CASE REPORT

Ann L. Habenicht, DO, FAAO

Resolution of Stridor, Gastritis and Cephalgia in an Adolescent with Reflex Sympathetic Dystrophy Following Osteopathic Manipulative Treatment—A Case Report

Introduction

Reflex sympathetic dystrophy (RSD) is a syndrome that commonly affects adults after trauma, resulting in severe pain and the loss of the use of one or more ipsilateral limbs. RSD in children is extremely rare. To compound this syndrome with stridor, gastritis and cephalgia would make a young life unbearable. The following is a report of such a case. This 12-year-old male endured 9 months of these symptoms, which were unresponsive to traditional pharmacological and physical therapeutic treatments. Using the tenets of osteopathy—structure denotes function—the stridor, gastritis and cephalgia were resolved after a series of osteopathic manipulative treatments (OMT).

Case Study

The patient, B.R., was a 12-year-old, right-hand-dominant male who presented in April 1992, for osteopathic treatment with a complaint of left elbow pain and stridor. The patient had been diagnosed with reflex sympathetic dystrophy (RSD) in November, 1991, following a football injury in September 1991. He complained of severe pain and hyperesthesia of his left elbow. He denied radiation of the pain, but admitted to the inability to fully extend his left elbow due to the pain. He stated that his elbow and forearm were swollen, and that he had increased sweating of his left forearm. Additionally, he complained of stridor, which had been present for the past 6 months. Prior to his injury, the patient had no arm pain or stridor.

On September 3, 1991, the patient was injured while playing football. He was the quarterback for his team and, while attempting to throw a pass, was tackled from the front by an opponent. The patient stated that as he was tackled, the other player's helmet struck the anterior portion of his elbow, pinning his elbow against the ground. B.R. admitted that he knew that he was injured, but he continued to play the remainder of the game. He returned home that evening with his elbow bent and painful. His arm was visibly bruised from the elbow to the wrist.

The next morning he was seen by a physician who diagnosed his injury as a "contusion" because his x-rays were negative for a fracture. After several days without improvement, B.R. was seen by an orthopedic surgeon, whose findings were the same. After 2 weeks, B.R. was referred to a pediatric orthopedic surgeon. An MRI of his left elbow was obtained and confirmed the previous diagnosis of "contusion." B.R.'s left

Corresponding Author

Ann L. Habenicht, DO, FAAO

Email: anniehaben@gmail.com

Editor's Note

This thesis, submitted to the Academy in 1995, was completed as part of Dr. Habenicht's requirement toward earning the designation of Fellow in the American Academy of Osteopathy, conferred in 1996. Opinions expressed in this article are those of the author and do not necessarily reflect the viewpoint or official policy of the American Academy of Osteopathy, and it was edited to conform to *AAOJ* style guidelines.

Keywords

Adolescent RSD, OMT, OMM, Stridor, Gastritis, Cephalgia, Complex Regional Pain Syndrome, CRPS

arm was placed in a sling, nonsteroidal anti-inflammatory medication was prescribed, and he was referred to physical therapy for treatment. After 1 week of physical therapy, the therapist suggested further evaluation. The orthopedic surgeon suggested an arthroscopy but this was declined by B.R.'s parents.

Several weeks later, on October 18, 1991, after laughing in class, B.R. suddenly began to cough. After approximately 30 minutes of uncontrollable coughing, B.R.'s respiratory rate suddenly increased to 30/minute. His breathing was shallow, with use of accessory muscles of respiration. A high-pitched, loud "cough" (consistent with stridor) was noted with each exhalation. He became pale and lethargic. He was transported to an emergency room for treatment. He was diagnosed with an upper respiratory infection, treated with oxygen, and asked to follow up with a local pediatrician. His stridor persisted.

The following day, he was seen by a pediatrician who immediately referred him to a pediatric otolaryngologist. A bronchoscopy was performed and the respiratory tract was found to be normal. Over the next several months, B.R. was evaluated by several allergists, a pediatric neurologist and a pediatric pulmonologist. He was treated with albuterol, steroids, theophylline, hydroxyzine, and amitriptyline without resolution of the stridor. Speech pathology therapy also had no affect. He underwent an additional laryngobronchoscopy with the inhalation of an oxygen-helium mixture, which did not contribute to his diagnosis. Tourette's syndrome was contemplated and treatment with pimozide was unsuccessful. Meanwhile, he continued his search for treatment of his elbow pain. He was referred by the pediatric orthopedic surgeon to a pediatric physiatrist who diagnosed B.R. as having RSD due to his arm's physical findings. His left arm and forearm were now puffy with a bluish color. The skin texture was slick with decreased hair. The physiatrist recommended a guanethidine regional block. B.R. underwent 6 such blocks with minimal change. B.R. was referred to an anesthesiologist for pain management.

At about the same time, B.R. also began having stomach upset and suboccipital/frontal headaches. He was evaluated by a pediatric gastroenterologist who performed an upper endoscopy. Although the findings were normal, B.R. was started on ranitidine and sucralfate. He experienced no improvement.

During the ensuing months, B.R. also consulted a chiropractor, but received no treatment. Consultations with a clinical psychologist and a biofeedback therapist failed to resolve the arm pain, gastritis, or cephalgla.

Seven months after the injury, B.R. (now 12 years old) presented to my office. Physical examination on the initial visit in April 1992, showed his vital signs to be stable with a pulse of 82, respirations of 16 and a B.P. of 80/50. HEENT were normal with the exception of an audible stridor. Heart auscultation revealed a regular rate and rhythm. Lungs were clear to auscultation. Abdominal examination revealed bowel sounds in all quadrants, without masses, tenderness or organomegaly. Neurological exam was negative, with the exception of hyperesthesia of the antecubital and lateral epicondylar areas on the left. The left arm also demonstrated hyperhidrosis and swelling without ecchymosis of the left elbow and forearm. There was an approximate 30% restriction of full extension of the left elbow. Structural exam revealed flattening of the upper thoracic A/P curve. The left acromioclavicular joint and inferior scapular border were higher than the respective structures on the right. The left iliac crest and posterior superior iliac spine (PSIS) were lower than the right. The sacrum was noted to be sidebent to the left. There was a pelvic sideshift to the right. Acute tissue texture changes were noted in the left paravertebral areas at the levels of T4, T3, and C2. Segmental motion changes were noted and consistent with somatic dysfunction at T4 ESR_L, C2 FSR_L, and the third rib on the left. Fascial restrictions of the left hemi-pelvis and left hemi-diaphragm were appreciated. The CRI was 10 cycles/min with a good amplitude.

Diagnoses arrived at were RSD, possible sympathetic dystonia and somatic dysfunction of the cervical, thoracic and pelvic areas. Because of the severity of the somatic dysfunction, it was elected to treat B.R. initially using indirect myofascial technique.

Over the next 2 weeks, B.R. was treated twice using myofascial techniques and, finally, high velocity, low amplitude technique (HVLA) for the recurrent T4 ESR_L, T3 rib posterior left, and C2 FSR_L.

On the fourth visit, B.R.'s mother reported a decrease in the incidence of his stridor. T4 was still dysfunctional, however, there appeared to be less restriction of forward bending. A significant fascial strain of the thoracic inlet was now noted with the fascial pull to be directed into the left cervical area near C2. Again, he was treated using myofascial release and HVLA.

Following the fourth treatment, 7 weeks after treatment was begun, the stridor was no longer present. Additionally, his gastritis and cephalgia had resolved. Structurally, T4 left now showed normal motion with overall good thoracic motion. The C2 left dysfunction was improved. B.R.'s arm pain was now noted to be at the level of 8 on a scale of 1 (no pain) to 10 (maximum pain), and B.R. had begun to participate in sports again.

Six months later, after falling on his outstretched left arm while playing basketball, B. R.'s stridor suddenly returned. His structural exam revealed the return of the T 4 left extended segment with a fascial restriction of the upper thoracic area. B.R. was treated with HVLA and myofascial release (MFR) techniques with good results. Over the next 4 months, B.R. was treated 3 additional times. The stridor resolved, once more, and the normal motion was restored to the thoracic area. To date, B.R. has remained free of stridor, gastritis and cephalgia.

Discussion

This case demonstrates the osteopathic tenets of structure/function. In the case of B.R., the maintenance of the upper thoracic segment somatic dysfunction appears to be the "keystone" in his dysfunction. B.R.'s structural dysfunctions were not addressed until he was evaluated by an osteopathic physician.

In examining the vector forces of the initial injury, it appears that his upper thoracic area was forced into hyperextension at the T4 level. This would be consistent with a fall backwards, with the left arm slightly outstretched. The backward force into the ground with the arm slightly abducted would cause the upper thoracic area at the level of T4 to move into extension. Thus, an initial extension injury of this area was most probable. The subsequent fascial restrictions that resulted from the injury were maintained due to the patient's physiologic splinting of his arm. This was further facilitated by the amount of time that elapsed before he received specific osteopathic treatment.

Due to B.R.'s failure to respond to traditional allopathic treatment, it is most probable that his symptoms were a direct effect of his structural dysfunctions. How his structural dysfunctions caused the symptoms of stridor, gastritis, cephalgia and possibly the RSD can be addressed by the following hypotheses:

1. The stridor was a result of fascial restrictions due to the hyperextension of the T4 segment.
2. The gastritis was the result of a somatovisceral reflex from the chronic T4 facilitated segment causing the resultant gastritis due to the innervation of the stomach at the same thoracic level.
3. The cephalgia may be a direct result of the fascial restriction of the prevertebral fascia and its restriction in the suboccipital area OR It may be a result of the vagal response to the gastritis, causing a viscerosomatic reaction in the suboccipital area resulting in headache.
4. The RSD may be a result of the continued inappropriate sympathetic tone that may result in this area due to the restriction and subsequent tissue edema of the T4 area. The high thoracic area has a significant structural relationship to the sympathetic chain and ganglia.

In order to consider the validity of each of the hypotheses, one must first have an understanding of fascia, somatovisceral interactions, and RSD.

Fascia

Fascia is loose-to-dense fibroelastic connective tissue that invests the entire body. It supports and creates the body framework. It limits and directs planes of motion throughout the body. It protects and allows contiguous structures to communicate with each other without disturbing their individual functions. Fascia has tensile strength. It induces platelet aggregation when disrupted.

Fascia has a viscoelastic property that, when under tension, allows it to deform.[21] Under a sustained load or tension, the fascia will stretch. This continued stretching is called "creep." When the load is removed, the tissue will begin to revert back toward the original state. This reversal of the stretch toward the original state, but still having a slight deformation is "hysteresis."[5] Each time the fascia is treated, less hysteresis occurs. Eventually, the fascia stabilizes.[5]

There are three kinds of fascia: superficial, deep and subserous. Superficial and deep fascia, found as complete ensheathments, from the base of the skull over the face and neck, around the trunk and around the limbs.[3] Superficial fascia also contains the deep pressure receptors, the larger lymph vessels, blood vessels and nerve trunks.[3]

The fascia of importance in this case is the prevertebral fascia, a tough layer of the cervical fascia which is attached to the base of the skull and to the transverse processes of the cervical vertebrae. It covers the

prevertebral muscles, the scalenes, and the deep muscles of the back. Therefore, it covers the floor of the posterior triangle of the neck.[8] It blends into the periosteum of the vertebral bodies of the thoracic vertebrae.[1] In front of the subclavian artery, the prevertebral fascia extends laterally as the axillary sheath and invests the brachial plexus.[8] An additional layer, the alar, or anterior lamina, lies anterior in the lower neck and goes into the mediastinum. It blends into the visceral fascia of the trachea and esophagus.[1,9] The recurrent laryngeal nerve lies within this fascia and is potentially affected by it.

The recurrent laryngeal nerve innervates all the muscles of the larynx, except the cricothyroid. The cricoarytenoid, which controls the glottis, could be affected by the fascial strain in this nerve, thereby causing stridor.[1]

Additionally, the prevertebral fascia is also connected to the carotid sheath.[9] The carotid sheath is in direct contact with the vagus nerve.[1]

Fascial tension (as in this case) developing from a backwardbent, sidebent, and rotated fourth thoracic segment could directly affect the recurrent laryngeal nerve, causing stridor. Additionally, the cough can cause increased fascial imbalance, affecting the thoracic area/diaphragm and subsequent prevertebral fascia. Since the prevertebral fascia also encompass the brachial plexus, and the brachial plexus may be related to upper extremity dysfunction, the fascial restriction may also result in symptoms resembling RSD.

The origin of the prevertebral fascia is at the base of the skull. Increased tension in this fascia can cause suboccipital cephalgla. One must also remember that the vagus nerve is in close association with the carotid sheath and the tension transmitted into the sheath as a part of the prevertebral fascia will have an effect on the vagus nerve. Vagal nerve irritation may also account for the cephalgia. This is due to the connections the vagus has with the first two cervical somatic nerves. These cervical somatic nerves provide neurologic pathways for fibers carrying pain fibers from the posterior portion of the head (the occipital nerves).[16]

Somatovisceral Reflexes

The concept of viscerosomatic and somatovisceral reflexes has been acknowledged in the literature for quite some time.[2,6,10,20] In order to understand these concepts, one must first acknowledge the concept of the facilitated segment.

I. M. Korr, PhD describes the facilitated segment as a spinal cord segment in which the neurons which mediate sensory, motor and autonomic function are maintained in a state of hyperexcitability. This allows these neurons to respond more easily to stimuli: normal, abnormal or prolonged.[15,20] This means the resting potential is elevated and tissues, somatic or visceral, innervated from the facilitated segment will become hypersensitive to all stimuli coming into the segment.[20] Facilitation creates a situation wherein the efferent impulses, somatic and visceral, reaching the spinal cord find much easier access to the spinal cord and higher cortical centers.[20]

Myron Beal, DO reminds us that:

> stimuli from viscera, somatic (sic), and higher centers all converge at the T cells of lamina 5 of the dorsal horn of the spinal cord. The proximity of sensory input provides a theoretical opportunity for interrelationship between the somatic and visceral systems and the higher centers.[2]

Thus, the concepts of viscerosomatic reflexes (visceral afferents returning to the facilitated segment at the cord affecting somatic efferents) and somatovisceral reflexes (somatic afferents affecting visceral efferents) are defined.

The autonomic nervous system plays an important role in viscerosomatic and somatovisceral reflexes. William deGroat, PhD stated that striated muscles and visceral organs are controlled by somatoneurons and autonomic neurons respectively.[6] Furthermore, the somatic and autonomic neuronal pathways are closely coordinated in their function. He notes that interactions between visceral and somatic components of the nervous system can be altered by disease, injury or drugs, and that the changes in the linkage between visceral and somatic reflex pathways can induce or exacerbate organ dysfunctions or symptoms.[6]

William Johnston, DO described somatovisceral reflexes or interactions as a continuing uneven barrage of proprioceptive stimuli of the afferent pathway. This barrage is due to the stress of asymmetric behavior of the "lesioned spinal segments" during movement. This altered spinal reflex control then modifies visceral function.[10] He further noted that restoration of mobile symmetry at the dysfunctional segment through osteopathic manipulation reduces the inappropriate activation of somatic afferents.

Therefore, chronic somatic dysfunction producing a

facilitated segment has the potential to affect visceral function at that same segmental level. The level of involvement in the case of B.R. is T4 left. The literature notes various organ systems that share innervation from this level including heart, stomach and lung.[2,20]

Since facilitation of the visceral component is mediated through the autonomic nervous system, sympathetic and parasympathetic nervous supply to the involved viscera are important. In the case of B.R.'s symptoms, the visceral component is the stomach. As previously noted, T4 segmentally may involve the stomach. The sympathetic input would involve the sympathetic chain. Anatomically, the sympathetic chain ganglia are closely applied against the ventral aspect of the costovertebral articulations by the deep prevertebral fascia.[19] It is thought that reduced motion in any musculoskeletal articular structure results in decreased local blood and lymph flow. This resulting congestion leads to local accumulation of metabolic byproducts and irritation. Proper fluid motion is essential for local tissue health and function. Therefore, the loss of motion ultimately results in visceral sympathetic dysfunction. This occurs due to the effect of somatic dysfunction and spinal facilitation upon the sympathetic ganglia. The restoration of motion/function should improve visceral function.[19] Johnston noted that following successful manipulative intervention, the return of symmetry in motor function initiates changes in nervous, circulatory and metabolic activity locally. The remote effects in distant visceral tissues result from the changes in reflex activity at the cord segment.[10]

Therefore, the above mechanism supports the hypothesis that the lesioned or dysfunctional segment at T4 left caused a somatovisceral reflex to the stomach resulting in symptoms of gastritis which was unresponsive to traditional pharmaceutical treatment. Furthermore, the influence of the vagus nerve on the stomach and the influence from the prevertebral fascial restriction on the vagus nerve, may have also compounded the dysfunction of the stomach.

Reflex Sympathetic Dystrophy

Reflex sympathetic dystrophy (RSD) was first described in 1864[18] as reflex neurovascular dystrophy. Although it is a well known entity in adults, it is rarely diagnosed in children.[14] RSD results from an abnormal excessive sympathetic nervous system response affecting the extremities.[14] Reflex sympathetic dystrophy almost always begins as a result of minor trauma, such as a sprain, which resolves. However, not only does the pain continue, but it spreads to the entire limb. This can be accompanied by hyperesthesia, followed by swelling, hyperhidrosis, discoloration, coolness, and loss of motion due to the persistent pain.[4,13] The pathophysiology involves reflex of abnormally circuited discharges of pain fibers and sympathetic fibers. The abnormal circuit includes referral of the distal pain to more centralized areas In the nervous system and sympathetic transmission, causing abnormal activity.[4]

There are many disorders described that have the same characteristics as RSD, such as causalgia, post-traumatic pain syndrome, spreading neuralgia, and shoulder hand syndrome. The present confusion regarding the various definitions, diagnoses, and pathophysiologic mechanisms underlying these entitles leads one to question: "Who truly has RSD and what is it?"[7] In all probability, there are multiple pathophysiologic mechanistic explanations for the different components for any given symptom of RSD. Some may involve the central nervous system, some the peripheral nervous system, and some may even have no pathologic basis in the pain pathways.[7]

If we look at the osteopathic literature, we find a syndrome that seems to have similar symptoms as can be found in a mild form of RSD. This syndrome was first recognized by Norman Larson, DO in 1970 describing the sensory change affecting one-half of the body, initially the head, shoulder and arm. He continued to further describe the symptoms as pain or anesthesia. If this condition is protracted, secondary changes involving the circulation, such as swelling and coldness may appear. Later, joint changes with pain and swelling may be noted. This syndrome is known as functional vasomotor hemiparaesthesia.[17] Dr. Larson further made the observation that the somatic dysfunction seems to be related to the upper thoracic spine and the related rib.[17] Of further importance in this case is to look at the observations of Robert Kappler, DO regarding patients with peripheral nerve problems who have received OMT. He observed that OMT alters autonomic function and that OMT of somatic dysfunction below the anatomic origin of the brachial plexus is effective in the treatment of many upper extremity complaints.[12]

With this information at hand, it then is reasonable to consider the hypothesis that the injury induced extended T4 segment was responsible for the maintenance of the RSD in the patient described in this case.

Summary

We may now consider the four hypotheses concerning the structure-function interaction in this case.

> "1. The stridor was a result of fascial restrictions due to the hyperextension of the T4 segment."

We have seen that the prevertebral fascia can be placed on tension due to the anatomical relationship with the backwardbent fourth thoracic segment. We also know that the prevertebral fascia is of great importance in the tension that can be placed on nerves within this fascia, specifically the recurrent laryngeal and the vagus. It is highly probable that the tension on the recurrent laryngeal nerve by the fascial strain caused the production of the stridor. This hypothesis is further supported by the fact that once the fascial strain had been removed, by treating the prevertebral fascia and the causative T4, the stridor ceased. Additionally, when trauma once again reinstated the thoracic dysfunction, the stridor returned, only to cease once the motion of the thoracic segment and fascia had been restored. Kappler notes that, as a rule, areas where the anteroposterior (spinal) curves are flattened (in this case extended), "trouble spots" tend to occur.[11] This certainly is the case of this thoracic segment.

> "2. The gastritis was the result of a somatovisceral reflex from the chronic T4 facilitated segment causing the resultant gastritis due to the innervation of the stomach at the same thoracic level."

We have seen that the chronic fourth thoracic segment somatic dysfunction would be expected to be in a state of facilitation. The close proximity of the sympathetic ganglia to this thoracic segment and the prevertebral fascia will definitely affect the autonomic impulses from this segment. This somatic dysfunction is capable of producing the gastric symptoms through a somatovisceral reflex. Affirmation of this hypothesis is obtained by the resolution of the gastric symptomatology when motion of the thoracic segment and fascia were restored.

> "3. The cephalgia may be a direct result of the fascial restriction of the prevertebral fascia and its restriction in the suboccipital area OR It may be a result of the vagal response to the gastritis, causing a viscerosomatic reaction in the suboccipital area resulting in headache."

We have seen how the fascial tension on the recurrent laryngeal nerve affected the cricoarytenoid muscle producing stridor. The fascial tension could also cause tension on the suboccipital musculature causing restriction of motion of the cervical/suboccipital area (as seen by the $C2FSR_L$). Motion restriction in this area will cause headache starting in the suboccipital area that then affects the frontal musculature. Resolution of the suboccipital tension by the restoration of the motion of the fascia and the cervical segment should cause the headache to cease. This is what has resulted in this case.

Additionally, the viscerosomatic reflex from the vagus nerve as a causative agent for headache is well known.[16] The restoration of normal function of the structure caused the gastric symptoms to resolve. The resolution of the gastric symptoms should cause the viscerosomatic reflex to resolve, thus the cephalgia should resolve. The cephalgla resolved after the thoracic and cervical motion was restored.

> "4. The RSD may be a result of the continued excessive sympathetic tone that may result in this area due to the restriction and subsequent tissue edema of the T4 area. The high thoracic area has a significant structural relationship to the sympathetic chain and ganglia."

We have seen that somatic dysfunction in the high thoracic area has a significant effect on the sympathetic nervous system.[17,19] The observation by Kappler regarding OMT and altered autonomic function in upper extremity complaints also refers us to the upper thoracics. It is therefore reasonable to consider that the extended segment of the fourth thoracic vertebra could be a factor in the RSD. This is verified by the fact that the patient's pain decreased after restoration of thoracic motion. The thoracic segment was not the sole contributor in the production of the RSD. If it had been, B.R.'s pain should have significantly resolved with the restoration of motion. As it stands, it is my opinion that a significant contributing factor in this patient's left arm pain was due to the fascial restriction of the arm and elbow. B.R. was unwilling to allow me to touch his elbow or allow me to release the fascial dysfunction in this area. An interesting note is that several weeks after my last visit with B.R., his mother reported that he had fallen on his outstretched left arm. He heard a loud "pop," and his arm pain dropped immediately to 2 out of 10.

Conclusion

In conclusion, B.R.'s case is an excellent example of

structure-function. It shows the integration of the somatic with the visceral system. B.R. had failed to be diagnosed with traditional methods simply because no one had addressed the structural system. Once properly addressed, B.R.'s system was able to respond and allow him to begin to live as a normal 12-year-old.

References

1. Allin, Edgar M.D., personal communication. Professor of Anatomy. Chicago College of Osteopathic Medicine. January, 4, 1995.
2. Beal, M.C.: "Viscerosomatic Reflexes: A Review" Journal of the American Osteopathic Association, Vol.85, No.12, December 1985, pp. 786-801.
3. Becker, RF.: "The Meaning of Fascia and Fascial Continuity" Osteopathic Annuals, Vol.3, No.6, June 1975, pp.35-47.
4. Berger, G., Izzo, K.: "Hand Pain and Paresthesias, Part 2: Reflex Sympathetic Dystrophy, Thoracic Outlet Syndrome, and Entrapment Syndromes of the Median and Ulnar Nerves" Osteopathic Medical News, Vol.8, No.3, March 1991, pp. 1-13.
5. Chila, A.G.: "Fascial-Ligamentous Release: The Indirect Approach" in Foundations for Osteopathic Medicine ed by Robert c. Ward, Chicago, 1996.
6. deGroat, W.C.: "Viscerosomatic Interactions and Neuroplasticity in the Reflex Pathways Controlling Lower Urinary Tract Function" in The Central Connection: Somatovisceral Niscerosomatic Interaction ed by M.M. Patterson and J.N. Howell, University Classic ltd, Athens, OH, 1989, pp. 30-46.
7. Dotson, RM.: "Causalgia-Reflex Sympathetic Dystrophy-Sympathetically Maintained Pain: Myth and Reality" Muscle & Nerve. 1993, pp. 1049-1055.
8. Gardner, Grey, O'Rahilly: Anatomy. 5th edition, Saunders, 1986, pp. 722-23.
9. Hollinshead, W.H., Rosse, C.: Textbook of Anatomy, 4th edition, Harper & Row, 1985, pp. 824-27.
10. Johnston, W.L.: "Osteopathic Clinical Aspects of Somatovisceral Interaction" in The Central Connection: Somatovisceral Niscerosomatic Interaction ed. by M.M. Patterson and J.N. Howell, University Classic Ltd, Athens, OH, 1989, pp. 30-46.
11. Kappler, RE.: "Postural Balance and Motion Patterns" Journal of the American Osteopathic Association, Vol.81, No.9, May 1982, pp. 598-609.
12. Kappler, R.E., Kelso, A.F.: "Thermographic Studies of Skin Temperature in Patients Receiving Osteopathic Manipulative Treatment for Peripheral Nerve Problems" Journal of the American Osteopathic Association, Vol.84, No.10, September 1984, p. 76.
13. Karstetter, K.W., Sherman, R.A.: "Use of Thermography for Initial Detection of Early Reflex Sympathetic Dystrophy" Journal of the American Podiatric Medical Association, Vol.81, No.4, April 1991, pp. 198-205.
14. Kelly, W.N., Harris, E.D., Ruddy, S., Sledge, C.B.: Textbook of Rheumatology. 2nd edition, W.B. Saunders, Philadelphia, 1985, pp. 446-7.
15. Korr, I.M.: "The Neural Basis of the Osteopathic Lesion" in The Collected Papers of Irvin M. Korr ed by B. Peterson, American Academy of Osteopathy, 1979, pp. 120-127.
16. Kuchera, M., Kuchera, W.A.: Osteopathic Considerations in Systemic Dysfunction, KCOM Press, Kirksville, MO, 1991, p. 85.
17. Larson, N.J.: "Functional Vasomotor Hemiparesthesia Syndrome" Yearbook of the American Academy of Osteopathy. 1970, pp. 39-44.
18. Mitchell, S.W., Morehouse, G.R., Keen, W.W.: Gunshot Wounds and Other Injuries of Nerves, J.B. Lippincott Co., Philadelphia, 1864, p. 164.
19. Patriquin, D.: "Viscerosomatic Reflexes" In The Central Connection: Somatovisceral Niscerosomatic Interaction ed by M.M. Patterson and J.N. Howell, University Classics Ltd, Athens, OH, 1989, pp. 4-12.
20. Walton, W.J.: Textbook of Osteopathic Diagnosis and Technique Procedures, 2nd edition, Matthews Book, St. Louis, MO, 1970, pp.91-102.
21. Ward, R.C.: "Basic Concepts of Myofascial Release" in Foundations for Osteopathic Medicine ed by Robert C. Ward, Chicago, 1996. ■