A.T. Still's Osteopathic Lesion Theory and Evidence-Based Models Supporting the Emerged Concept of Somatic Dysfunction

Torsten Liem, MSc Ost, MSc Paed Ost

Financial Disclosures:
None reported.

Support: None reported.

Address correspondence to Torsten Liem, MSc Ost, MSc Paed Ost, Osteopathie Schule Deutschland, Mexikoring 19, 22297 Hamburg, Germany.

E-mail:
tliem@osteopathie-schule.de

Submitted November 13, 2015; final revision received June 4, 2016; accepted June 10, 2016.

Andrew Taylor Still, MD, DO, coined the original idea of *lesion* based on the obstruction of flow of body fluids, but primarily referring to bony structures and more precisely to the spine. Throughout the 20th century, this idea was shaped and developed into the concept of somatic dysfunction, a term that is familiar to both US-trained osteopathic physicians and foreign-trained osteopaths and has been an essential cornerstone of osteopathic practice and teaching. The present historical narrative review offers an overview of the evolution of Still's original lesion concept, major evidence-based models of somatic dysfunction that attempt to explain the clinical findings, and a critique of the concept.

J Am Osteopath Assoc. 2016;116(10):654-661
doi:10.7556/jaoa.2016.129

Keywords: Andrew Taylor Still, A.T. Still, osteopathic lesion, osteopathy, somatic dysfunction

In 1874, Andrew Taylor Still, MD, DO, the founder of osteopathy and its clinical, diagnostic, and therapeutic implementations, developed a theory that the disease process arises when the flow of life is interrupted.[1] A *lesion*, as he termed this interruption of flow, is any change of tissue structures in size, texture, structure, and position.[2(p398)] Throughout the 20th century, other US-trained osteopathic physicians and foreign-trained osteopaths evolved Still's original term to become *osteopathic lesion* and to what is now known as *somatic dysfunction*.

To understand and classify osteopathic approaches, knowledge of the lesion concept (ie, somatic dysfunction) and its history are important. Models of the concept based on research findings published as recently as 2016 may aid in clinical reasoning of the most appropriate treatment strategies, as well as specific osteopathic manipulative treatment, osteopathic manipulative therapy (manipulative care provided by foreign-trained osteopaths), and nonmanual therapeutic approaches.

In the present article, I provide an overview of Still's original lesion concept and its evolution, as well as a brief introduction to evidence-based models that took shape in the second half of the 20th century and continue to evolve.

The Original Lesion Concept

Still's fascination with machinery and technology was based on the Industrial Revolution of the 19th century and classical Newtonian mechanics, whose laws describe a perfectly constructed motion under a system of forces. Likewise, Still described the human body as a delicate and perfect machine and the osteopath as the mechanic who examines the machine man

for stress, strain, and variations from the norm and then corrects or adjusts to reestablish the fine balance so that healing can commence.[1(pp287,341),3(p7)] Still was also influenced by the emerging wave of anatomization in medicine, such as cadaver dissections and the anatomical study of the human body, as well as by the ideas of spinal irritation and obstruction.[4(pp250,260)] He was also inspired by Emanuel Swedenborg's concept of obstruction.[5] Relating to spiritual and physical aspects, Swedenborg believed that the soul was dissolved in the body fluids and distributed all over the body; any obstruction of body fluids by disease had to be removed to restore the unhindered flow, and obstructions in the spiritual sense had to be addressed to restore the divine order.[6(p204)] Still combined bone setting (ie, joint manipulation) and mesmerism[7(p19),8(p202)] with an anatomical point of view.

Still developed a predominantly mechanistic theory on how disorders of the vital body fluids (ie, blood, lymph, cerebrospinal fluid) and their flow occur. He posited that bones, muscles, membranes, organs, nerves, blood, and lymph are interlinked harmoniously. If these mechanical disturbances were eliminated, the unhindered flow could be restored.[9(p218)] To Still, disease was the result of an anatomical abnormality that could lead to physiologic impairments. He believed that health is based on structural integrity and develops when the flow of body fluids is unhindered.[10] Obstructions of these fluids or their neural control centers can lead to disturbances in the flow, which Still referred to as *lesions*.[10(p10)]

These lesions were to be corrected by osteopathic "adjustment" (osteopathic manipulative treatment or osteopathic manipulative therapy) rather than pharmaceutical drugs because flow constituted the foundation of health itself.[10(p12)] Still's theoretical structural view of the lesion acknowledges that the body protects itself against disease by its own defenses and regains health by its inherent abilities.[2(p509)] The aim was therefore to adapt the body from the abnormal to the normal state (ie, to manipulate bones, ligaments, and muscles to achieve normal flow of fluid and function of nerves and excretory systems).[3(pp8,14)]

Still generally referred to bony lesions, mostly of spinal structures, but also to other joints, such as hip, rib, and pelvic joints.[3(pp16,37,44,45,65,94,107,115),11(pp11-56)] He used the term, though rarely, in relation to structures of the nervous system, the viscera and its membranous structures, the skin, the intestines, and blood.[3(pp113,137,151,186,190),12(p242)] With lesions, the nervous and vascular functions are affected[3(p65),13(p355),14(p83)]; for example, ligaments are held under tension as a result of bone displacement, compressed nerves, or compressed blood vessels.[11(pp3,72,74),14(pp44,49)] Spinal vertebrae may be displaced for many reasons, such as strain or overstretched ligaments.[11(p6)]

Still and other osteopaths of his time essentially agreed that primary lesions were caused by more or less strong external forces, particularly in the region of the spinal column, and that the main cause in secondary lesions was not within the respective joint but in a location distant from the spine.[2(pp42,145,161),10(p35),11(pp68,89),13(pp36,38),14(pp93,173),15(p43)]

The lesion model was important for the early teaching of osteopathy in the United States. At the sixth annual meeting of the Committee of Education of the American Association for the Advancement of Osteopathy in Milwaukee, Wisconsin, in 1902, the prevalence of lesions in all states of disease was an important point on the agenda.[2(p263)]

Osteopathic Lesion and Spinal Lesion

Although Still used *lesion* relatively imprecisely and without further definition, other osteopaths developed and shaped it over time. The term *osteopathic lesion* was developed on the basis of Still's understanding of obstruction of body fluids or their neural control centers. To define an impairment as an osteopathic lesion, it was initially necessary to have a structural disorder of interrelated parts or a change in the size of the individual parts, such as overgrowth, growth arrest, and atrophy, which then led to a functional disorder.

Guy Dudley Hulett, DO, was first to document the concept of an osteopathic lesion as "any structural perversion which by pressure produces or maintains functional disorder."[16(p76)] He not only refers to the bony lesion, but he also includes all types of tissue, including muscles, ligaments, and viscera. Hulett[16] differentiated 3 types of osteopathic lesions, characterized by a change of the positional relationships of bones, joints, and organs (*Table 1*). Dislocation and subluxation mainly referred to bony tissue, making a distinction between complete (dislocation) and incomplete (subluxation) separation of the joint surfaces. Displacement referred in particular to flexible structures, such as organs (eg, a prolapsed uterus).

Hulett used the term *spinal lesion*, not to define diseases or malformations of the vertebrae, but instead to describe mostly unobtrusive subluxations that were involved in the maintenance of that lesion, usually in conjunction with bones, ligaments, and muscles.[10(pp11,20),16(pp81,83,85)] The spinal lesion played a special role in the etiologic consideration of disease. Hulett for example, mentioned the involvement of spinal lesions in cardiac disorders and constipation.[16(pp96,161)]

In 1935, George Malcom McCole, DO,[10] criticized Hulett's definition for the inclusion of bone diseases, tumors, and major injuries, which he claimed would not be amenable to osteopathic manipulation.[10(p18)] He, in contrast, defined the osteopathic lesion as any restriction of spinal joint movement that could be resolved by an osteopathic intervention.[10(p17)] McCole further stated that the osteopathic lesion was a result of the joint rhythm or the action of the tissue of these joints (including spinal cord segments and sympathetic ganglia), which in turn can cause local or peripheral tissue disorders.[10(p17)] These changes can occur both in the anatomical normal state as well as in abnormal joints and can be corrected by osteopathic manipulation.[10(p20)]

McCole differentiated 4 types of lesions: traumatic, reflectory, acute, and chronic, as well as a combination of these lesions (*Table 2*). These lesions change over time and should be understood to some degree as a mixture of lesions rather than in isolation.[10(p17)] All of these lesions, regardless of their cause or the time of their origin, are responsible for causing the same diseases.

At the time, *osteopathic lesion* generally referred to disturbances of the spinal structures.[10(p11)] Hence, the terms *osteopathic lesion* and *spinal lesion* were often used interchangeably. A spinal lesion was said to be characterized by the nonphysiologic articulation of affected joint surfaces in the resting phase or by a disturbed intra-articular tension caused by paravertebral contractures or contractions of tissue (eg, muscle, ligaments, capillaries, nerves, nerve centers).[10(pp11,19)] Spinal lesions were also thought to occur as a reflectory spinal muscle tension caused by visceral irritation and illness.[16(p81)]

An anatomical deformity or positioning of the spine and ribs would directly affect the "vital channels" (ie, the free flow of all fluids in arteries, veins, lymphatic vessels, and the cerebrospinal fluid) and the free conduction of the nerves, according to Carl Philip McConnell, DO, who considered Still's entire original approach in his theory.[17(p16)]

Yale Castlio, DO, differentiated an osteopathic lesion as a faulty position and movement restriction of bony joint structures from a spinal lesion as a lesion of one of several joint facets between 2 vertebrae.[10(pp18,19)] According to Castlio,[17] an osteopathic lesion also affected tissues and organs distant from the lesion. In 1930, Castlio expanded Hulett's definition of spinal lesion[16] and other definitions by applying an exclusive focus on joint disorders.[18] He considered palpable changes of associated soft tissue (ie, muscle contractions; thickened ligaments; edema; acidosis; neuritis; vasomotoric, trophic, and metabolic changes; and movement restrictions) involving the spinal cord and sympathetic ganglia.[18(pp23,26,28)]

According to Castlio, an osteopathic lesion affected nerves that innervate an organ and may impair the organ as well as the overall health of the organism and thus predispose it to disease. An osteopathic lesion also potentially affected an organ in immediate physiologic interaction with a different tissue and therefore also

affected this tissue.[18(p69)] Effects included increased metabolism and increased and often disturbed motor and secretory activity, accompanied by stasis.[18(p76)] Long-term effects were thought to be inflammatory or degenerative changes with impaired metabolism and function of the affected organ or tissue.[18(p83)]

Table 1.
Hulett's Classification of the Osteopathic Lesion, 1906[15(p77)]

Type of Lesion	Process	Tissue Involved
Dislocation	Complete shift	Bone
Subluxation	Partial shift	Bone
Displacement	Dislocation	Organs

Further Evolution of the Osteopathic Lesion Concept

Greater Osteopathic Lesion Complex

In 1923, the osteopathic lesion concept was extended by Carter Harrison Downing, MD, DO,[19] who used the term *greater osteopathic lesion complex* to describe adaptive consequences in the nervous system, circulatory system, secretory system, and excretory system.[19(pp15,16)] The greater osteopathic lesion complex included impairments of normal spinal joint mobility within anatomic limits of movement that were accessible to osteopathic manipulation and thus excluded diseases or disorders of the spine, such as fractures or ankylosis.[10(p10)] This impairment is a reflectory effect of spinal cord regions and can cause disorders close to and distant from tissue.[10(p10)]

Environmental Lesion

John Martin Littlejohn, PhD, DO, MD, explored a lesion concept that differed from previous definitions and stated that the body is not a mechanism but an organism. He went on to say that purely mechanical lesions therefore may not occur, as they are, for example, coupled with mental and psychological states, health, function, and structure. In this regard he used the term *environmental lesion*.[20(p66)]

Total Structural Lesion or Total Lesion

Harrison Fryette, DO, expanded on the thoughts of Arthur D. Becker, DO, who considered the total structural lesion as the primary mechanical lesion in addition to all consequential mechanical compensations.[21(pp41,80)] Fryette chose the term *total lesion* and no longer referred to only mechanical factors but any factors that predisposed patients to diseases.[21(p41)] These factors include, for example, environmental factors, infectious agents, nutritional factors, and emotional factors that can potentially affect health.[21(p41)]

Primary and Secondary Lesion

In 1935, George MacDonald, DO, and W. Hargrave-Wilson, DO, classified lesions according to causal aspects as primary and secondary lesions.[22(p36)] The primary lesion is a lesion of a joint, caused by an acute torsion; compression; load (strain); an acute, mostly very small trauma; or stress resulting from chronic torsion, compression, or load.[22(p36)] The latter usually occur at weak points of the spine, which are also dependent on posture, such as the spinal segments L5-S1, T11-12, C1-2, and C2-3.[22(p37)] Each active primary lesion was thought to be reinforced by additional secondary factors or a nonactive primary lesion could be reactivated by these factors.[22(p39)] The secondary lesion was also called a *reflectory lesion*; the primary cause was not in the respective joint but in an organ or other tissue distant from the spine, or it resulted from thermal conditions, or mental disorders, such as anxiety.[22(pp36,38)] Secondary lesions were caused by means of a reflex arc or by an irritation of the spinal cord segment (viscerosomatic reflex) that was caused by failure of an organ. This reflex or irritation could lead to ligamentous and muscular tension in the corresponding spinal area, which could become an active lesion that could in turn affect the organ even more (ie, somatovisceral reflex).[22(p38)]

Table 2.
McCole's Classification of the Osteopathic Lesion, 1935[10(pp17,29,35)]

Type of Lesion	Cause	Symptoms
Traumatic	Accident or trauma	Reflectory impulses of the associated spinal cord centers
Reflectory	Abnormal impulses that come from distant locations of the body	Stimuli in the appropriate centers of the spinal cord of the adjoining segment; influence on muscles and vessels of the joint
Acute	Traumatic or reflectory	Can form chronic manifestations such as fibrous infiltrations
Chronic	Traumatic or reflectory; often from untreated acute osteopathic lesions (ie, fibrous contracture)	Acute symptoms can be caused repeatedly; no clear boundaries between acute and chronic lesions

The effects of these lesions would manifest as local pressure phenomena, peripherally by vascular, sympathetic, or somatic nervous somatovisceral reflexes and generally directly or reflexly on the nervous, vascular, or endocrine system.[22(p61)]

The classifications MacDonald and Hargrave-Wilson[22] described are integral because they took into account, for example, pathophysiologic organ influences that would be supported by later research findings.[23(p19),24(pp636-642)]

Somatic Dysfunction and Its Evidence-Based Models

In the mid-1960s, the Hospital Assistance Committee of the Academy of Applied Osteopathy, chaired by Ira Rumney, DO, developed definitions for osteopathic diagnosis and treatment for inclusion in the *Hospital International Classification of Disease, Adpated*.[25,26] *Osteopathic lesion* was replaced with the term *somatic dysfunction* to provide insurance companies and the public with specific criteria for osteopathic service provision.[26,27] Today, the term *somatic dysfunction* is widely established and commonly used in osteopathic medical education and practice. The Educational Council on Osteopathic Principles defines somatic dysfunction as follows:

> Impaired or altered function of related components of the somatic (body framework) system: skeletal, arthrodial and myofascial structures, and their related vascular, lymphatic, and neural elements. Somatic dysfunction is treatable using osteopathic manipulative treatment.[28]

Typical diagnostic indicators for somatic dysfunction are tissue texture abnormality, asymmetry, restriction of motion, and tenderness of affected tissues.[29] Somatic dysfunction is commonly classified as being acute or chronic.[29] Plausible causes for the clinical signs of somatic dysfunction may be acute tissue inflammation or long-term degenerative changes. Both causes can be accompanied by neurologic and functional changes, which may relate to the acute or chronic nature of somatic dysfunction.[30]

Early evidence for a neurologic explanation of somatic dysfunction was provided in the late 1940s when J. Stedman Denslow, DO, and Irvin Korr, PhD, first introduced the spinal facilitation theory to explain the common findings of soft tissue changes, pain and tenderness, and muscular hypertonicity.[310,32] Denslow and Korr investigated neurophysiologic aspects of somatic dysfunction, especially the hyperarousal of the sympathetic nervous system, with their research findings later embedded in the concept of spinal facilitation.[31,32] In a series

of experimental studies in healthy individuals, Korr observed varying motor thresholds among participants, different patterns of sweating and skin conductivity, different vasomotor activity, some viscerosomatic changes, and different thermal patterns.[33(pp33-40,45-75,77-89)] Based on these studies, their model proposed that a constant afferent barrage by injured somatic or visceral structures would lead to segmental excitation, facilitating neuronal transmission and in turn producing excessive efferent response by the segment in question.[31,32]

Wilbur Cole, DO, added to this theory in 1952 by evaluating the effects of induced somatic dysfunction in animals and identifying histologic changes.[34,35] He hypothesized that stimulation of receptors in the striated muscles may lead to activation of the autonomic nervous system in the spinal cord, which in turn produces a predominantly parasympathetic-mediated muscle contraction and histologic changes in the viscera distant to the segmental spinal lesion by transmission of afferent impulses to the hypothalamus via the vagus.[34-36]

In 1976, Michael M. Patterson, PhD, suggested a possible mechanism for genesis and maintenance of spinal facilitation, assigning an active, vital role to the spinal neural pathways in the generation of somatic dysfunction.[37] He proposed that an afferent input from a skeletal or visceral organ could begin the sensitization of the neural pathways, resulting in increased output and resetting the excitability of the affected areas of the spinal cord. Thus, the control of higher centers in the sensitized areas would decrease and lead to impaired segments.[37] This process would lead to changes in skeletal and autonomic activity, as well as visceral function.[37]

Richard L. Van Buskirk, DO, PhD, introduced his own model of somatic dysfunction in 1990 based on the central role of nociceptors in the development of segmental somatic dysfunction. Van Buskirk proposed that pain-related sensory neurons and their reflexes cause motility restrictions and visceral, immunologic, and autonomic changes.[38]

Gary Fryer, PhD, BSc, has reviewed and updated this somatic dysfunction based on nociceptors concept theory over time, taking into account the newest literature on the topic.[30,39,40] His most recent model proposes that tissue injury leads to inflammation and activation of nociceptors, resulting in neurogenic tissue inflammation due to dorsal root reflexes.[30] This nociceptive drive may inhibit the activity of the deep segmental muscles while increasing the activation of superficial musculature, resulting in guarding activity of the musculature. Pain causes impairment of proprioception and motor control, leaving the segment more vulnerable to further injury.[30] However, Fryer[30] stresses that confounding factors for palpation of tissue tenderness and texture change could be consequences of central sensitization, such as hyperalgesia and allodynia, which occur as a result of increased excitability of neurons in the central nociceptive pathways.

Regarding signs of somatic dysfunction, tissue texture changes may be produced by soft tissue inflammation and guarding activities. Tenderness will most likely occur because of nociceptor activation and central sensitization processes, and change in range of motion would be the result of degenerative changes caused by sprain and inflammation.[30,40]

By providing an extensive evidence-based review of the literature, Paolo Tozzi, MsC Ost, DO, PT,[41] suggested a model that combined dysfunctional processes and manual therapeutic effects. Tozzi[41] proposed a change from the nociceptive model to a neuro-fasciogenic model by integrating neurologic processes into a multidimensional interpretation of the process of somatic dysfunction that may be mediated by fascia in some way.

Critical Voices

With an ever-expanding evidence base in modern neurobiology, an increasing number of publications have begun to challenge the spinal facilitated theory, both from within and outside the osteopathic medical profes-

sion.[30,39,40,42-48] Several authors stress that their criticism does not necessarily relate to the actual research that was performed by Korr and colleagues in the 1940s and 1950s, but rather to the interpretation of the research findings at the time the research was carried out.[30,47,48]

A 2016 review by Fryer[30] explored the plausibility of the concept and questions its relevance to the modern profession both within and outside the United States, taking into account its unclear pathophysiologic processes and poor reliability in detection of tissue manifestations. Fryer points out that the International Classification of Diseases may serve the interests of US-trained osteopathic physicians but has little relevance to foreign-trained osteopaths or to members outside the profession.[30] The author also suggests that the use of the term *somatic dysfunction* may have little clinical meaning for diagnostic purposes and for communication between foreign-trained osteopaths.[30] Furthermore, the use of this term in conversations with patients may be counterproductive because it may give an impression of a serious structural disorder.[30] Future research should address the significance of the terminology in daily practice and of the concept for the modern profession, including a meaningful language directed toward patients with regard to their physical findings and diagnosis.[30]

Conclusion

The founder and early developers of osteopathy used metaphors from mechanistic materialism to define the osteopathic lesion as a monocausal event—that is, with 1 source of dysfunction. Over time, the terms and definitions have evolved, leading to today's understanding of somatic dysfunction, which is multifaceted. With the introduction of evidence-based models, this concept has been updated, shaped, and challenged, with a current viewpoint on the nociceptor-based model. The most up-to-date evidence derived from bioscience and medicine has led to a multidimensional interpretation of somatic dysfunction, including the suggestion of a neurofasciogenic model, in which the role of the fascia in the development of its palpable features is taken into account. More collaborative research on somatic dysfunction is needed to add to the evidence base for today's practice of osteopathic medicine in the United States and osteopathy abroad.

References

1. Still AT. *Autobiography of Andrew T. Still With a History of the Discovery and Development of the Science of Osteopathy.* Rev ed. Kirksville, MO: published by the author; 1908.
2. Booth ER. *History of Osteopathy and Twentieth-Century Medical Practice.* Cincinnati, OH: Caxton Press; 1924.
3. Still AT. *Das große Still-Kompendium: Autobiografie, Philosophie der Osteopathie, Philosophie und mechanische Prinzipien der Osteopathie, Forschung und Praxis.* Jesse S, Clement B, Hamilton A, Saal L, trans-eds. 2nd ed. Pähl, Germany: Jolandos; 2005.
4. Schiller F. Spinal irritation and osteopathy. *Bull Hist Med.* 1971;45:250-266.
5. Swedenborg E. *The Animal Kingdom: Considered Anatomically, Physically and Philosophically.* Vol 1. Trans. J. J. G. Wilkinson; 1843. Reprint. London, England: Walton & Mitchell; 1960.
6. Stark J. *Stills Faszienkonzepte.* 2nd ed. Pähl, Germany: Jolandos; 2007.
7. Gevitz N. *The DO's: Osteopathic Medicine in America.* 2nd ed. Baltimore, MD: Johns Hopkins University Press; 2004.
8. Trowbridge C. *Andrew Taylor Still, 1828-1917.* Kirksville, MO: Thomas Jefferson University Press; 1991.
9. Fuller D. Hartman C, ed. *Osteopathie und Swedenborg: Swedenborgs Einfluss auf die Entstehung der Osteopathie, im Besonderen auf A.T. Still und W.G. Sutherland.* Pähl. Germany: Jolandos; 2015.
10. McCole GM. *An Analysis of the Osteopathic Lesion: A Study in Pathology, Physiology and Anatomy.* Great Falls, MT: published by the author; 1935.
11. Hazzard C. *Lectures on Principles of Osteopathy.* Kirksville, MO: Advocate Book and Job Print; 1898.
12. Still AT. *The Philosophy and Mechanical Principles of Osteopathy.* Kansas City, MO: Hudson-Kimberly Pub Co; 1902.
13. Still AT. The principles of osteopathy. *J Osteopath.* 1905;12:351-356.
14. Tasker DL. *Principles of Osteopathy.* 4th ed. Los Angeles, CA: Bireley & Elson; 1916.
15. Lane MA. *Dr. A.T. Still: Founder of Osteopathy.* Waukegan, IL: The Bunting Publications Inc; 1925.

16. Hulett GD. *A Textbook of the Principles of Osteopathy.* 4th ed, Kirksville, MO: Journal Printing Company; 1906.

17. McConnell CP. *Practice of Osteopathy, 1899.* Kirksville, MO: Journal Printing Company; 1906.

18. Castlio Y. *Principles of Osteopathy.* Kansas City, MO: Kansas City College of Osteopathy and Surgery; 1930.

19. Downing CH. *Principles and Practice of Osteopathy.* Kansas City, MO: Williams Publishing Co; 1923.

20. Littlejohn JM. *Principle of osteopathy. J Am Osteopath Assoc.* 1908;7(6).

21. Fryette HH. *Principles of Osteopathic Technique.* Carmel, CA: American Academy of Applied Osteopathy; 1954.

22. MacDonald G, Hargrave-Wilson W. *The Osteopathic Lesion.* London, UK: Heinemann: 1935.

23. Liem T, Dobler TK, Puylaert M, eds. *Leitfaden Viszerale Osteopathie* [in German]. 2nd ed. München, Germany: Elsevier Urban & Fischer; 2014.

24. Kales S. Experimentale forschung in der viszeralen osteopathischen medizin. In: Liem T, Dobler T, Puylaert M: *Leitfaden Viszerale Osteopathie.* 2nd ed. München: Elsevier Urban & Fischer; 2014.

25. Rumney IC. Recording musculoskeletal findings in hospitals. *J Am Osteopath Assoc.* 1969;68(7):732-734.

26. Rumney IC. The relevance of somatic dysfunction. *J Am Osteopath Assoc.* 1975;74(8):723-725.

27. Comeaux ZJ. Somatic dysfunction: a reflection on the scope of osteopathic practice. *Am Acad Ostepath J.* 2005;12:17-21.

28. Educational Council on Osteopathic Principles. *Glossary of Osteopathic Terminology.* Chevy Chase, MD: American Association of Colleges of Osteopathic Medicine; 2011. http://www.aacom.org/docs/default-source/insideome/got2011ed.pdf?sfvrsn=2. Accessed July 27, 2016.

29. DiGiovanna, EL. Somatic dysfunction. In: DiGiovanna EL, Schiowitz S, Dowling DJ, eds. *An Osteopathic Approach to Diagnosis and Treatment.* 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2005:16.

30. Fryer G. Somatic dysfunction: an osteopathic conundrum. *Int J Osteopath Med.* In press.

31. Denslow JS, Korr IM, Krems AD. Quantitative studies of chronic facilitation in human motoneuron pools. *Am J Physiol.* 1947;150(2):229-238.

32. Korr IM. The emerging concept of the osteopathic lesion. *J Am Osteopath Assoc.* 1948;48(3):127-138.

33. Peterson B, ed. *The Collected Papers of Irvin M. Korr.* Colorado Springs, CO; American Academy of Osteopathy; 1979.

34. Cole WV. The osteopathic lesion syndrome: X: the effects of an expiermental vertebral articular strain on the sensory unity. *J Am Osteopath Assoc.* 1952;51(8):381-387.

35. Cole W. The osteopathic lesion syndrome. In: *1952 AAO Yearbook.* Indianapolis, IN: American Academy of Osteopathy; 1952:149-178.

36. Cole WV. Experimental evidence. In: Hoag JM, Cole WV, Bradford SG. *Osteopathic Medicine.* New York, NY: McGraw-Hill Book Co; 1969:108-124.

37. Patterson MM. A model mechanism for spinal segmental facilitation. *J Am Osteopath Assoc.* 1976;76(1):62-72.

38. Van Buskirk RL. Nociceptive reflexes and the somatic dysfunction: a model. *J Am Osteopath Assoc.* 1990;90(9):792-805.

39. Fryer G. Somatic dysfunction: updating the concept. *Aust J Osteopath.* 1999;10(2):14-19.

40. Fryer G. Intervertebral dysfunction: a discussion of the manipulable spinal lesion. *J Osteopath Med.* 2003;6(2):64-73.

41. Tozzi P. A unifying neuro-fasciagenic model of somatic dysfunction: underlying mechanisms and treatment—part I [published online January 19, 2015]. *J Bodyw Mov Ther.* 2015;19(2):310-326. doi:10.1016/j.jbmt.2015.01.001.

42. Belanger AY, Morin S, Pepin P, Tremblay M-H, Vacho J. Manual muscle tapping decreases soleus H-reflex amplitude in control subjects. *Physiotherapy Canada.* 1989;41(4):192-196.

43. Kukulka CG, Beckman SM, Holte JB, Hoppenworth PK. Effects of intermittent tendon pressure on alpha motoneuron excitability. *Phys Ther.* 1986;66(7):1091-1094.

44. Leone JA, Kukulka CG. Effects of tendon pressure on alpha motoneuron excitability in patients with stroke. *Phys Ther.* 1988;68(4):475-480.

45. Newham DJ, Lederman E. Effect of manual therapy techniques on the stretch reflex in normal human quadriceps. *Disabil Rehab.* 1997;19(8):326-331.

46. Sullivan SJ, Williams LR, Seaborne DE, Morelli M. Effects of massage on alpha motoneuron excitability. *Phys Ther.* 1991;71(8):555-560.

47. Lederman E. Facilitated segments: a critical review. *Br Osteopath J.* 2000;22:7-10.

48. Janig W. Somatic dysfunction and the spinal segmental dysfunction. In: King HH, Janig W, Patterson MM, eds. *The Science and Clinical Application of Manual Therapy.* London, UK: Churchill Livingstone; 2011:277-282.