrophy, somatic dysfunction, facilitation, osteopathic manipulative treatment)**

Reflex sympathetic dystrophy (RSD) is a chronic pain condition that affects the limbs. It is characterized by diffuse pain, altered skin color, altered skin temperature, edema, and motion restriction.[1] This condition has been repeatedly reported since the Civil War, when the term *causalgia* was first used to describe the persistent, excessive, posttraumatic appendicular pain; however, the basic physiology of RSD has not been clearly identified.[2]

The similarity is striking between the signs and symptoms of RSD and those described in association with upper thoracic somatic dysfunction. Furthermore, the responsiveness of these signs and symptoms to osteopathic manipulative treatment (OMT)[3-6] suggests that considering RSD in terms of our current understanding of somatic dysfunction might prove beneficial. Reflex sympathetic dystrophy may be merely the peripheral symptoms of untreated upper thoracic somatic dysfunction, or it may represent an extreme presentation of neurophysiology shared with somatic dysfunction. In either case, OMT appears to offer the potential for specific therapeutic intervention in the treatment of RSD.

Dr Nelson is the director, Division of Osteopathic Manipulative Medicine, Chicago College of Osteopathic Medicine, Downers Grove.

Correspondence to Kenneth E. Nelson, DO, FAAO, Director, Division of Osteopathic Manipulative Medicine, Chicago College of Osteopathic Medicine, 555 31st St, Downers Grove, IL 60515.

## Characteristics of RSD

Reflex sympathetic dystrophy consists of a group of signs and symptoms that typically refer to one limb, appear to be mediated by the sympathetic nervous system, and usually have their onset following trauma. This complex may also develop following visceral diseases; it may accompany lesions of the central nervous system, or, less frequently, may develop without any specific antecedent event.[2]

A perplexing condition, RSD persists, often for years, after the precipitating event has healed. The course of RSD begins with a relatively minor injury that resolves normally. The associated pain, however, persists and spreads, affecting the entire limb. Resultant hyperalgesia, diffuse atrophy, and eventually, osteoporosis develop. The affected limb has decreased cutaneous temperature ($\geq 2^{\circ}$ C), as demonstrated thermographically.[7]

In a prospective study of 829 patients with RSD, Veldman and colleagues[1] reported the following findings (expressed in percent) among their patients: appendicular pain (93%); neurologic symptoms—including hypoesthesia (69%) and exaggerated response to painful stimuli (75%); and weakness resulting in impaired motion (95%). However, electromyographic studies, when performed, were normal.

Appendicular edema was present (69%) with a higher incidence in cases studied during the first 12 months of the condition. Temperature difference was detected in the skin of the affected limb when compared with the opposite extremity (92%). Furthermore, the longer the condition was present, the greater the incidence of decreased temperature on the involved side. Hyperhidrosis of the involved extremity was reported (57%). Also, 19 patients (2%) had recurrent unexplained hematomas localized to the affected limb.

Beyond these known characteristics, questions remain: How does a minor initiating insult result in such a debilitating condition? Why does the condition persist after the insult has resolved? The osteopathic medical literature offers some answers. A cause-and-effect relationship between upper thoracic somatic dysfunction and appendicular symptoms that resemble RSD was described by Larson[3] in 1970. Likewise, osteopathic medical research, some dating back more than 50 years, has employed the clinical signs and symptoms of RSD to identify somatic dysfunction.[5,6,8,9-11,24]

## Review of the osteopathic medical literature

Spinal somatic dysfunction has been identified by palpation and correlated with segmental irritability as confirmed by electromyography.[8] Somatic dysfunction is associated with a facilitated area in the spinal cord,[9] an area that responds with neurologic activity to less stimulus than adjacent unaffected areas. Patients with RSD also demonstrate an exaggerated response to painful stimuli. Somatic dysfunction manifests a segmentally related increase in sudomotor activity.[10] More than half of the patients in the study by Veldman and coworkers[1] exhibited hyperhidrosis. Spinal facilitation may have as its etiology foci of mechanical

irritation in the associated musculoskeletal system.[11] As RSD frequently develops following seemingly minor musculoskeletal injuries that resolve, could the segmentally related spinal facilitation maintain the RSD?

Pre-existing mechanical stresses within the musculoskeletal system—such as unilateral short lower extremity, errors in locomotion, congenital anomalies and somatic dysfunction in the pelvis, lumbar region or upper body—have been described by Schwab[12] in the 1930s as etiologies for musculoskeletal complaints. The mechanical pattern associated with postural imbalance exhibits certain areas of increased incidence of somatic dysfunction. Dysfunctional mechanics are more frequently found in areas where the anteroposterior curves are decreased and in transitional points in the mechanics of lateral curves.[13] Spinal facilitation may also result from segmentally related visceral disease or dysfunction.[14] Viscerosomatic reflexes for kidney,[15] lung,[16] duodenum,[17] and heart[18,19] have been specifically delineated.

## Relationship to segment facilitation

Whether somatic or visceral in origin, spinal segmental facilitation is the result of increased afferent activity emanating from a focus of irritation located outside the central nervous system. Local tissue inflammation liberates neurologically active substances that stimulate unmyelinated and lightly myelinated nociceptive neurons.[20] Impaired function in somatic structures increases activity of local mechanoreceptors.[21] These sensory neurons enter the dorsal horn of the spinal cord and synapse with internuncial neurons.[22] The sustained stimulation of these internuncial neurons that is thought to produce the facilitation of that particular level of the spinal cord.[23]

The location of facilitated segments in the spinal cord differs from person to person.[9] However, like RSD, facilitation consistently remains in the same locations in a given person for prolonged periods.[24] This consistency is understandable because postural mechanics are relatively stable, barring significant environmental effect on them, such as trauma, or the impact of repetitive activity patterns. The adult weight-bearing pattern is established as early as the end of the first decade of life.[25] Facilitation from viscero-somatic reflex manifestation should be present as long as the visceral disease or dysfunction is present. In chronic disease states, this could be many years.

In 1970, Larson[3] described what he termed the functional vasomotor hemiparesthesia syndrome, a condition that resembles RSD. In a later article,[4] he described a modified presentation of RSD with syndromes of the brachial plexus. He described the full syndrome in this manner:

> "Sensory change (develops) affecting one half of the body. The initial complaint involves the head, shoulder and upper extremity and a descending distribution to include the trunk and lower extremity. The sensory complaint may be pain, or anesthesia, or . . . paresthesia. . . .
>
> "If the disturbance is protracted there may develop secondary changes involving the circulation . . . . These changes may involve swelling, coldness, and extreme sensitivity to temperature and later even joint changes such as pain and swelling . . . .
>
> "The reaction seems to be related to a vasoconstriction causing an ischemia to the sensory nerves of the related side of the body."(pp 39,40,41)

Larson identified the etiology of this syndrome as "lesion pathology related to the upper thoracic spine and related rib."(p 39) It is particularly noteworthy that in the initial description of Larson's syndrome, he identified "in a few protracted problems" the development of "subcutaneous suggillation" (bruising) and "vasomotor reaction which caused an erythema" in the affected limb.

As noted earlier, Veldman and coinvestigators[1] reported spontaneous hematomas in 2% of his patients. Larson,[5] and later Kappler,[6] went on to demonstrate with thermography that patients with the syndrome, similar to patients with RSD, had decreased skin temperature in the affected limb(s). Both authors demonstrate the efficacy of specifically applied OMT for increasing peripheral cutaneous temperature and reducing the patient's complaints of appendicular pain. Thus, somatic dysfunction produces signs and symptoms very similar to RSD. The spinal facilitation of somatic dysfunction, like RSD, can persist for prolonged periods. Further, OMT is effective treatment for the signs and symptoms of somatic dysfunction that resemble RSD.

Larson's syndrome, peripheral signs and symptoms that bear a striking resemblance to RSD, is a manifestation of upper thoracic somatic dysfunction. It is frequently encountered as a partial presentation of the complete syndrome. It readily responds to specifically applied OMT.

## Treating Larson's syndrome

In my experience, the most common presentation of appendicular symptoms associated with spinal somatic dysfunction is unilateral cervicothoracic pain that radiates diffusely into the homolateral upper extremity. Typically, the appendicular complaint consists of diffuse pain, dysesthesia, paresthesia, hypesthesia, or anesthesia without any clearly demonstrable motor deficit. Although presentation of complete hemicorporic symptomatology is uncommon, an initial complaint of lower extremity pain is very common. I routinely inquire as to concomitant or antecedent problems involving the upper extremity or torso. Homolateral distribution of upper and lower extremity symptoms most always is associated with significant upper thoracic dysfunction. As described by Larson,[3] significant somatic dysfunction is demonstrated in the upper thoracic spine, usually between T2 and T5, although occasionally lower. Patients presenting with a chief complaint in the lower extremity usually manifest somatic dysfunction at the level of T5 or lower.

## Diagnosis

Diagnosis of the somatic dysfunction of Larson's syndrome is based on the identification of paravertebral tissue texture change, specific segmental asymmetry of position, and motion restriction. The typ-

ical paravertebral tenderness to palpation can be of significant diagnostic value in these patients. Palpating the area of most intense tissue texture change frequently reveals not only tenderness, but a trigger point reaction that results in pain radiating toward, and occasionally into, the painful extremity.

The importance of specific diagnosis of the mechanical pattern of the dysfunction cannot be overemphasized. If the dysfunctional area can be specifically identified, it can be precisely treated. Failure to identify specific dysfunctional motion restriction should lead the examining physician to seek a viscerosomatic etiology for the segmental facilitation.[26]

The patient's immediate response to precise manipulation limited to the primary dysfunctional vertebral unit is of great diagnostic value. Frequently, the patient reports immediate symptom reduction. This relief is commonly followed by an intensification of the original complaint. Such a rebound reaction should not last more than 24 hours. If it does, the intensity of the second OMT intervention should be appropriately reduced. As the rebound reaction subsides, a period of resolution typically follows in which the chief complaint is significantly reduced or absent. This period of resolution may last a few hours, or occasionally, it may last indefinitely. Ideally, the patient should be re-evaluated 48 hours after the treatment. By this time, the rebound reaction should have subsided, and residual somatic dysfunction can be specifically diagnosed and treated.

Symptom resolution should last progressively longer following each application of manipulative treatment, and treatment intervals should be adjusted accordingly. By the fifth therapeutic intervention, symptoms should resolve completely. Failure of this resolution period to increase indicates that an incomplete diagnosis has been made. Contributing causes must be thoroughly explored, identified, and treated. These may include mechanical stress from repeated minor activities, predisposition to this condition because of the patient's neutral weight-bearing pattern, or a viscerosomatic contribution.

## Discussion

Without doubt, RSD presents a perplexing clinical problem. However, when one considers RSD in terms of what is now known about somatic dysfunction, some possible explanations arise. Reflex sympathetic dystrophy and somatic dysfunction both demonstrate associated dysesthesia and altered sudomotor and vasomotor activity. Larson[3] describes appendicular distribution of signs and symptoms resulting from upper thoracic somatic dysfunction that are remarkably similar to those of RSD, including the spontaneous development of appendicular hematomas. The greatest density of sympathetic internuncial cell bodies is located in the intermediolateral cell column of the spinal cord between T1 and T4.[27] This area may act as an axial vasomotor center. The spinal facilitation associated with somatic dysfunction includes facilitation of the segmentally related sympathetic nervous system. This facilitation is thought to occur in the internuncial neurons.[23]

Somato-visceral reflexes significantly involve the autonomic nervous system. The same mechanism may exist for somato-somatic reflexes as well. Therefore, an injury to the upper extremity could, through "sympathetic afferent" activity, have a reflexive affect on the upper thoracic region from which sympathetic supply to the arm emanates. Under normal circumstances, one would expect to find a somato-somatic reflex that would diminish as the appendicular injury healed. However, the pre-existence of spinal segmental facilitation in the region could result in an exaggerated response. Symptom persistence following resolution of the peripheral focus of irritation may be explained by chronic pre-existent somatic dysfunction and sensitization of the spinal cord. Patterson[23 (p 11)] wrote, "Once a sensitized state is established in the spinal neural pathways either the continuation of the sensitizing input or the presence of normal input through sensitized interneurons would maintain the process, allowing the abnormal situation to continue."

Thus, in the presence of pre-existing spinal facilitation, an appendicular injury could trigger the development of signs and symptoms that might continue indefinitely.

The development of RSD-type symptoms following myocardial infarction has been documented for years.[28,29] Most often, the viscero-somatic reflex response from cardiac disease is located on the left side between T1 and T5.[14,18,19] This same area has been implicated as etiologic in Larson's syndrome. Apparently, visceral afferent activity following myocardial infarction, whether sensitizing or having a further impact on a previously sensitized level of the spinal cord between T1 and T5, could result in the development of post-myocardial infarction shoulder-hand syndrome.

Whether a patient will have signs and symptoms of RSD develop because of a given insult is probably best summarized by MacBain in his introduction to Fryette's [30] text, *Principles of Osteopathic Technique*: "Whenever the joints of the body wall are subjected to direct mechanical injury . . . there is some degree of inflammation set up . . . the degree of inflammation depends partly on the nature and severity of the injurious force and partly on the pre-existing state of the injured part. . . ."

## Conclusion

Specifically applied OMT reduces the appendicular symptoms associated with upper thoracic somatic dysfunction.[5,6] The similarity is striking between the signs and symptoms of RSD and those peripheral manifestations of upper thoracic somatic dysfunction, so much so that one might be tempted to suggest that RSD is merely the peripheral effect of untreated spinal somatic dysfunction. Certainly, patients with clinical presentation resembling RSD respond well to OMT. However, in my opinion, it is too soon to classify RSD as a complex of symptoms resulting from somatic dysfunction.

The physiology of spinal somatic dysfunction has been extensively studied. Our current understanding of this physiology might greatly clarify our understanding of RSD. Whether RSD is the result of somatic dysfunction or a man-

ifestation of shared neurophysiology, the relationship between somatic dysfunction and RSD—and the effect of OMT on RSD— warrant definitive study. Such a study might well be attempted as an outcomes project in which persons with independently diagnosed RSD are diagnosed for somatic dysfunction and treated as outlined herein with OMT.

## References


**1.** Veldman P, Reynen H, Arntz I, Goris R: Signs and symptoms of reflex sympathetic dystrophy: Prospective Study of 829 patients. *Lancet* 1993;342 1012-1016.

**2.** Dotson RM: Causalgia—Reflex sympathetic dystrophy—Sympathetically maintained pain: Myth and reality. *Muscle & Nerve* 1993;16: 1049-1055.

**3.** Larson NJ: Functional vasomotor hemiparesthesia syndrome. *Academy of Applied Osteopathy 1970 Yearbook* Carmel, Calif, Academy of Applied Osteopathy 1970:39-44.

**4.** Larson NJ: Osteopathic manipulation for syndromes of the brachial plexus. *JAOA* 1972; 72 378-384.

**5.** Larson NJ: A Study of the response of uncomplicated peripheral sensory disturbances to specific osteopathic manipulative treatment. *JAOA* 1972;72:62.

**6.** Kappler R, Kelso A: Thermographic studies of skin temperature in patients receiving osteopathic manipulative treatment for peripheral nerve problems. *JAOA* 1984;84:76.

**7.** Karstetta K, Sherman, R: Use of thermography for initial detection of early reflex sympathetic dystrophy. *J Am Podiatr Med Assoc* 1991;81;198-205.

**8.** Denslow J, Clough G: Reflex activity in the spinal extensors. *J Neurophysiol* 1941;4:430-437.

**9.** Denslow J, Korr I, Krems A: *Quantitative studies of chronic facilitation in human motoneuron pools.* Am J Physiol 1947;150:229-238.

**10.** Korr I, Wright H, Chace J: Cutaneous patterns of sympathetic activity in clinical abnormalities of the musculoskeletal system. *J Neural Transm* 1964;25:589-606.

**11.** Korr I, Wright H, Thomas P: Effects of experimental myofascial insults on cutaneous patterns of sympathetic activity in man. *J Neural Transm* 1962;23:330-355.

**12.** Schwab W: Principles of manipulative treatment: The low back problem. *JAOA* 1934;33: 286-288.

**13.** Kappler R: Postural balance and motion patterns. *JAOA* 1982;81:598-609.

**14.** Beal M: Viscerosomatic reflexes: A review. *JAOA* 1985;85:786-801.

**15.** Johnston W, Kelso A, Hollandsworth DL, Karrat JJ: Somatic manifestations in renal disease: A clinical research study. *JAOA* 1987; 87: 22-35.

**16.** Beal M, Morlock J: Somatic dysfunction associated with pulmonary disease. *JAOA* 1984; 84:179-183.

**17.** Korr I: Abstract: skin resistance patterns associated with visceral disease (1949). *Federation Proceedings* 1949;8:87.

**18.** Larson N: Summary of site and occurrence of paraspinal soft tissue changes of patients in the intensive care unit. *JAOA* 1976;1975: 840-842.

**19.** Beal M, Kleiber G: Somatic dysfunction as a predictor of coronary artery disease. *JAOA* 1985;85:302-307.

**20.**Selzer M, Spencer WA: Convergence of visceral and cutaneous afferent pathways in the lumbar spinal cord. *Brain Research* 1969; 14:331-348.

**21.** Buzzell Keith A: The potential disruptive influence of somatic input. In, *The Physiologic Basis of Osteopathic Medicine.* Glen Rock, New Jersey, Ronald Park Davis Publishing Co, 1970, pp 39-51.

**22.** Beal Myron C:Viscerosomatic reflexes. In, Patterson MM, Howell JN (eds). *The Central Connection: Somatovisceral/Viscerosomatic Interaction* Athens, Ohio, University Classics Ltd, 1989, pp 19- 26.

**23** Patterson M: A model mechanism for spinal segmental facilitation.*JAOA* 1976;76:62-72.

**24.** Korr I, Price T, Wright H: Patterns of electrical skin resistance in man. *J Neural Transm* 1958;17:77-96.

**25.** Hagen D: A continuing roentgenographic study of rural school children over a 15-year period. *JAOA* 1964; 63:546-557.

**26.** Patriquin DA:Viscerosomatic reflexes. In, Patterson MM, Howell JN (eds). *The Central Connection: Somatoovisceral/Viscerosomatic Interaction* Athens, Ohio, University Classics Ltd,1989, pp 4-12.

**27.** Appenzeller O: *The Autonomic Nervous System: An Introduction to Basic and Clinical Concepts.* New York, NY, Elsevier Publishing Co, Inc, 1970; 65.

**28.** Johnson A: Disabling changes in the hands resembling sclerodactylia following myocardial infarction. *Ann. Intern Med.* 1943;19:433-456.

**29.** Isselbacher KJ, Braunwald E, Wilson JD, et al:*Harrison's Principles of Internal Medicine,* ed 13. New York, NY, McGraw-Hill Health Professions Division; p 52.

**30.** Fryette H: *Principles of Osteopathic Technique* Carmel, Calif, Academy of Applied Osteopathy, 1954; p 4