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Tradition Shapes the Future

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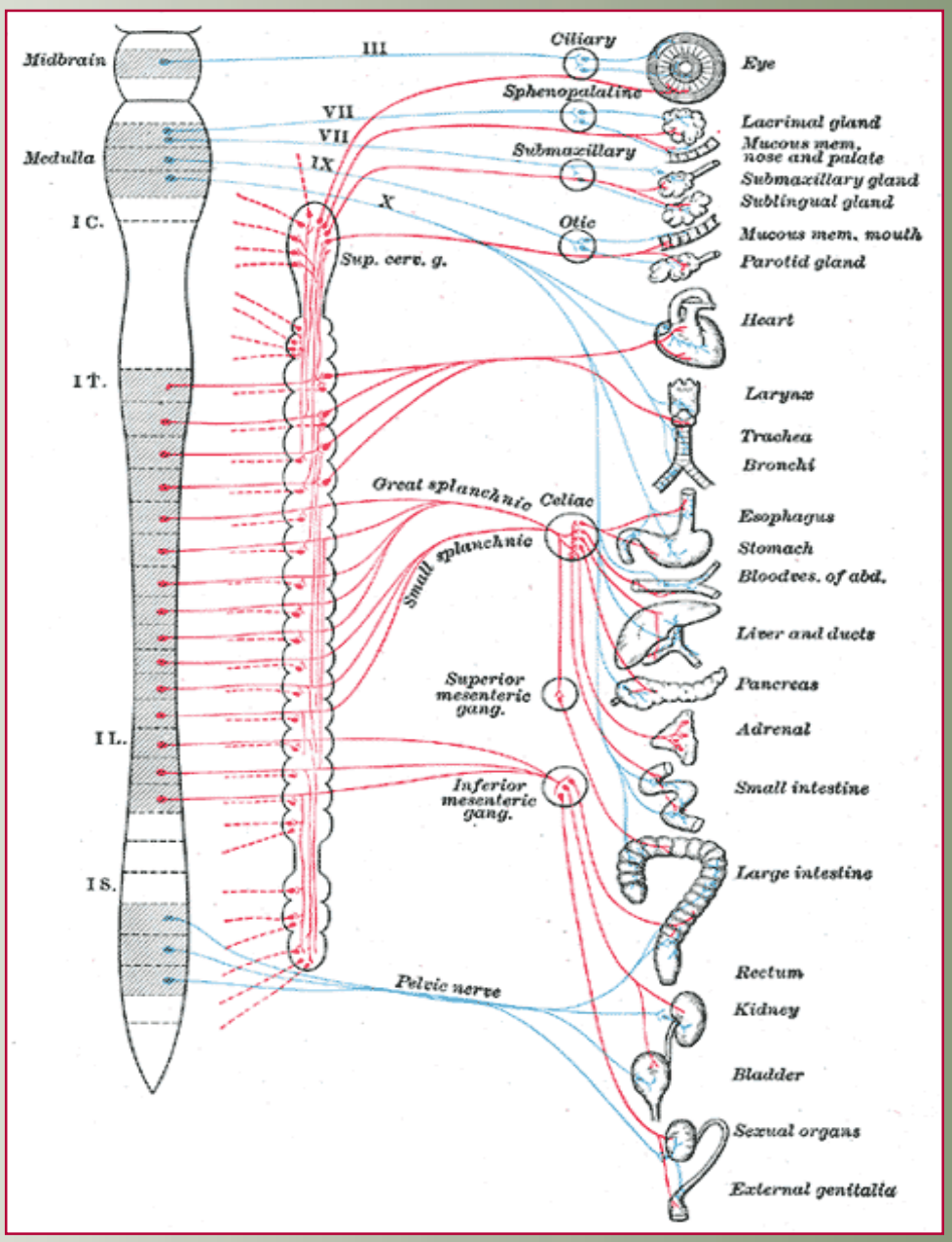

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Letter to the Editor

I have several thoughts about Dr. Hruby's September "View From the Pyramids" (New Treatment Guideline for Low Back Pain: Big Change or Big Noise?)

1. Modern medicine caught up with Dr. A. T. Still! It only took 115 years or so!
2. OMT for low back pain– This should have been the standard of osteopathic practice without interruption. We, of course, have a very good idea of the reality of this in the recent past and current practice of some (many) DO practitioners.
3. Implications – There should be nothing new, but our heritage guides us that OMT should be part of low back pain treatment (in most cases), and now professional guidelines exist to legitimize our heritages. Twenty years ago, I predicted some smart lawyer would make a good malpractice case based on professional heritage. Now guidelines give that lawyer even more ammunition! What a thought – give OMT or get a malpractice claim!
4. The algorithm is good. As I read it, I kept thinking "that is what they told us in OMT classes at KCOM nearly 40 years ago!"

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View From the Pyramids

Integrating Osteopathic Principle into Daily Practice

Raymond J. Hruby, DO, FAAO

Osteopathic medical students frequently ask, "How exactly *do* we integrate the principles of osteopathic medicine into everyday practice?" If we are to ensure that every patient we see is treated in a truly osteopathic fashion, what would these patients experience that is different from any other kind of approach?

In order to answer these questions we should first remind ourselves of the basic principles of osteopathic medicine:

1. The body is a unit.
2. The body has self-regulating and self-healing capabilities.
3. Structure and function are interrelated.
4. Rational treatment is based on the intelligent application of the above three principles.

These principles tell us that the body functions as an integrated whole, and no one body system can function independently of the others. If all body systems are operating at their optimum levels, the body has its best chance for self-healing and maintenance of health. Osteopathic medicine places great emphasis on the relationship between structure and function. If all of the structural components of the body are in proper anatomic relationship, then the body is at its most energy efficient level, and all body systems function at their optimum levels. The osteopathic physician has the unique capability to diagnose by palpation these abnormal structural relationships (called somatic dysfunctions), and is able to use osteopathic manipulative treatment (OMT) to reduce or eliminate these somatic dysfunctions. Collectively, these principles provide the rationale for the osteopathic approach to patient care.

There are those osteopathic physicians who believe, because OMT is utilized on a given patient, osteopathic principles have been served. I believe the use of OMT is only a part of the application of osteopathic principles in patient care. If the use of OMT were the only thing that made the osteopathic physician unique, then the solution to the difference between the osteopathic approach and any other approach would be simple: train the other professions in the use of OMT and we would all be the same! No, the full application of osteopathic principles to the patient is much more than this. The use of OMT is only the culmination of a different philosophically principled approach used by osteopathic physicians to solve patient problems.

Perhaps an example will illustrate the application of these principles. Consider the case of the twenty-year-old male, otherwise healthy, who arrives at his physician's office with a fever, sore throat, difficulty swallowing, and swollen lymph glands in his neck. After taking a focused history, performing a focused physical examination and an in-office strep test, the physician determines the patient is suffering from streptococcal pharyngitis.

This is not an uncommon problem in the daily practice of medicine. A standard approach at this point would be to prescribe an appropriate antibiotic (typically penicillin), advise the patient to rest, drink fluids, take aspirin or acetaminophen, and refrain from work for a short period of time. This will usually take care of the pharyngitis and help the patient avoid more serious complications.

But the osteopathic physician thinks differently, and has other questions to ask. He or she knows, as does any other physician, *how* the patient is sick—the patient has streptococcal pharyngitis. But the osteopathic physician also asks *why* the patient is sick. Why has this otherwise healthy person become ill rather than resist this infection? What can be done to facilitate his self-healing and self-regulatory mechanisms in overcoming and further avoiding this problem?

In this case, the osteopathic physician's physical examination of the patient shows he has somatic dysfunctions in the cervical and upper thoracic spinal area, and the upper rib cage. The nerves that innervate the head and neck area arise from these spinal segments. In addition, these somatic dysfunctions inhibit the blood

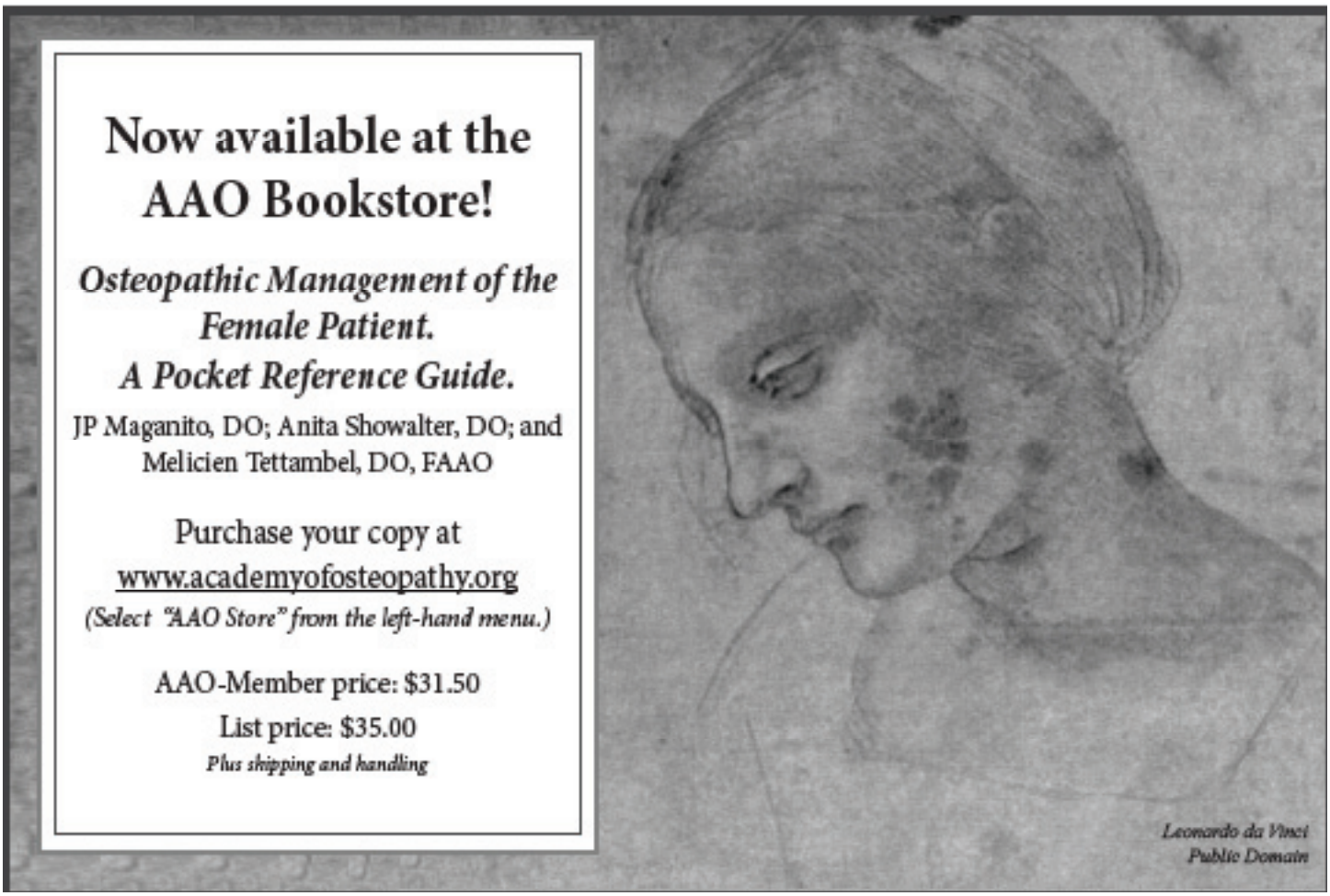
flow to and from the throat area, and impede the lymphatic drainage from this area. The osteopathic physician also discovers that the patient has been under more stress at work recently, and has had to work longer hours than normal.

This information provides for a much different and more complete approach to the problem. In addition to prescribing antibiotics, fluids, aspirin or acetaminophen, and rest, the osteopathically oriented physician now uses OMT to relieve the somatic dysfunctions in the previously noted body areas. This in itself enhances the body's self-healing mechanisms by stimulating the immune system, and providing for better blood and lymphatic flow to the affected area. The osteopathic physician also points out to the patient that his increased stress and workload has likely caused his resistance to become diminished, making him susceptible to illness. This can be avoided by finding ways to alleviate stress, and exerting more control over his work schedule.

Thus, the osteopathically oriented physician, given the same diagnosis any physician would make, has approached the problem in an entirely different way. He or she has considered the whole patient by way of determining the circumstances that may have caused the patient to become susceptible to the infection in the first place. The patient's self-regulatory and self-healing mechanisms have been maximized by the use of rest, fluids and OMT. The disturbed structure-function interrelationships that contribute to this adverse situation have been addressed. The patient has more awareness of what caused him to become ill, and how he can avoid the same situation in the future. The rationale for this approach comes from the ability to apply *all* osteopathic principles in the care of the patient.

The integration of osteopathic principles into patient care requires more than just the application of a few manipulative techniques. It is an approach to patient problem solving that starts with a unique set of principles, and shapes the osteopathically oriented physician's thinking processes much differently than those of other practitioners.

These are my thoughts on the integration of osteopathic principles into the daily practice of medicine. *What are yours?*



On the Role of the *Glossary*

Murray R. Berkowitz, DO, MA, MS, MPH

I have observed and read about the various arguments and discussions among a number of my esteemed colleagues and personal friends regarding the meaning of various terms used commonly within our profession. These terms include, but are not limited to, "osteopathic physician," "osteopath," "osteopathic medicine," and "osteopathy." While there is more than adequate documentation that these terms were defined long ago, there has been recent debate regarding when these definitions have effect. One of my aforementioned esteemed colleagues and personal friends stated that the terms and definitions in the *Glossary* (i.e., the *Glossary of Osteopathic Terminology*[1]) are to be used in written and oral scientific discourse, but not in discussing policy. This came about because another one of my aforementioned esteemed colleagues and personal friends had earlier held up the *Glossary* as he was trying to make a point at the House of Delegates meeting of the American Osteopathic Association. An earlier, ongoing and contentious issue stems from the fact that there is no official requirement that manuscripts submitted to, and accepted for publication in, the *Journal of the American Osteopathic Association* (JAOA) be consistent with, and conform to, the *Glossary*. Hence, the overarching question comes down to the role of the *Glossary* and the terminology defined therein.

According to Webster's Unabridged Dictionary[2], "glossary" is defined as "a collection of textual glosses or of specialized terms with their meanings;" according to the Oxford English Dictionary,[3] "glossary" is defined as "an alphabetical list of defined terms in a specialized field, such as medicine or science." I think we can all see, and hopefully agree, that the *Glossary of Osteopathic Terminology* is, or at least purports to be, an alphabetical list of defined terms within the osteopathic profession. It includes not only terms that are purely unique to the profession, but also includes how (at least some) terms found in more common, everyday vocabulary are used within our profession. We have still not fully approached the overarching question posed earlier. We still need to look at when and how to apply the contents of the *Glossary*.

I feel we must be diligent in the application of terminology. It adds both precision and accuracy to our discourse. Allow me to point out an unintended consequence of failing to consistently apply our terminology. Both "Osteopathic Manipulative Treatment" (OMT) and "Osteopathic Manipulative Therapy" (OMTh) are listed in the *Glossary*. I have both seen and heard the indiscriminate use of these terms interchangeably. As physicians, we prescribe "treatment;" mid-level practitioners provide "therapy." If we permit the interchangeable use of "therapy" instead of insisting upon "treatment" in OMT, then the various third-party payers can get away with discounting what we do in giving OMT to our patients as providing therapy—thus, the continued attempts by third-party payers to bundle and down-code OMT as if it were performed by physical therapists. This impacts our "bottom line" by decreasing our reimbursements. We do not want, and should not accept, this unintended consequence.

I feel the role of the *Glossary* is to define our osteopathic profession's terminology and phraseology, and needs to be diligently applied in all our discourse, scientific and policy-related, written and oral. This especially needs to be rigorously maintained by the premier journals and writings within our profession. Paraphrasing the philosopher Will Durant, "A great profession is not destroyed from without until it has destroyed itself from within."[4]

References

1 *Glossary of Osteopathic Terminology*. Chevy Chase, MD: American Association of Colleges of Osteopathic Medicine. 2009.

2 *Webster's Unabridged Dictionary.* 2nd Ed. New York: Random House. 2005.

3 *The Oxford English Dictionary*. 2nd Ed. Oxford, UK: Oxford University Press. 2009.

4 Durant W. *The Story of Civilization*. Vol. 3. New York: Simon and Schuster. 1944. 665.

An Osteopathic Approach to Hypothyroidism

Denise K. Burns, DO, FAAO

Introduction

Osteopathic physicians since the late 1800s have utilized osteopathic manipulation in the treatment of thyroid disorders. This paper will discuss hypothyroidism in totality and routes of manual treatment. Anecdotal case histories of effective treatment of thyroid conditions utilizing osteopathic manipulation have been documented in osteopathic literature. In the book *Osteopathy Research and Practice*, Dr. A.T. Still presents his experiences in the osteopathic diagnosis and treatment of disease conditions. In his explanation of the etiology and treatment of thyroiditis, Dr. Still relates the malposition of the clavicles, scapula, sternum and that of the first and second ribs as a possible cause for the backup of blood or other fluids within the thyroid gland, resulting in inflammation and thyroiditis. He states that the loss of the normal anatomic relationship between these structures interferes with the proper function of nerves, lymphatics and blood vessels, ultimately leading to disease. In addition, malfunction of the structures that drain the organs from noxious elements hinder the natural healing processes of the body. It follows that the removal of these structural obstructions would allow the return of optimal performance of the thyroid gland. Dr. Still further explained that osteopathic treatment should be geared toward enhancing the drainage of the venous system and of the flow of the arterial supply in order for the inflammation to subside and obtain normal function. Several cases of the treatment of thyroid goiter were documented by Dr. Thomas Ray in his article Osteopathic Treatment of Goiter.[1] The most common structural abnormalities found by Dr. Ray were related to thyroid goiter; at the cervical spine, upper thoracic, upper ribs, clavicles and associated muscular attachments. Dr. Ray reported that thyroid goiters and associated symptoms could be cured with appropriate osteopathic manipulative treatment and that this often resulted in permanent resolution. Further, that in his forty plus years of experience treating goiter, he most often found that the primary cause of goiter was structural obstacles affecting the function of the thyroid gland. Dr. Howard Lamb states that nodular goiter commonly causes pressure symptoms on the trachea or other neighboring structures.[2] He states that intelligently applied osteopathic manipulative therapy has a beneficial effect on the sympathetic nervous system and affects the course of disease. Dr. Robert Clark postulated that structural abnormalities might indeed be a direct or indirect cause of hypothyroidism.[3] These structural dysfunctions could be in the immediate vicinity of the thyroid gland or at a remote location. He reported improvement in his patients' symptomatology with osteopathic manipulation alone or in conjunction with thyroid extract. Dr. Clark assumed that the irreversible structural lesion pathology in those patients required a permanent maintenance dose of thyroid extract. Some patients recovered completely. He recommended that osteopathic manipulative treatment include correction of dysfunctions from the occiput to the feet to improve thyroid function. Moreover, bony, soft tissue and visceral lesions must be addressed. Dr. Clark found that the need for thyroid extract was diminished with osteopathic manipulative treatment. This was echoed by Dr. Cotrille in his article on Detection and Management of Hypothyroidism.[4] Dr. Cotrille suggests that manipulative treatment for hypothyroidism should also include reduction of restrictions of connective and soft tissue in order to optimize systemic response to the thyroid. It also helps prepare the tissues for proper nutrition. While many anecdotal accounts of manipulative treatment for thyroid disorders have been published, a literature search revealed little information on the effects of manipulative treatments on the clinical syndrome of hypothyroidism. Consideration of structural and functional features of the thyroid may suggest potential routes through which osteopathic manipulative treatment could influence glandular function.

Background

Thyroid disorders are common in clinical practice, second in frequency to diabetes mellitus among endocrine illnesses. Thyroid disorders affect millions of Americans annually. Autoimmune disorders of the thyroid gland can stimulate the overproduction of thyroid hormone (thyrotoxicosis) or cause glandular destruction and hormone deficiency (hypothyroidism). Hypothyroidism is the clinical syndrome that results from decreased secretion of thyroid hormone from the thyroid gland. Most cases are seen in adulthood. The incidence of hypothyroidism varies among geographic areas. Primary hypothyroidism accounts for > 95% of all cases, the remainder being of pituitary or hypothalamic origin, which in turn results in marked slowing down of metabolic processes. The most common cause of hypothyroidism is Hashimoto's Thyroiditis. In areas of adequate iodine supply, like the United States, hypothyroidism occurs in .8-1% of the population.[5] In iodine-deficient areas, the incidence is 10-20 fold higher.[5] The incidence of hypothyroidism increases with age. Common symptoms of hypothyroidism include slow thinking, lethargy, fatigue, dry, cool skin, thickened hair, hair loss, muscle stiffness, myalgias, paresthesias, constipation, hypothermia, menorrhagia,

diminished libido and weight gain.[6] Common signs of hypothyroidism include round, puffy face, slowed speech, hoarseness, hypokinesia, generalized muscle weakness, delayed relaxation of deep tendon reflexes, depression, and mental clouding.[7] Researchers found that if myopathy and delayed relaxation of deep tendon jerks are present, then biochemically severe hypothyroidism was likely (thyroxine levels < 20 nmol/l).[8] Dyslipidemia, cardiovascular compromise and pulmonary compromise can result. Fluid retention due to secondary antidiuretic hormone excess may occur with serous effusions and edema. Constipation is a common complaint and is caused by slowed peristalsis. Dermatologic findings may include carotnemia and myxedema (a characteristic diffuse, non-pitting puffiness of the skin).[9] Deposition of glycosaminoglycans (mostly hyaluronic acid and mucin) in intracellular spaces, particularly in skin, heart muscle and striated muscle, produce the clinical picture of myxedema. Longstanding severe, untreated hypothyroidism may lead to a state called myxedema coma. Diffuse thyroid enlargement or goiter is usually present. It results from prolonged stimulation of the thyroid gland by thyroid stimulating hormone (TSH). There is progressive iodine turnover in the gland with cellular hyperplasia, lymphocyte infiltration, necrosis, hemorrhage, and nodule formation. Fibrosis can result in the late stages of the disease. Goiter can compress the trachea, esophagus and recurrent laryngeal nerves.[10] Current accepted medical treatment standards for primary hypothyroidism are primarily limited to pharmacologic management. However, the anatomical location of the thyroid gland is readily assessable to the osteopathic physician, and can be manipulated osteopathically to improve its function. The effects of hypothyroidism on other targeted organ systems of the body can also be treated with manual manipulation.

Embryology

The thyroid gland develops from the floor of the primitive pharynx during the third week of gestation. The embryonic pharynx is located at the foramen cecum in the dorsum of the tongue.[10] The gland migrates from the base of the tongue into the neck along the thyroglossal duct passing anterior to the hyoid bone and thyroid cartilages to reach its final position anterolateral to the superior part of the trachea.[10] Thyroid hormone synthesis normally begins at 11 weeks gestation. Thyroid gland development is coordinated by the expression of several developmental transcription factors. In combination, they dictate thyroid cell development and induction of thyroid specific genes and proteins such as thyroglobulin, thyroid peroxidase, the sodium iodide symporter and thyroid stimulating hormone receptors.[11]

Musculature and Connective Tissues

The thyroid gland is the body's largest endocrine gland with a rich blood supply and drainage.[10] It weighs approximately 20-25 gms in the normal state.[12] It produces thyroid hormone, which controls the rate of metabolism and calcitonin—a hormone controlling calcium metabolism. Its Greek root implies it is shield shaped, although its form varies from an H-shaped to a U-shaped gland. The thyroid gland is located in the neck and lies deep to the sternothyroid and sternohyoid muscles from the level of C5 through T1 vertebrae. It consists of two lobes, right and left, anterolateral to the larynx and trachea. An isthmus unites the lobes over the trachea, usually anterior to the second and third tracheal rings. In 50% of the cases, there is a pyramidal lobe, which ascends from the isthmus upward, sometimes as high as the hyoid bone.[13] This lobe represents a remnant of distal thyroglossal duct tissue. Muscular attachments pertaining to the thyroid cartilage are the sternothyroid, cricothyroid, thyrohyoid and stylopharyngeous muscles. The *sternothyroid muscle* is innervated by the ansa cervicalis, derived from the hypoglossal nerve and C1 through C3. It attaches to the posterior manubrium and the thyroid cartilage. It depresses the larynx after elevation from swallowing. The *cricothyroid muscle* runs obliquely from the anterior lateral cricoid cartilage to the inferior aspect of the thyroid cartilage. Its innervation comes from the external branch of the superior laryngeal nerve via vagus nerve. It tenses the vocal cords. The *thyrohyoid muscle* is attached to an oblique line of the lamina of the thyroid cartilage to the inferior border body and greater horn of the hyoid bone. It is innervated by the hypoglossal nerve (CN12) and depresses the hyoid bone. The *stylopharyngeous muscle* arises from the styloid process of the temporal bone and attaches to the posterior and superior margin of the thyroid cartilage. It elevates the pharynx and larynx during swallowing and speaking. It is innervated by the glossopharyngeal nerve (CN 9). The thyroid gland is surrounded by a thickened fibrous capsule, which sends septa deeply into the gland. External to the capsule is a loose sheath formed by the visceral layer of the pretracheal deep cervical fascia. There are several layers of fascia in the neck. The superficial fascia is usually a thin layer of subcutaneous connective tissue that lies between the dermis and the deep cervical fascia. The deep cervical fascia consists of three fascial layers—investing, pretracheal (visceral) and prevertebral. These layers support the thyroid gland. This fascia attaches at the foramen magnum and the pterygoid processes of the sphenoid bone. Dense connective tissue (Berry's ligament) attaches the capsule of the thyroid gland to the cricoid cartilage and the superior tracheal rings.[10] Ligamentous attachments link the thyroid gland to the thyroid and cricoid cartilages and the first and

second tracheal rings. The *stylohyoid ligament* is firmly bound to the thyroid cartilage.

Histology

Microscopically, the thyroid gland consists of spherical follicles. The follicles consist of a single layer of epithelial cells surrounding a lumen filled with a proteinaceous thyroid colloid consisting mostly of thyroglobulin (an iodine containing glycoprotein, which is a precursor to active hormone).[14] Thyroglobulin is formed by the follicular epithelial cells, which synthesize and store hormone. The hormone and thyroglobulin are stored within thyroid colloid. Thyroid follicular cells are polarized.[15] Follicular cells are surrounded by capillaries and stroma. The basolateral surface is apposed to the bloodstream, and an apical surface faces the follicular lumen. The second type of cell found in the thyroid gland is the C-cell. These cells contain and secrete calcitonin, and are seen throughout the gland.

Physiology

The thyroid gland produces two hormones—thyroxine (T4) and triioddothyronine (T3). The hormonal regulation of the thyroid gland begins with the hypothalamus. Thyroid hormone is derived from iodination and the linkage of two residues of the amino acid tyrosine on specific thyroid proteins called thyroglobulin. The tyrosine residues are part of the primary structure of thyroglobulin. The synthesis of hormone precursor occurs on these residues, and they remain at all times part of the structure of thyroglobulin. TSH increases synthesis of thyroglobulin via cyclic AMP.[16] The hypothalamus releases thyroid releasing hormone (TRH). The pituitary gland then responds to this by releasing TSH. Subsequently, TSH is released into the systemic circulation and binds to receptors on follicular cells at its basolateral surface of the thyroid gland. This leads to thyroglobulin reabsorption from the follicular lumen, and stimulates the follicular cells to produce the amine derived hormones, thyroxine (T4) and triioddothyronine (T3). Activation of TSH receptors stimulates growth and vascularity of the thyroid gland, as well as modulates multiple aspects of thyroid gland metabolism and function related to hormone production. This includes stimulating proliferation of the follicular cells themselves and increasing their uptake of iodine; the rate of synthesis of thyroid hormone precursor and its storage as colloid; and the rate of breakdown of stored colloid with release of T3 and T4. The production of thyroid hormone is negatively regulated in a feedback mechanism. This regulation and feedback mechanism is referred to as the Hypothalamus-Pituitary-Thyroid axis.[17] Eventually, secretion of TRH and TSH is suppressed directly by T4 and T3 through this negative feedback loop. Physiologically, it is the free (unbound) hormone in plasma that are active and inhibit pituitary secretion of TSH.[14] Excess iodine exerts an inhibitory effect as well. The metabolic status of follicular cells directly affects hormone release.

Iodine is an essential trace element required to produce thyroid hormone. Approximately .2 mgs of iodine are needed for the daily production of thyroid hormone.[4] The thyroid gland has the ability to metabolize iodine and incorporate it into organic compounds. The thyroid gland concentrates iodide via an electrochemical gradient by a carrier mediated mechanism driven by ATP.[16] Iodine uptake from the plasma is a critical first step in thyroid hormone synthesis.[18] Dietary iodine is absorbed in the gastrointestinal tract (GI). Iodine is converted to iodide in the GI tract before absorption. Iodide is removed from the bloodstream by uptake and concentration in the thyroid gland. Excretion of iodide is via the urine. Uptake into the thyroid gland is performed by a Na/I symporter (iodide pump), which transports plasma iodide anion (I^-) into the apical part of the follicular cells and elevates its concentration there. The iodide pump is located on the basolateral part of the follicular cell next to the extracellular space and the vasculature.[16] The efficacy of the pump is characterized by the thyroid (follicular) to serum iodide (T/S) concentration ratio.[19] The T/S ratio is regulated by TSH. Higher TSH levels cause higher T/S ratios.[12] Once inside the follicular cells, iodide is oxidized to iodine. The oxidation is catalyzed by thyroid peroxidase (TPO), a multi-enzyme complex, and is driven by hydrogen peroxide. The thyroid peroxidase complex is membrane bound and located on the apical (colloid-facing) aspect of thyroid follicular cells.[18] Once oxidized, the iodine can be incorporated into hormone precursors within the colloid. Oxidized iodine then reacts with tyrosine residues. The iodination and coupling reactions occur in the vicinity of the apical membrane. The first reaction is the addition of a single oxidized iodine to the ring of a tyrosine residue, forming the compound monoiodotyrosine (MIT). In some cases, a second iodine is added to the ring to form diiodotyrosine (DIT). Subsequently, a set of TSH rate dependent "coupling reactions" occurs. MIT and DIT residues are brought together to form the precursor of thyroid hormone T3. In other cases, two DITs are reacted together to form the bound precursor of T4. The fully iodinated and reacted thyroglobulin is secreted into the colloidal space of the thyroid follicles, where it constitutes the gel-like colloid. This can be stored for months before being broken down to release hormone. Approximately 10 times as much T4 precursor as T3 precursor is present within the colloid. The thyroid secretes about 103 nmol (80 ug) of T4 and 7 nmol (4 ug) of T3 per day.[14] Normal serum T4 level is approximately 8 ug/dl (103 nmol/L)

and T3 is 0.15 ug/dl (2.3 nmol). Elevated TRH and TSH levels signal release of thyroid hormone. Upon stimulation, the colloid from the thyroid follicles is taken up by endocytosis. The resulting membrane-bound vesicles subsequently fuse with lysosomes to form endosomes. Lysosomal enzymes hydrolyze thyroglobulin into amino acids, free T3, and T4, MIT, and DIT. The latter two are not released; their iodine is enzymatically cleaved and recycled. T3 and T4 are released into the circulation. The mechanism by which the hormone leaves the cell is thought to be diffusion, but this is not known for certain. Most of the released thyroid hormones are bound to carrier proteins, and only about 0.02% is the free physiologically active fraction. The postulated physiologic role of plasma proteins is to provide a circulating reservoir of thyroid hormone and a buffer against drastic and abrupt changes in hormone concentration. The three major thyroid hormone binding proteins are thyroid binding globulin (TBG), transthyretin (found in the cerebrospinal fluid that binds T4) and albumin. T3 is the more potent of the two thyroid hormones; it is less tightly bound to protein and enters the peripheral tissues more readily. Most (about three quarters) of the T3 is produced peripherally by conversion from T4 (87%).[12] T4 is only produced within the thyroid gland. Thyroid hormone enters the peripheral cells via passive diffusion or specific transport through the cell membrane to the cytoplasm.[14] Within the cytoplasm T4 is converted to T3. A single iodine atom is enzymatically cleaved from T4 in peripheral tissues via deiodinases to glucuronides. T3 attaches to its receptors on the cell nucleus of the brain, gonads, liver, heart, muscles and kidneys. The nuclear receptors are hormone sensitive, and either stimulate or suppress genes via transcription factors. The cell nuclear receptor for T3 is similar to the nuclear receptors for steroids, vitamin D and retinoic acid. Thyroid hormone receptors recognize specific gene sequences and cause a conformational change in the receptors that modify its interactions with accessory gene transcription factors.[11] These hormones regulate central nervous system developmental processes, cell differentiation, morphogenesis, growth, metabolic rate, body temperature and myocardial contractility and carbohydrate absorption. Anabolism and catabolism rates affect fat, carbohydrate and protein levels in the body. Specifically, T3 stimulated genes were involved in gluconeogenesis, glycogenolysis, lipogenesis, cell proliferation and apotosis.[15] Genes repressed by T3 affected cell immunity, insulin signal transduction, cell architecture and mitochondrial function.[18] Energy for muscle contraction relies on mitochondrial production of adenosine triphosphate (ATP). The production of ATP increases when T3 concentrations increase. In hypothyroidism, mitochondrial enzymes are reduced, which can cause muscle weakness and fatigue.[20]

Circulation

Blood flow through the gland is very high (approximately 5 ml/g/min), exceeding that of the kidney.[19] The principal arterial blood supply to the thyroid gland comes from the paired superior and inferior thyroid arteries. Small branches can sometimes be found from laryngeal and tracheoesophageal arteries. These vessels lie between the fibrous capsule and the pretracheal layer of deep cervical fascia. Usually the first branch of the external carotid, the superior thyroid artery descends to the superior pole of each lobe of the gland, pierces the pretracheal layer of deep cervical fascia and divides into anterior and posterior branches. The superior thyroid artery runs adjacent to the omohyoid and sternohyoid muscles. The main branch runs over the anterior surface of the superior pole of the gland with other small branches entering the gland posteriorly.[10] The inferior thyroid artery arises from the thyrocervical trunk of Subclavian artery, and arches medially behind carotid artery toward posterior part of the thyroid gland.[10] It has several branches that pierce the pretracheal fascia, and it supplies the inferior thyroid gland.[10] The inferior thyroid artery is intimately related to the recurrent laryngeal nerve in most cases. The venous drainage of the thyroid gland is formed by three pairs of thyroid veins, which usually drain the venous plexus on the anterior surface of the thyroid gland. The superior thyroid veins drain the superior

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poles of the thyroid gland, the middle thyroid veins drain the middle lobes, and the inferior thyroid veins drain the inferior poles and run anterior to trachea. The superior and middle veins drain into the internal jugular vein, and the inferior thyroid veins drain into the brachiocephalic veins posterior to the manubrium of the sternum.[10] Venous variants have occurred.

Lymphatic drainage from the head and neck is primarily via the lymphatic ducts bilaterally to the Subclavian veins. These ducts and great veins run underneath the clavicles, making them important to drainage. Lymphatic vessels of the thyroid gland run in the interlobular connective tissue, often around the arteries, and communicate with a capsular network of lymphatic vessels. From here, the vessels pass to prelaryngeal, pretracheal (infra and supraisthmic), and paratracheal lymph nodes. Laterally, lymphatic vessels located along the superior thyroid veins pass to the inferior deep cervical lymph nodes. Some lymphatic vessels may drain into the brachiocephalic lymph nodes or into the thoracic duct.

Nerve Innervation

Dense capillary networks of sympathetic and parasympathetic nerves surround the follicles.[5] Sympathetic innervation to the thyroid gland originates from the spinal segments of T1-T4. These presynaptic fibers synapse in the superior, middle and inferior ganglia of the sympathetic cervical chain (located at the level of C2, C6 transverse process and anterior to C7). From these ganglia, post synaptic fibers pass via gray rami to cervical spinal nerves, or leave as direct visceral branches from splanchnic nerves. They reach the gland through the superior and inferior thyroid periarterial plexuses that accompany the thyroid arteries.[10] Autonomic innervation regulates hormonal synthesis, secretion and blood flow.[19] The specific role of the autonomic nervous system in relation to glandular secretion is not clearly understood, but it is postulated that most of the effect is on blood vessels and perfusion rates of the glands. Sympathetic fibers are vasomotor and cause vasoconstriction of blood vessels.[10] Parasympathetic control of the thyroid gland is via the vagus nerve, whose fibers reach the thyroid gland through the superior and inferior laryngeal nerves. Dr. Unverferth stated that stimulation of the sympathetics causes increased secretion of the part of the glandular structures of the thyroid, and parasympathetic stimulation likely opposes the action.[21] The recurrent laryngeal nerves traverse the lateral borders of the thyroid gland, and must be identified during thyroid surgery to prevent vocal cord paralysis.

Osteopathic Manipulative Treatment

Structure and function are not only interrelated, but are inseparably linked at all levels of the biologic spectrum. A patient's thyroid problem is unresolved until structure and function of all organs involved have been repaired to the extent that the thyroxin-elaborating function is restored.[22] Musculoskeletal manifestations are prevalent in the hypothyroid patient.[23] Directed osteopathic treatment will increase the thyroid's capacity to metabolize the iodine present.[22] By alleviating musculoskeletal restrictions, enhancement of self-regulatory and self-healing mechanisms can be fostered. The vasculature and the nervous system influence the thyroid gland. The hypothalamic-pituitary-thyroid axis will function more efficiently when myofascial, neurologic, endocrine, lymphatic and articular structures anatomically related to the thyroid gland act harmoniously. The thyroid gland's anatomic position is such that it is enveloped in cervical fascias and muscular structures. A functional vulnerability exists if musculoskeletal harmony and visceral function are not optimal, especially in the cranial, cervical, upper limbs and thoracic regions of the body. Vertebral and peripheral joints and myofascial restrictions can increase soft tissue tension on and around the gland and its related structures. Abnormal tensions associated with chronic fluid stasis, myofascial and dural pulls, and abnormal extracellular protein deposition likely create macro and micro insults to the gland. Cellular shape and structure are altered. These insults and injury can cause cellular damage, circulatory compromise, edematous changes, inflammatory mediator infiltration in the tissues (due to tissue injury) and waste product buildup.[14] Arterial, venous and lymphatic compromise can foster an internal glandular environment deprived of oxygen and thyroid hormone precursors, such as tyrosine and iodine. These cause excessive buildup of cellular waste products, which can have negative micro and macro cellular consequences. Cellular imbalance occurs with ionic gradient shifts and impaired membrane transport mechanisms. Follicular cells are polarized normally and are vulnerable to these subtle changes. The bioelectric capacity of the intracellular and extracellular environment will be impeded. Important cellular processes involved in hormone production, membrane transport dysfunction for the ATPase driven iodide anion, may be impacted negatively. With chronicity, facilitation of body tissues occur. Sympathicotonia can cause vascular vasoconstriction and poor blood and lymph flow. Hypothalamic-pituitary dysfunction from local and central facilitation could hasten the disease state and possibly cause further endocrine organ dysfunction. Cellular ischemic changes, and even cellular death, can result. In cases where the disease has been prolonged and thyroid gland has been inflamed and edematous due to chronic infectious or autoimmune insult (thyroiditis), osteopathic manipulation may be able to prevent irreversible damage to the gland, such as fibrosis and atrophy.

Osteopathic manipulation would likely restore, in part or totally, glandular structure, perfusion, and function. Treatment described by A.T. Still focused on increasing fluid flow and nerve conduction. Dr. Still treated many goiters and their related exophalamos. As Dr. Still stated, "Goiter is the effect of failure of the arterial blood to reach the brain and return normally". Pressure on the neck structures including the thyroid gland is the result of manubrium, clavicles, ribs or upper dorsal vertebrae having lost their normal position by violence, he said. "A strangulation of venous blood of the brain and neck follows such variations from normal articulations of the bones. They press upon all of the blood vessels that go through the neck to the brain and produce enlargement of the muscles and glands of the neck, head and face." Dr. Ray also found that goiter was a result of prolonged congestion. Still suggests in his treatment of the gland to place one's fingers so they pass over, back and under the enlargement, and gently lift it superior and anterior with a small squeezing motion. Common sites of thyroid induced somatic dysfunction in the literature include the hyoid bone, Occipito-Atlantal joint, maxillae bones, C2 vertebral segment, cervico-thoracic junction, ribs 1-2, T1-T8 vertebral segments (specifically T2) and the sacroiliac joints. C2 has been consistently reported to be rotated left as a result of abnormal vagal tone.[24] Cotrille discusses correction of abnormalities found at reflex centers just below the junction of the third rib and the sternum, the condylar portion of the occiput and the greater wings of the sphenoid.[4] Dr. Still, in his book *Osteopathy Research and Practice*, states that a very common cause of goiter is the "slipping of the first rib off, back and under the transverse process of the upper dorsal vertebrae. Routledge, DO, treated the hypothyroid patient with mobilization of the cervical, upper dorsal and lumbar regions."[25] He found that the lesioned area seemed to be more a group lesion versus any specific lesion. Other musculoskeletal manifestations reported in the literature for thyroid disease include hypersympathetic responses associated with generalized increased muscle tone. This is postulated to be the result of cyclic enhancement of segmental somatic irritation by the viscus, which occurs with subsequent increased sympathetic response upon the viscera.[24] Hypoesthesia of C2, C3 and C4 dermatomes unilaterally has been expressed with related trigger point presence in the Sternocleidomastoid (C2-C3), Trapezius (CN 12, C3-C4) and Splenius Capitus (C4, C5) muscles of hypothyroid patients.[26] Patients with low thyroid function are more susceptible to myofascial trigger points.[20] Cervical muscles that have direct attachments to the thyroid capsule receive their motor innervation from the upper cervical spinal levels. Trigger points and other muscular tissue texture changes associated with thyroid disease are the likely result of an initial viscerosomatic response initiated by the thyroid gland. With facilitation and further neurologic imbalances, one or more somato-somatic reflexes occur, which can be manifested locally or distally. Clinically, any compromise of muscle energy metabolism can cause myofascial trigger points.[20] There are two major types of fibers for skeletal muscles—Type I and Type II. Type I fibers are good for endurance and are slow to tire because they use oxidative metabolism. Type II fibers are used for short bursts of speed and power, and use both oxidative metabolism and anaerobic metabolism depending on the particular sub-type, and are therefore quicker to fatigue. In prolonged hypothyroidism, skeletal muscle may increase its bulk due to hypertrophy but decrease its force.[27] Abnormal oxidative metabolism is seen with Type I fibers and abnormal glycolytic metabolism affecting Type II fibers in hypothyroidism.[20] Electromyelographic studies demonstrated loss of Type II muscle fibers, primarily in hypothyroidism and hypertrophy of the Type I fibers.[27] Myofascial trigger points can result. This relationship is normalized to some degree with hormonal treatment.

Cranial manipulation targeting cranial bone motion restrictions and membranous strain patterns can affect pituitary, hypothalamic and thyroid function. Vascular supply to and from the pituitary gland can be augmented via sphenoid manipulation, and its relations to dural and the deep cervical fascial. Sphenoid bone mobilization can increase vascular nutrition to and from the pituitary gland, and release abnormal cervical fascial tensions at its attachment to the pterygoid processes bilaterally. Cranial base manipulation via base spread would be used to augment sphenobasillar motion. Occipital bone mobilization, parasympathetic output from the jugular foramen, and myofascial attachment strains at the foramen magnum and upper cervical regions, can also be addressed. Vagotonic and allergic reactions with resultant histamine release are due in many cases to insufficient thyroid stimulation.[4] Temporal bone balancing would affect the thyroid cartilage attachments via the stylohyoid ligaments and stylopharyngeous muscle groups. A.T. Still discussed treatment of specific mandibular muscle attachments such as the masseter, buccinator and inferior maxilla. Cranial manipulation will likely improve memory and mentation in the hypothyroid patient. Thyroid hormonal carrier proteins within the central nervous system can be appropriately mobilized and utilized more efficiently. Vagal tone can be enhanced by cranial osteopathic manipulative treatment, which can help with the constipation commonly seen in these subjects. Sacral mobilization, which enhances cranial motion, can be increased with sacral rocking techniques. Pretracheal myofascial release can also be augmented by techniques such as tracheal mobilization, thyroid mobilization, cricoid mobilization, hyoid bone mobilization

and anterior cervical soft tissue mobilization. The thyroid gland is directly attached to the thyroid and cricoid cartilages by ligamentous attachments. The arteries, lymph vessels and veins of the thyroid gland run within this fascial layer. Treatment of the anterior cervical tissues may help alleviate the goiter and hoarseness and recurrent laryngeal nerve alterations. Dr. Still discusses treatment of the gland proper.

In hypothyroidism, there is a generalized accumulation of body fluid that can be mobilized from the tissues. Fluid mobilization techniques can be utilized locally and distally, using drainage enhancement techniques such as cervical effleurage and cervical paravertebral muscular inhibition. These will allow soft tissue relaxation and better vascular flow. Vertebral somatic dysfunction correction, sternal lymphatic pump, thoracic outlet release, sternal release, T1/first rib release would also help restore vascular flow. The clavicles are near the thyroid gland, and attach to the first rib and manubrium. Clavicular somatic dysfunction should be treated to ensure venous and lymphatic drainage of the thyroid gland. Compression of the thyroid gland and its surrounding structures by the clavicles and the manubrium would diminish nutrition to the gland, and perhaps directly alter the structural integrity of the thyroid gland. Sternal restriction with posterior displacement has the potential to impede vascular flow.[24] The inferior thyroid veins drain the inferior poles of the thyroid gland, and run posterior to the manubrium of the sternum, directly affecting drainage. Large retrosternal goiters cause venous distention over the neck and difficulty breathing, especially when the arms are raised.[11] The ligamentous connections locally influence central venous and lymphatic drainage.[28] This may promote enhanced organ function to those organ systems directly affected by thyroid hormone via thyroid receptors (cardiac, gastrointestinal, pulmonary, renal and neurologic). Sympathetic supply can be normalized to promote maximum vascular patency using osteopathic techniques that will treat the superior, middle and inferior cervical sympathetic ganglia and upper thoracic spine—T2-T3 as per Louisa Burns's viscerosomatic research—surrounding soft tissues. Unnecessary arteriolar vasoconstriction can be alleviated, increasing the flow of vital nutrients to and from the thyroid gland. Counterstrain points in the upper thoracic region due to facilitation and somatic dysfunction are probable. Chapman's points, which are abnormal gangliform contractions induced from hypersympathetic tone, result in edema and myofascial thickening (tender points). These points lie deep to the skin in deep fascia or periosteum. They are typically paired reflex points having an anterior and a posterior component. Anterior points can be palpated at the anterior T2 interspace bilaterally. Posterior Chapman points are palpated at the T2 perispinous and spinous process in thyroid disease. Treatment to these points can improve lymphatic drainage and help to normalize sympathetic tone to the thyroid gland, which can in turn affect thyroid function and enhance cardiovascular flow.

Somatic relationships palpated during my personal treatment experiences with thyroid patients are similar to others, with some added insights. "TART" findings have a direct correlation with symptom severity. In some cases, a "sidedness" exists to these somatic findings, with one side being in a more exacerbated state than the other. This reflected the patient's current disease state. The palpatory changes from one side of the gland to the other also paralleled the somatic findings. Common recurrent musculoskeletal findings palpated included clavicular restrictions at both the proximal and distal ends, impingement type shoulder dysfunction due to malposition of the acromioclaviclular joint and manubrium from a progressing goiter. Cervical and upper thoracic muscle hypertonicity was prevalent with tenderness at C2 and T2 vertebral levels. Tenderness at the second intercostal space anteriorly was particularly reproducible in patients with labile thyroid hormonal levels and moderate symptomatology. Cranial dysfunction was also common, particularly sphenobasillar compression or extension patterning.

Early intervention with OMT in the euthyroid (TSH high > 5 mU/L) patient to preserve homeostasis is ideal. These patients have subclinical thyroid disease; TSH is high but there are normal circulating T4 and T3 hormone levels. As the disease progresses and thyroid hormone synthesis worsens, there is a progressive fall in serum T4, and a slow rise in serum TSH. Early OMT in patients suffering from non-thyroidal illness syndrome would be helpful. In this syndrome, severe systemic illness, physical trauma and psychiatric disturbances substantially alter thyroid hormone function and decrease thyroid hormone levels. A decrease in serum total and free T3 concentration levels is the critical component of the syndrome. As the serum hormone levels drop, mortality risk increases. Impairment of T3 conversion peripherally within targeted end organs is the most likely cause.[5] The osteopathic physician can augment the thyroid gland processes and accelerate end organ function, affecting the short and long term sequelae of thyroid disease.

To reflect, the severity of the patient's thyroid disease seemed to be reflected in their musculoskeletal findings. Cervical, upper thoracic and shoulder girdle soft tissue changes were most prominent with tenderness in refractive cases mirroring the Fibromyalgia patient. Diffuse muscle tenderness is common in these patients, and may be the major physical finding in mild hypothyroidism.[20] When the

patient is symptomatic their pain tolerance was lowered. When serum thyroid function tests normalized, the patient's symptoms lessen, which included their musculoskeletal manifestations. Ray reports success with three treatments per week, as well as strong exercise of the neck, shoulders and upper spinal muscles. Ironically, in my experience, when serum thyroid hormone levels are not stabilized on thyroid hormone replacement, the patients do get symptomatic relief with OMT, however the effects of the treatment lasted for shorter periods of time. To achieve positive treatment effects initially, increased treatment time and visit frequency were helpful. In the chiropractic literature, hypothyroid patients responded promptly to therapeutic manual procedures, and had lasting effects after hormonal replacement therapy was initiated.[26] Travell reiterates similar findings. Treatment of the hypothyroid condition (with hormonal therapy) makes myofascial trigger points more responsive to manual therapy.[20] In the literature, there was considerable reduction of myofascial pain within 4-6 weeks of achieving a TSH of (.5-.2 mIU/L) in the hypothyroid fascial pain patient.[20]

OMT helps lessen patient symptoms and restores the body's inherent neurologic, endocrine, mechanical and circulatory capabilities. Post OMT, the affected soft tissues seem to display more motion, compliance, bounce and recoil. They have a healthier feel to superficial and deep palpation. Fluid seems to move more easily throughout the tissues during primary and secondary respiration. There is less facilitation present locally and distally. Tissue and joint tenderness are palpably decreased, and range of motion is restored. Normal tissue turgor, texture, temperature and color resulted with increased myofascial and joint symmetry after osteopathic treatment. In clinical practice, OMT has shown to be quite helpful in the restoration of function in all areas of the body affected by the thyroid gland.

In autoimmune mediated thyroid disorders, multiple endocrine organs can be affected by antibodies against their organ proteins, creating polyglandular deficiency states.[5] The thyroid gland affects other endocrine and non endocrine organs, and treatment of the thyroid patient must incorporate treatment of these secondary manifestations. When there is a dysfunction in one part of the endocrine system, all parts are stressed from this lack of function. Anecdotal case reports reiterate this point.[21, 29] It is assumed that patients with permanent pathologic structural changes in their thyroid anatomy will be more resilient to manual treatment, since structure affects function. It stands to reason that if thyroid patients are treated earlier with manual osteopathic intervention, function may be preserved for longer periods of time, and irreversible pathologic structural changes may be limited.

By utilizing osteopathic manipulative treatment in addition to hormonal replacement, the patient can lead a more productive life. The ramifications of osteopathic treatment in the hypothyroid patient can be further seen in the sequelae of long term pharmaceutical management in thyroid disease. Perhaps hormonal replacement therapy can be minimized, or alleviated completely, with positive somatic and visceral responses to osteopathic manipulation; the need for surgical interventions can be diminished. These responses are expressed as overall well being, better health, less symptomatology and a better quality of life. Osteopathic manipulative treatment can enhance current standard medical treatment for this condition as we know it.

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CME QUIZ

The purpose of the quiz found on page 32 is to provide a convenient means of self-assessment for your reading of the scientific content in "An Osteopathic Approach to Hypothyroidism" by Denise K. Burns, DO, FAAO.

Answer each question listed. The correct answers will be published in the March 2011 issue of the AAOJ.

To apply for Category 2-B CME credit, transfer your answers to the AAOJ CME quiz application form answer sheet on page 32. The AAO will record the fact that you submitted the form for Category 2-B CME credit and will forward your test results to the AOA Division of CME for documentation. You must have a 70% accuracy in order to receive CME credits.

AAO Calendar of Events

December 2010

10-12 The Cranial Approach of Beryl Arbuckle, DO
Kenneth J. Lossing, DO
UMDNJSOM, Stratford, NJ

February 2011

4-5: Education Committee Meeting
University Place Conference Center and Hotel, Indianapolis, IN

9 PS&E Committee Teleconference

March 2011

12-15 *New Manual Articular Approaches to the Spine*, Featured speaker Jean Pierre Barral, DO (France)
Broadmoor Hotel, Colorado Springs, CO

14-15: *OMM for Pediatric Gastrointestinal Conditions*
Gregg Lund, DO and Jane Carreiro, DO
Broadmoor Hotel, Colorado Springs, CO

15: Fellowship Committee Meeting
Broadmoor Hotel, Colorado Springs, CO

15: Education Committee Meeting
Broadmoor Hotel, Colorado Springs, CO

16: Board of Governors Meeting
Broadmoor Hotel, Colorado Springs, CO

16: Board of Trustees Meeting
Broadmoor Hotel, Colorado Springs, CO

16: Investment Committee Meeting
Broadmoor Hotel, Colorado Springs, CO

17: Annual Business Meeting
Broadmoor Hotel, Colorado Springs, CO

19: Board of Trustees Meeting
Broadmoor Hotel, Colorado Springs, CO

Other committee meetings will be listed as they are confirmed.

June 2011

10-12 Osteopathic Considerations in Sports Medicine
Kurt Heinking, DO, FAAO
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Atypical Pathologic Somatic Dysfunctions: Techniques Revisited

David S. Miller, DO

Abstract

The difference between the biomechanics of Pathophysiologic Somatic Dysfunctions (Type I & II) and Atypical Pathologic Somatic Dysfunctions that do not fit the biomechanical definitions for Type I or II are described below. Four types of Atypical Pathologic Somatic Dysfunctions are characterized. They do not follow standard nomenclature definitions biochemically, therefore they form a new nomenclature—"Type III Somatic Dysfunction" is proposed. The diagnosis, treatment, and importance of these somatic dysfunctions are discussed.

Background

Spinal Biomechanics is taught at all osteopathic medical schools, with emphasis on Fryette's Laws[1]. The differences between Neutral (Type I) group curve spinal biomechanics, and Non-Neutral (Type II) single segment spinal biomechanics are emphasized. Most schools place little or no emphasis on the presence or importance of other types of somatic dysfunction which do not follow these biomechanical principles. Rotating residents, interns and medical students are surprised at the prevalence of these somatic dysfunctions (once they are trained in their diagnosis and treatment of these dysfunctions), and how little emphasis was given to the diagnosis and treatment of these types of somatic dysfunctions in their training.

Throughout this article, the word "lesion" will be used interchangeably with the current terminology, "somatic dysfunction," because of the use of references dating back to the turn of the previous century when this archaic term was the norm.

Fryette briefly mentioned two of the four types of lesions[1], naming these Atypical Lesions "Simple Flexion" and "Simple Extension." [3] The diagnosis and treatment of these lesions was also described by Walton[4], yet neither Walton nor Fryette discuss the incongruence of their biomechanics with standard spinal biomechanical nomenclature. Since they do not follow the rules for Type I or Type II spinal biomechanics, I propose the new terminology, Type III Somatic Dysfunction, and I will refer to them as such for the remainder of this discussion. Furthermore, neither Fryette nor Walton characterized laterally translated vertebra, or single segment neutral spinal dysfunctions.

The importance of these lesions was not mentioned in any of these previously cited works.. They were seemingly included for completeness, as if to say these dysfunctions exist. I suggest that the presence of these dysfunctions is of great significance. In my experience, they are frequently causally related to chronic pain, chronic fatigue and end organ dysfunction via somatovisceral reflexes. When found in patients with chronic respiratory conditions, like chronic obstructive pulmonary disease (COPD) and asthma, or heart conditions, like coronary artery disease (CAD), arrythmias, and congestive heart failure (CHF), etc., they frequently cause or contribute to significant comorbidity.[5]

Classically, Type I lesions are described as posturally induced group curves, whereas Type II lesions are described as traumatically induced, single segment dysfunctions. In my experience, in contradistinction to Type I and II lesions, Type III somatic dysfunctions are traumatically induced, *with significant or repetitive forces.* The difference is represented in the force required to attain a pathologic vs. pathophysiologic barrier, (e.g., the mechanism of producing a Type II lesion might occur while picking up a television and twisting with it, whereas the mechanism of producing a Type III lesion might be while carrying a television overhead, and being struck from behind [hyperextension]).

The mechanism of injury required to produce a Type III somatic dysfunction involves hyperflexion, hyperextension, or lateral translation of a single vertebral segment, in relation to the segments above and below, in a traumatic manner such as in a motor vehicle accident or a slip and fall. Less commonly, these types of somatic dysfunctions are caused by cumulative trauma (e.g, a gymnast repetitively whipping his or her thoracolumbar junction backwards) or chronic postural dysfunction. It may also be caused when a single segment is laterally flexed and rotated in opposite directions, without producing a group curve. This is rare, but exists and is therefore included for completeness.

Diagnosis

There are four types of Type III Somatic Dysfunctions: hyperflexed, hyperextended, single segment neutral dysfunctions and laterally translated segments. Acute and subacute Type III Somatic Dysfunctions are

usually associated with markedly increased pain overlying the spinous process, posteroanterior (PA) translation (most easily tested in the supine position), and a marked decrease in PA translation of the pathologic segment in comparison to the segment above and below.

In the thoracic spine, the hyperflexed and hyperextended dysfunctions frequently provide a constellation of vertebral and rib somatic dysfunction findings that give clues to their presence, even in the absence of symptoms, as is frequently the case with chronic Type IIIs. Symmetric rib somatic dysfunctions can be associated with hyperflexed and hyperextended segments. Bilaterally inhaled ribs (e.g., bilateral rib 7 exhalation dysfunctions) may be associated with a hyperextended Type III somatic dysfiinction of T7, and bilateral exhaled ribs (e.g., inhalation dysfunction of rib 6 bilaterally) may be associated with a hyperflexed Type III somatic dysfunction of T6.

In my experience, unlike acute Type III somatic dysfunctions, chronic Type III somatic dysfunctions are not usually associated with marked pain overlying the spinous process with PA translation. They can be diagnosed by the presence of these bilateral symmetrically inhaled or exhaled rib dysfunctions coupled with poor PA translation of the involved vertebral segment when compared to adjacent segments.

A laterally translated vertebral somatic dysfunction may be diagnosed by examining the spinous processes. Purely laterally translated vertebral segments will be noticeably "out of line" laterally in relation to the spinal segments above and below (without any rotation or lateral flexion components). To clarify this statement, I would say this is relatively true. When I say it is purely laterally translated, without a rotational component, this is actually not possible. There must be a few millimeters of rotation for the vertebra to glide laterally on the facet joints, but the amount of rotation is minimal compared to the amount of lateral translation (which usually approximates a centimeter). It is important to remember that there can be significant asymmetry of the spinous processes in some regions of the spine, and that static findings of a spinous process "out of line" must correlate to motion palpation findings.

Because pathologic somatic dysfunctions are frequently underdiagnosed and undertreated, these types of somatic dysfunctions can be deeply held chronic restrictions. They are often recalcitrant to treatment and prone to reoccur, requiring repetitive treatments over time to eradicate the pattern of dysfunction completely. Patients should be counseled to avoid postural stances that recreate these dysfunctions, and to avoid stretching their spine backwards when they have recurrent hyperextension lesions. Since subacute and chronic lesions are traumatically induced with significant forces, the presence of fibrosis is an axiom. For this reason, percussion hammer treatment prior to High Velocity Low Amplitude (HVLA) treatment for hyperflexed and hyperextended vertebra is extremely beneficial and highly recommended.

According to Fulford, the percussion hammer creates a standing wave phenomenon capable of breaking the covalent disulfide bond crosslinks between the beta pleated sheets of collagen molecules.[6] This effectively increases the fluidity in the fibotic region.

Treatment

In my opinion, in the appropriate patient, the treatment of hyperflexed Type III somatic dysfunctions can best be accomplished using a supine HVLA (e.g. "Kirksville Crunch" or "Double Arm Thrust") technique. Hand and spine positioning varies from the standard HVLA technique. In this method, a partially open volar fist is placed *overlying the spinous process of the involved segment.* In one treatment method, the patient's arms are crossed over the chest with the elbow contralateral to the operator being superior. The elbows are positioned directly over the pathologic segment. Respiratory cooperation is utilized, and, upon, exhalation an anteroposterior (AP) thrust is directed in a mainly posterior, slightly superior direction toward the involved segment, while lowering the patient's head toward the table to maintain the spine in a relatively extended position. Another treatment method which, in my experience, works better for higher level thoracic dysfunctions, utilizes bilaterally flexed elbows in a supine position, with the patient's fingers interlaced behind the neck, again with a posterior, superior directed force.

The treatment for hyperextended Type III somatic dysfunctions is similar to the previous description, however, the hand placement and spinal position differs from the above description. The partially opened volar fist is placed *directly over the spinous process of the spinal segment, immediately below the pathologic spinal segment.* (e.g., hand placement overlying the SP of T8 to treat a hyperextended T7 vertebral segment). This variation of hand placement is necessary to allow the spinous process of the involved segment to "flip-up" in relation to the vertebra below. During this treatment, the spine is maintained in maximal flexion (by having the patient's chin on his or her chest), with a mainly posterior, slightly inferiorly directed force at T7.

Combined Technique Treatment of laterally translated vertebral segments involves indirectly accentuating the vertebral segment in the direction that it is laterally translated, followed by directly laterally translating the

vertebra in the opposite direction back to neutral. In my opinion, this can best be accomplished in a seated position, with the spine in flexion to effectively unload the facet joints. However, some practitioners might find it easier to perform supine HVLA with a marked amount of lateral flexion corrective force. When I find these in the upper thoracics, I do predominantly seated lateral thrusts in the flexed position.

In morbidly obese patients, or in patients where HVLA is poorly tolerated, another treatment option for hyperflexed or hyperextended Type III somatic dysfunctions is what is referred to as the "Reverse Sit-up Technique." In the Reverse Sit-up Technique, hand placement is critical and unchanged from the previously described positions. The patient is placed in a seated knee-chest position with his or her fingers interlaced behind the neck (similar to the standard sit-up position.) The head is placed in a flexed position for hyperextended vertebra, and in a slightly extended position for hyperflexed vertebra. Respiratory cooperation is utilized, with deep relaxation emphasized to the patient. Upon complete exhalation, the patient rolls backward down into a supine position onto the operator's partially opened relaxed fist, overlying the appropriate spinous process.

Alternately, these spinal dysfunctions can be treated with indirect techniques in a lateral position (e.g., hyperextending the spine at the T7 vertebral segment to treat at Type III hyperextended vertebral segment). In my experience, these techniques seem to be less effective in chronic, recalcitrant lesions, but are beneficial as a pre-treatment, partial or soft tissue release prior to the HVLA treatment of these lesions.

It is sometimes taught that treatment of the spine can obviate the need to treat the ribs, as frequently the associated rib dysfunction resolves after treatment of the spine. After treating these thoracic vertebral dysfunctions, I have found it critical to aggressively treat the associated ribs (and if at a relevant level, diaphragmatic findings), *even if it seems as if the ribs are improved after vertebral treatment.* Freeing the ribs individually, and releasing the diaphragm, will provide the patient substantial relief and ease of breathing, above and beyond the amount of relief obtained from treating the vertebra alone.

Similar Type III somatic dysfunction can be found in the lumbar spine. They are diagnosed by the poor PA translation technique, coupled with the aberrant spacing of the spinous processes described above. Hyperextended vertebra can be associated with marked lumbar lordosis. Hyperflexedvertebrae can be associated with flattening of the lumbar spine. In the upper lumbar spine, they can be treated with the HVLA techniques previously described; in the lower lumbar spine (or with very large patients), the reverse sit up technique is usually utilized.

Similar Type III somatic dysfunctions can be found in the cervical spine.[4] Classically, these dysfunctions create what is attributed to "palpable arthritis," as the cervical vertebra are not lined up in a normal lordotic curve. They have either a marked anterior step-off in the lower cervical spine (usually C5-6), or a marked posterior step-off in the mid cervical spine (usually C2-3). Laterally translated segments are out of line laterally, yielding a palpable abnormality that correlates with gross X-ray findings.

For hyperextended cervical vertebra, HVLA treatment begins with disengagement by cephalad traction, with bilateral middle fingers on the articular pillars on the segment above, coupled with a corrective HVLA counterforce (a forward flick of the wrists), with bilateral thumbs on the anterior transverse processes of the hyperextended vertebra. For the inexperienced practitioner, care must be taken to avoid carotid occlusion with the thumbs. HVLA treatment of hyperflexed vertebra is essentially the opposite, with bilateral thumbs on the anterior transverse processes of the segment above, and middle fingers on the hyperflexed segment's transverse processes posteriorly.

The non-specific general mobilization HVLA thrust of these segments can be performed similarly to the technique described by DiGiovanna[7] --by cupping the occiput and mandible and distracting superiorly, then performing an HVLA traction tug. The difference is, for Flexion Dysfunctions, one tugs with the head extended down to the segment. For Extension Dysfunctions, one tugs with the head flexed down to the involved segment. Muscle Energy treatment for hyperextended cervical vertebra can be accomplished by holding the segment below, and having the patient flex his or her neck while the operator resists flexion efforts. Purely laterally translated cervical vertebra may be treated by indirect vertebral treatment, laterally translating them to a neutral point, waiting for a release, then following indirectly until a subsequent release occurs. Alternately, they may be treated by indirect vertebral treatment followed by a gentle Low Velocity Moderate Amplitude (LVMA) corrective force (with some superior traction) toward the midline. A HVLA laterally directed thrust can be utilized as well, but one must insure a component of superior direction at the appropriate angle for the vertebra involved to avoid jamming the facet joints.

Conclusion

Type III somatic dysfunctions are underdiagnosed and undertreated somatic dysfunctions that lead to significant

chronic pain, debility and morbidity. When present in the thoracic region, their impairment of diaphragm function leads to significant immune system dysfunction, and impairment of the body's ability to fight disease through the mechanism of impairing lymphatic return through cysterna chyli. When present in the cervical spine, they can irritate the sympathetic cervical chain ganglia. Through the mechanism of irritation of the sympathetic chain ganglia and the propagation of facillitated segments, they cause significant end organ dysfunction.

These asymptomatic Chronic Type III somatic dysfunctions might be analogous to the Painful Minimal Intervertebral Joint Dysunctions (PMIDs) Mainge discussed in his book *Diagnosis and Treatment of Pain of Vertebral Origin*. In contradistinction to their conclusion that asymptomatic vertebral dysfunctions need not be corrected, it is my opinion that it is vital these Pathologic Vertebral Dysfunctions are agressively diagnosed and treated, until the patient returns without them. Due to their severity and the proximity of their location to the sympathetic chain ganglia, the presence of Type III somatic dysfunctions in the thoracolumbar region can lead to somatovisceral propagation.[5] Their presence in the spine is the nidus for the creation of facillitated segments,[7] and the end organ pathology these create. The end result is "dis-ease"of the organs supplied by the nerves related to that segment.

It is my experience that these types of somatic dysfunctions are highly correlated with not only chronic pain, but chronic fatigue as well. I have noticed a high correlation with the presence of hyperextended dysfunctions at T7 and T10 associated with the complaint of persistent fatigue. It is the presence of chronic hyperextension of T7 and T10, coupled with bilaterally inhaled 7th and 10th ribs, which leads to marked diaphragmatic exhalation restriction. This causes air trapping and subsequent chronic fatigue. These chronic rib dysfunctions have obvious negative sequelae on chronic respiratory conditions such as COPD and asthma, and can be responsible for the "barrel chest" associated with these conditions.

In addition, these common dysfunctions would tend to have significant impact on immune system function, and the body's ability to fight disease. Since these dysfunctions cause diaphragm flattening and directly impair diaphragmatic function, they can impair pumping of lymph from the lower extremities and abdominal and pelvic viscera through the cysterna chyli. This would cause lymphatic congestion and stasis of the abdominal and pelvic viscera. By the same mechanism, they would cause stasis of the sinusoidal organs, (e.g., liver, spleen) as these organs are pumped via normal diaphragmatic function. Longstanding dysfunction would tend to yield cholestasis and eventually cholelithiasis due to the effects on the liver-gallbladder complex, and polycythemia due to splenic stasis. Splenic stasis leads to immune dysfunction via decreased circulating mast cell populations from sequestration in the spleen.

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Restoration of a McManis Treatment Stool

Robert C. Clark, DO, MS

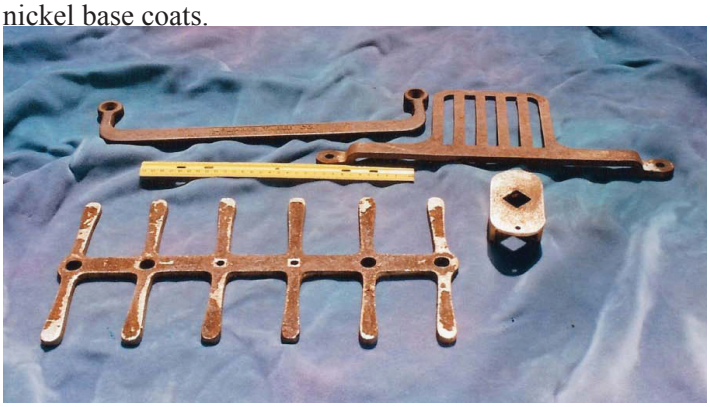
The seat before restoration

Several years ago, a colleague gave me the skeleton of a McManis Treatment Stool. The stool had been partially disassembled and was missing a couple parts. The upholstery was shredded and rotting. The wood components had severe water damage and mold. Paint was flaking off and the chrome parts were severely rusted. It was clearly unusable and required complete disassembly and restoration.

The disassembly process was fairly straightforward-simply remove all the nuts, bolts and screws holding it together. All the fasteners were discarded. The way the seat was assembled required removing all the remaining fabric and padding to get to the wood seat parts.

The resoration process began with sandblasting all the painted parts. I discovered brass caps on the legs of the stool under the paint. The brass was polished and lacquered to prevent tarnish. The metal parts were primed and painted.

The plated parts were sent to a restoration chrome plating company for cleaning, de-rusting and chrome plating. Originally, many McManis tables and stools were nickel plated, but some were chrome plated. Chrome plating is a three step process that applies layers of copper, nickel, then chrome. Chrome is actually transparent and gives a hard shiny finish to protect the softer copper and nickel base coats.



Samples of the chrome work

For stability, the wood parts were treated with a coat of oil, then used as patterns to make replacement parts so they are exact replicas of the originals. The wood parts were sent to an upholstery shop for new padding and fabric.

When all the parts were finished, the stool was reassembled. The new hardware consisted of chrome plating where the fasteners would be visible. For fasteners not visible, standard bright finish hardware was used. I discovered that the screws used on McManis stools and tables are no.12 screws. These are not used much today, but are essential to fit the holes in the original McManis parts.

Since the seat's height adjustment handle was missing, I found a machine tool supplier who made a handle of the correct thread size, albeit a different style from the original. Teflon buttons were also put on the bottom of the feet of the stool to make it easier to move on my treatment room carpet floors.

The finished product

Today, I have a treatment stool that is rugged and reliable and delightfully comfortable.

Author:

Robert C. Clark, DO, MS, has seen three McManis Treatment stools and owns two of them. This stool was his first McManis restoration project. His OMT practice is based in Concord, California. His son, Arthur Clark, who is a machinist, mechanic and computer service technician, assists him in his restoration work.

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From The Archives

From: Still AT. *Philosophy of Osteopathy*. Kirksville, MO. Andrew Taylor Still, Publisher. 1899. pp. 104-113.

Editor's Note: In this issue, we look at the lymphatic system and its importance in osteopathic medicine. For this, we go directly to the writings of Andrew Taylor Still, MD, DO. *-Raymond J. Hruby, DO, FAAO*

Chapter VI: The Lymphatics

Importance of the Subject

Possibly less is known of the lymphatics than of any other division of the life-sustaining machinery of man. Thus, ignorance of that division is equal to a total blank with the operator. Finer nerves dwell with the lymphatics than even with the eye. The eye is an organized effect, the lymphatics the cause; in them, the spirit of life more abundantly dwells. No atom can leave the lymphatics in an imperfect state and get a union with any part of the body. There, the atom obtains form and knowledge of how and what to do. The lymphatics consume more of the finer fluids of the brain than the whole viscera combined. By nature, coarser substances are necessary to construct the organs that run the blast, and rough forging divisions. The lymphatics form, finish, temper and send the bricks to the builder with intelligence, that he may construct by adjusting all according to nature's plans and specifications. Nature makes machinery that can produce just what is necessary, and when united, produces what the most capable minds could exact.

The lymphatics are closely and universally connected with the spinal cord and all other nerves, long or short, universal or separate, and all drink from the waters of the brain. By an action of the nerves of the lymphatics, a union of qualities necessary to produce gall, sugar, acids, alkalies, bone, muscle and softer parts, with the thought that elements can be changed, suspended, collected and associated, and produce any chemical compound necessary to sustain animal life, wash out, salt, sweeten and preserve the being from decay and death by chemical, electric, atmospheric or climatic conditions. By this, we are admonished in all our treatment not to wound the lymphatics, as they are undoubtedly the life giving centers and organs. Thus, it behooves us to handle them with wisdom and tenderness, for by and from them a withered limb, organ or any division of the body receives what we call reconstruction, or is builded anew, and without this cautious procedure, your patient had better save his life and money by passing you by as a failure, until you are by knowledge qualified to deal with the lymphatics.

Demands of Nature on the Lymphatics

Why not reason on the broad plain of known facts, and give the why he or she has complete prostration. When all systems are cut off from a chance to move and execute such duties as nature has allotted to them, motor nerves must drive all substances to, and sensation must judge the supply and demand. Nutrition must be in action the time and keep all parts well supplied with power to labor or a failure is sure to appear. We must ever remember the demands of nature on the lymphatics, liver and kidneys. They must work all the time, or a confusion for lack in their duties will mark a cripple in some function of life over which they preside.

Dunglinson's Definition

Dunglinson's scientific definition of the lymphatics is very extensive, comprehensive and right to the point for our use as doctors of Osteopathy. He describes the lymphatic glands as countless in number, universally distributed all through the human body, containing vitalized water and other fluids necessary to the support of animal life, running parallel with the venous system, and more abundantly there than in other locations of the body, at the same time discharging their contents into the veins while conveying the blood back to the heart from the whole system. Is it not reasonable to suppose that, besides being nutrient centers, they accumulate and pass water through the whole secretory and excretory systems of the body in order to reduce nourishment to that degree from thick to thin, that it may easily pass through all tubes, ducts and vessels interested in distribution, as nourishment first, and renovation second, through the excretory ducts? The question arises whence cometh this water?

Dangers of Dead Substances

This leads us back to the lungs as one of the great sources of which you have been informed under the head of "Lungs, Gases and Water." With this fountain of lifesaving water provided by nature to wash away impurities as they accumulate in our bodies, would it not be great stupidity in us to see a human being burn to death by the fires of fever, or die from asphyxia by allowing bad or dead lymph, albumen, or any substance to load down the powers of nature and keep the blood from being washed to normal purity? If so, let us go deeper into the study of the lifesaving powers of the lymphatics. Do we not find in death that the lymphatics are dark, and in life, they are healthy and red?

Lymph Comtinued

What we meet with in all diseases is dead blood, stagnant lymph, and albumen in a semi-vital or dead and decomposing condition all through the lymphatics and other parts of the body, brain, lungs, kidneys, liver

and fascia. The whole system is loaded with a confused mass of blood, that is mixed with much or little unhealthy substances, that should have been kept washed out by lymph. Stop and view the frog's superficial lymphatic glands; you see all parts move just as regular as the heart does; they are all in motion during life. For what purpose do they move if not to carry the fluids to sustain by building up, while the excretory channels receive and pass out all that is of no further use to the body. Now we see this great system of supply is the source of construction and purity. If this be true, we must keep them normal all the time, or see confused nature in the form of disease the list through. Thus we strike at the source of life and death when we go to the lymphatics.

With this fountain of lifesaving water, provided by nature to wash away impurities as they accumulate in our bodies, would it not be great stupidity in us to see a human being burn to death by the fires of fever, or die from asphyxia, by allowing bad or dead lymph, albumen or any substance to load down the powers of nature to keep the blood washed to normal purity? If so, let us go deeper in the study of the life-sustaining powers of the lymphatics.

Nature's Solvents

The brain flushes the nerves of the lymphatics first and more than any other system of the body. No part is so small or remote that it is not in direct connection with some part or chain of the lymphatics. The doctor of Osteopathy has much to think about when he consults natural remedies, and how they are supplied and administered, and, as disease is the effect of tardy deposits in some or all parts of the body, reason would bring us to hunt a solvent of such deposits, which hinder the natural motion of blood and other fluids in functional works, which are to keep the body pure from any substance that would check vital action. When we have searched and found that the lymphatics are almost the sole requisite of the body, we then must admit that their use is equal to the abundant and universal supply of such glands. If we think and use a homely word and say that disease is only too much dirt in the wheels of life, then we will see that nature takes this method to wash out the dirt. As an application, pneumonia is too much dirt in the wheels of the lungs, if so we must wash out; nowhere can we go to a better place for water than to the lymphatics. Are they not like a fire company with nozzles in all windows ready to flush the burning house?

Where are the Lymphatics Situated?

A student of life must take in all parts, and study their uses and relations to other parts and systems. We lay much stress on the uses of blood and the powers of the nerves, but have we any evidence that they are of more vital importance than the lymphatics? If not, let us halt at this universal system of irrigation, and study its great uses in sustaining animal life. Where are they situated in the body? Answer by: where are they not? No space is so small as to be out of connection with the lymphatics, with their nerves, secretory and excretory ducts. Thus, the system of lymphatics is complete and universal in the whole body. After beholding the lymphatics distributed along all nerves, blood channels, muscles, glands and all organs of the body, from the brain to the soles of the feet, all loaded to fullness with watery liquids, we certainly can make but one conclusion as to their use, which would be to mingle with and carry out all impurities of the body, by first mixing with such substances and reducing them to that degree of fluids in fineness that could pass through the smallest tubes of the excretory system, and by that method, free the body from all deposits of either solids or fluids, and leave nourishment.

The Fat and the Lean

A question: Why is he too fat and she only skin and bone, while a third is just right? If one is just right, why not all? If we get fat by a natural process, why not reverse the process and stop at any desirable point in flesh size? I believe the law of life is simple and natural in both respects if wisely understood. Have we nerves of motion to carry food to all parts, organs, glands and muscles? Have we channels to convey to all? Have we fluids to suit all demands? Have we brain power equal to all force needed? Is blood formed sufficiently to fill all demands? Does that blood contain fat, water, muscle, skin, hair and all kinds to suit each division, organ, and nerve? If so, and blood has builded too much flesh, can it not take that bulk away by returning blood to gas and other fluids? Can that which has been done be done again? If yes be the correct answer, then we should hope to return blood, fat, flesh and bone to gas, and pass them away while in gaseous condition, and do away with all unnatural size or lack of size. I believe that it is natural to build and destroy all material form from the lowest animated being to the greatest rolling world. I believe no world could be constructed without strict obedience to a governing law, which gives size by addition and reduces that size by subtraction. Thus, a fat man is builded by great addition, and if desired can be reduced by much subtraction, which is simply a rule of numbers. We multiply to enlarge, also subtract when we wish a reduction. Turn your eye for a time to the supply trains of nature. When the crop is abundant, the lading would be great, and when the seasons do not suit, the crops are short or shorter to no lading at all. Thus, we have the fat man and the lean man. Is it not reasonable as a conclusion of the most exacting philosophy that the train of cars that can bring loads of stone, brick and mortar until a great bulk is formed, can also carry away until this bulk disappears in part or all? This being my conclusion, I will say by many years of careful observation of the work of creating bodies and destroying the same, that to add to

is the law of giving size, and to subtract from is the law of reduction. Both are natural, and both can be made practical in the reduction or addition of flesh, when found too great in quantity, or we can add to and give size to the starving muscle through the action of the motor and nutrient system conveyed to, and appropriated from the laboratory in which all bodily substances are formed. Thus, the philosophy is absolute, and the sky is clear to proceed with addition and subtraction of flesh. I believe I am prepared to say at this time that I understand the nervous system well enough to direct the laboratory of nature, and cause it through its skilled arts to unload or reduce, he who is over-burdened with a super-abundance of flesh, and add to the scanty muscle a sufficiency to give power of comfortable locomotion and other forces, by opening the gate of the supply trains of nutrition.

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   A. Occipitomastoid restriction
   B. Temporomandibular joint dysfunction
   C. Ethmoid restriction
   D. Sphenobasilar compression
   E. Sagittal suture compression

2. In thyroid disease, posterior Chapman's points can be palpated at what spinal level?
   A. C2
   B. C7
   C. T2
   D. T9
   E. T12

3. Thyroid diseases are second in frequency to diabetes mellitus among endocrine illnesses.
   A. True
   B. False

September 2010 AAOJ CME quiz answers:

1. C
2. D
3. B

Answer sheet to December 2010 AAOJ CME quiz will appear in the March 2011 issue.

Component Societies and Affiliated Organizations Upcoming Calendar of Events

January 15-17, 2011
The Face: An Intermediate Course
Course Director: Douglas Vick, DO
TUCOM/NV, Henderson, NV
CME: 20 Category 1A AOA credits
Contact: Joyce Cunningham
Phone: (509) 469-1520
E-mail: **jcunningham4715@yahoo.com**
Web site: **www.sctf.com**

February 12-16, 2011
Introductory Course:
Osteopathy in the Cranial Field
Course Director: William Lemley, DO, FAAO
Hilton, Lake Buena Vista, FL
Phone: (317) 594-0411 Fax: (317) 594-9299
E-mail**: info@cranialacademy.org**
Web site:**www.cranialacademy.org**

February 17, 2011
Treating the Viscera Using the Primary Respiratory Mechanism, Part I
Course Director: Maurice Bensoussan, MD
Hilton, Lake Buena Vista, FL
Phone: (317) 594-0411 Fax: (317) 594-9299
E-mail**: info@cranialacademy.org**
Web site:**www.cranialacademy.org**

February 18-20, 2011
Treating the Viscera Using the Primary Respiratory Mechanism, Part II
Course Director: Maurice Bensoussan, MD
Hilton, Lake Buena Vista, FL
Phone: (317) 594-0411 Fax: (317) 594-9299
E-mail**: info@cranialacademy.org**
Web site:**www.cranialacademy.org**

March 4-6,2011
Biodynamics of Osteopathy: Phase I
UNECOM, Biddeford, ME
CME: Up to 22 Category 1A AOA credits
Contact: Joan Hankinson
Phone: (207) 781-7900
E-mail: **ohmjh@aol.com**
Web site: **osteopathichealthcareofmaine.com**

March 11-13, 2011
An Osteopathic Approach:
Introduction and Hip Joint & Lower Extremities
OMM at UNECOM, Biddeford, ME
CME: 20 Category 1A AOA credits
Phone: (207) 602-2589 Fax (207) 602-5957
E-mail: **cme@une.edu**
Web site: **www.une.edu/com/cme**

April 6-9, 2011
International Osteopathy Congress
Florence, Italy
Submit scientific works online by 11/30/10
Web site: **www.osteopatia2011.it**

April 13-16
Meeting Future Health Care Needs:
The Role of Interprofessional Education
The Joint AACOM and AODME 2011 Annual Meeting
Marriott Baltimore Waterfront, Baltimore, MD
Contact: Beth Martino (AAACOM) or Penny Friske (AODME)
Phone: (301) 968-4189 or (312) 202-8211
Email: bmartino@aacom.org or aodme@osteopathic.org
Web site: **www.aacom.org/events/annualmtg/**

May 13-15, 2011
An Osteopathic Approach: Introduction and Pelvis
OMM at UNECOM, Biddeford, ME
CME: 20 Category 1A AOA credits
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E-mail: **cme@une.edu**
Web site: **www.une.edu/com/cme**

June 2-6, 2011
Basic Course:Osteopathy in the Cranial Field
PNWUCOM, Yakima, WA
Course Director: Melicien Tettambel, DO, FAAO
CME: 40 Category 1A AOA credits anticipated
Contact: Joy Cunningham
Phone: (509) 469-1520 Fax: (509) 453-1808
Email: **jcunningham4715@yahoo.com**
Web site: **www.sctf.com**

June 3-6, 2011
Biodynamics of Osteopathy: Phase II
UNECOM, Biddeford, ME
CME: Up to 22 Category 1A AOA credits
Contact: Joan Hankinson
Phone: (207) 781-7900
E-mail: **ohmjh@aol.com**
Web site: **osteopathichealthcareofmaine.com**

Local and Systemic Mechanisms in Disturbance of Functions of the Vegetative Nervous System

N.P. Erofeev, MD

Introduction

Visceral therapeutic techniques, which are aimed at the activation of vegetative regulation mechanisms, are included in the arsenal of modern osteopathic medicine. Structural and craniosacral techniques realize their therapeutic effects through the autonomic reflex arch, and, consequently, also influence the parameters of the organism's internal medium. Therefore, there is a direct relationship between the results of classic osteopathic treatment and understanding of functional significance of the vegetative nervous system (VNS). Time had not "made old" the classic conceptions on physiology of the vegetative nervous system by the beginning of the 21st century. At the same time, its separate fields are developing rapidly, which are connected, first of all, with advances in modern biophysics, histology, genetics and biochemistry at the molecular level. Therefore, I cannot pretend to cover all problems of the VNS in my report. I have restricted myself to consideration of some historical issues of studying VNS, and modern knowledge of local and systemic mechanisms of visceral function control. The Symposium theme seems to be urgent also because osteopathy and VNS have the basic elements of commonness. The structural properties are the integral postulate of treatment in osteopathy, and the principle of VNS functional organization, which makes it autonomic.

Vegetative disorders (mechanisms of their development, clinical symptoms and treatment) are one of the most urgent problems in modern medicine in the whole world. This is connected, first of all, with the great prevalence of vegetative disorders. Numerous epidemiologic studies[1] have revealed that vegetative disorders are noted in 25%-80% of observations in the population, starting from the age of puberty, and also including people who consider themselves relatively healthy. At the same time, the vegetative pathology manifests as independent diseases relatively rarely.[1]

Dysfunctions of the vegetative nervous system (VNS) are evident in all types of pathology in man. In some cases, disturbances in VNS activity are manifested as a leading factor, dominant in disease manifestation; in other cases, they develop secondarily in response to affection of any systems and tissues in the organism.

First, let us consider some important issues of terminology and history. Until today, there was no unanimity in the world in the designation of the system, which is discussed at the Symposium. And this is a good occasion to remind colleagues of the contribution of French and Russian scientists and physicians in studying the morphology, physiology and pathology of the vegetative nervous system. These works are still the source of many ideas today. Although their studies are separated by time, the classic knowledge formed by them has great value for the theory and practice of world medicine and modern osteopathy (and not only within the limits of this Symposium).

The term "vegetative nervous system" has existed for more than two centuries. Undoubtedly, distinguishing VNS from other efferent innervation systems of the organism stimulated morphological, physiologic, biochemical and clinical approaches to studying the peculiarities and laws of functioning of both the somatic and vegetative nervous system. At the end of the 19th century J.N. Langley [2, 3, 4, 5, 6] named VNS the "autonomic nervous system," which resulted in a more clear understanding of the structure and function of this nervous system portion. It is appropriate to mention here the groundlessness of some of J.N. Langley's opponents, ascribing to him a statement of the complete independence of the autonomic nervous system from the central nervous system. J.N. Langley writes in his book *Autonomic nervous system*, "When I wished to describe the results…of studies… I meant the "local" autonomy". Also at the end of the 19th century, W. Gaskell[7] suggested to designate the VNS as the "visceral system", but this name is restrictive because vegetative innervation covers all tissues of the organism, not limited only to the internal organs. As a result, two terms prove to be viable today: "vegetative nervous system" used in Russian, German and French speaking countries, and "autonomic nervous system" widespread in English speaking countries.[1]

The first data concerning VNS structure and function dates from the times of Galen. He named the nerve trunk located along the vertebral column "sympathetic," describing the course and distribution of the vagus nerve. A more detailed study of sympathetic nodes, or ganglions, and the solar plexus was performed by Andreas Vesalius, who considered these structures connected the internal organs to the brain.

Anatomist Jacques-Bénigne Winslow (1669-1760), later a professor of operative surgery who worked at

Jardin du Roi in Paris, introduced the term **"symp**athetic nervous system" into science for the first time. This name corresponded to ideas dominant in medicine in that time—that diseases of any organ involved several other organs in the morbid process. Such interrelationship was considered a manifestation of concern (sympathy), with which one organ reacted to the disease of the other. According to J.B. Winslow, sympathetic nerves were those nerve paths, by which the **"**sympathies**"** of the organs were realized. Thus, the hypothesis gave the name sympathetic, compassionate (from the Greek word "sympatheticos") to one of the VNS portions.[1, 8, 9, 10] Jacques-B**é**nigne Winslow had a talent for observations, an ability to describe the structures of the human body systematically and in details. He skillfully eliminated foreign physiological details in the description of the human organism and avoided hypothetical explanations. His work *Exposition Anatomique de la Structure du Corps Humain*[10] became a well-known book, and was used by students and physicians in subsequent centuries. He was the first to prove that nerves going from different parts of the brain and spinal cord to the internal organs formed the other innervation system and belonged to the sympathetic nervous system, and that sympathetic ganglions were independent nerve centers coordinating the functions of different organs.

But the complete adaptation of the vital activity to environmental changes cannot be provided only by the sympathetic influences. This idea was expressed for the first time by another French anatomist, physiologist and surgeon, M.F.X. Bichat (1771-1802). In his scientific works Anatomie Generale, Appliquee a la Physiologie et a la Medecine and Recherches Physiologiques sur la Vie et la Mort,[11, 12] for the first time, he paid attention to the fact that a variety of adaptation reactions of man, animals and plants were manifested in the unity of vegetative, somatic and psychic processes. M. Bichat is not only the creator of the general anatomy of the nervous system, he also divided the nervous system into two portions. One portion was called "somatic;" it is peculiar to animals—their ability to actively move creates the possibility to avoid unfavorable environmental influences ("scandent and crawling" plants also have beginnings of movements). The other portion, the vegetative nervous system, (M.F.X. Bichat introduced this term at the beginning of the 19th century) controls the exchange of substances and energy between the organism and the environment and is common for animals and plants [1, 7-16]. This division still exists, and is used in physiology and medicine for scientific and practice purposes.

In Russia, traditions in studies of physiology and clinical problems of the vegetative nervous system originate from the organic heritage and creative development of nervism principles. Brilliant studies of vegetative (neurogenic) regulation of the activity of the gastrointestinal system performed by I.P. Pavlov (1840–1936) were awarded the Nobel Prize in 1904. I.P. Pavlov also demonstrated the significance of trophic innervations, having revealed in his experiments that one of the nerves of the cardiac plexus, during irritation, increased the intensity of contractions and, consequently, activated the cellular metabolic processes, not changing the cardiac rhythm. [8, 9]

The successors and disciples of I.P. Pavlov paid special attention to the vegetative nervous system in their studies. First of all, it is the teachings of L.A. Orbeli (1882 – 1958) on the adaptation-trophic role of the nervous system which take into account the special significance of the sympathetic system. In his classic experiments, L.A. Orbeli (1927) irritated the motor nerve, and thus brought the frog muscle to fatigue[17] (these experiments were confirmed in warm-blooded animals). Simultaneously, he irritated the sympathetic trunk on the same side, and revealed that addition of such irritation resulted in intensification of contractions of the tired muscle. He proved, with the help of special experiments, that such an increase of the working capacity was a result of the stimulating influence exerted by the sympathetic nerves on metabolic chemical processes. [18]

Another disciple of I.P. Pavlov, A.D. Speransky (1888-1961), theoretically analyzed and proved in experiments that disease was the reaction of tissues to the

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direct effect of an adverse factor. Thorough histological studies performed under the leadership of A.D. Speransky made it possible to establish all stages of successive involvement of separate nervous system elements into the pathological process: its movement along the nerve trunk, affection of the nerve centers, transition to the nerves of the opposite side, taking the process beyond the limits of the respective nerve segment. Consequently, it was possible to directly connect these tissue disorders with that function of the nervous system, which was extensively studied by I.P. Pavlov in his time, called the trophic function. Speransky called them dystrophic, i.e., caused by disturbance of the nerve trophism. Based on the results of his experiments, A.D. Speransky stated that the trophic function acted as the main function in all manifestations of nervous activity—that the consequences of any nerve effect, were connected with changes in trophism. It is not possible to change the functional state of the tissue without changing its trophism. [19, 20]

A new independent stage in the development of teachings on the regulatory connection of the nervous system and internal organs is connected to the activity of K.M. Bykov (1886-1958). While developing I.P. Pavlov's ideas, he created his theory on cortico-visceral relationships. The object of the cortico-visceral theory was to reveal the mechanisms by which the cerebral cortex influenced the activity of the internal organs. K.M. Bykov and his colleagues solved three fundamental problems. First, they proved the principal possibility of formation of conditioned reflexes of the internal organs or, in other words, they proved the connection of the cerebral cortex with the activity of the kidneys, liver, spleen, etc., and also revealed functional properties of these conditioned reflex processes. Second, they studied the transmission of signals coming from the internal organs to the cortex and back, i.e., established internal receptors, pathways and their representations in the cerebral cortex. Finally, they decoded the central mechanisms of the conditioned reflex activity of the internal organs and cortical effects on the general functions of the organism.[21] The cortico-visceral theory also explained the mechanisms of pulse transmission from the cortex to the internal organs. Clear ideas on the double (nervous and humoral) cortical control of the organ activity were formed within the limits of the cortico-visceral theory. [22, 23, 24] Besides that, K.M. Bykov, together with I.T. Kuritsin, developed a special theory of cortico-visceral pathology. According to this theory, the occurrence and development of some diseases of the internal organs are connected to the primary disturbance of higher nervous activity, which can take place as a result of disorder in the extero- and interoceptive signaling. The base of this disorder is formed by the functional weakening of the cortical cells because of overstrain of the intensity and mobility of nervous processes, especially the process of internal inhibition. Because of this, disturbance of functional interrelations between the cerebral cortex and subcortical structures take place, which involve the whole complex of vegetative and somatic functions in the sphere of the pathological process. [25] According to K.M. Bykov and I.T. Kuritsin, [25] a cyclic process occurs in the case of cortico-visceral pathology; on the one hand, the pulses, changing the function, trophism and blood supply of the organ, are sent to it from the cerebral centers, and on the other hand, the cerebral centers receive pulses increasing their pathological activity. Such disturbance in the organism's activity may be fixed and maintained with the help of conditioned reflex mechanism. Within the limits of the conception on cortico-visceral pathology, disturbances of the organism's activity are considered to have a psychotropic nature, i.e., new ideas of the visceral pathogenesis are formed. [24]

In his time, J.N. Langley distinguished the enteral system, i.e., Messner's and Auerbach's plexuses, as an independent portion. The name "enteral nervous system" limits understanding of the function of this very important nervous system portion. A.D. Nozdrachev (born in 1931), Academician of the Russian Academy of Sciences, suggested the term "metasympathetic nervous system," which considerably extended the concepts of the vegetative ganglions and their functional significance in other organs

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(heart, trachea, esophagus, uterus, kidney, ureters, etc.).[26] Based on his own studies, and analyzing the publications of other specialists, A.D. Nozdrachev substantiated and introduced into the literature a concept of intramural nervous structures, which is absolutely new for physiology, he called them the "metasympathetic nervous system". According to his research, this is an independent structure, the third (along with sympathetic and parasympathetic) portion of the autonomic nervous system. [26]

A.D. Nozdrachev formulated the main elements for construction of the theory of this system, which controls practically all current activity of the hollow internal organs. According to this view, neuron groups of the metasympathetic nervous system are able (with interference of centrifugal pulses) to regulate the motor activity of the visceral organs, control secretory and excretory processes, and dynamics of capillary permeability, and to regulate the activity of local (intraorgan) endocrine and immune elements. [26, 27, 28] A.D. Nozdrachev has proved in his experiments that the metasympathetic nervous system has a whole necessary set of functional elements: sensory cells, pacemakers, interneuronal system, tonic neurons, and effector neurons. The metasympathetic nervous system also has a very complex neurochemical structure. In addition to the aforementioned "traditional" mediator systems, its neurons contain ten neuropeptides and other physiologically active substances. A.D. Nozdrachev considers a functional module, i.e., a union of five to seven nervous nodes into a single ensemble with the help of interneuronal connections, a functional unit of the metasympathetic nervous system.[26, 27, 28]

Of course, with the availability of such a powerful theoretical and experimental store in the study of the VNS, clinical vegetology also developed in Russia. A.M. Vein (1928-2003) is an outstanding representative and scientist who productively worked in this direction. He organized and headed the department of nervous diseases (from 1990) and division of pathology of the vegetative nervous system (from 1970) at the Moscow I.M. Sechenov Medical Academy.[1, 29]

The main aspect in the activity of the vegetative nervous system is maintaining the constancy and optimum indices of the organism's internal medium (homeostasis). Strictly speaking, such a purpose of the VNS is not adequate today because the VNS participates in systemic reflexes, e.g., "struggle – escape". In this case, activation of the sympathetic nervous system includes diverse reactions functioning at all levels of the nervous and hormonal systems in the human organism: release of hormones from the adrenal glands, increase of heart rate and blood pressure, dilation of bronchi, intensification of glucose metabolism, dilation of pupils, dilation of vessels in skeletal muscles, inhibition of the intestinal motor activity and secretion, constriction of the skin and visceral blood vessels, pilo-erection. Thus, as is shown in the above example, the VNS uses the systemic mechanisms as an adequate coordinated response to external stimuli. The base of such systemic mechanisms is formed by the peculiarities of VNS functional structure as a component of the integral nervous system of the human organism. The VNS uses the following principles of regulatory system functioning in its activity: feedback (negative and positive), multiplicity and redundancy of controlling contours, hierarchical structure of controlling systems, priorities in the interest of maintaining the constancy of the whole organism's internal medium over the interest of homeostasis at the level of a cell, cell group or a separate organ. The VNS plays the role of the systemic regulator of visceral functions, and carries out such control with the help of reflexes (the controlling contours of visceral reflexes function according to feedback principle) and independently (autonomically) of the consciousness, but not from the activity of the brain and other portions of the nervous system.

Returning to the principle of structural properties mentioned in the beginning of the report, which is common for osteopathy and the VNS, let us note that the morphological and functional structure of vegetative ganglions creates an important integration phenomenon of multiplication; pulse convergence and divergence phenomena are simultaneously pronounced in the vegetative ganglion: pulses from several preganglionic neurons converge on the body of one postganglionic neuron, and any preganglionic neuron innervates many postganglionic neurons. Additionally, this also ensures the reliability of excitation transmission in the VNS.

The VNS is only one component of the integral nervous system, the separate elements of which are dynamically connected with each other. Their integration is achieved due to the extraordinarily high development of the interconnection. Thanks to this, the nervous system perceives all diverse changes in the conditions of the environment and the organism's internal medium, and adequately reconstructs its activity according to these changes. Of course, the effects aimed directly at the nervous system, independently from their point of application, cause the deepest and most extensive changes in the functioning and interrelations of its components. In this situation, the character of the corresponding effect is not of great significance; it is not important whether it causes intensification, perversion or lowering of any function—changes involving the nervous system as a whole will take place in all cases. Therefore, inactivation of the function is not only a "minus function" but also a "plus new function". In other words, it is not possible to

change something "locally" in the organism. Nevertheless, there are local mechanisms in VNS functions. I shall dwell on the trophic nerve factors discovered recently. The most intensive trophic influence on all basic processes of neuron activity is exerted by neurotrophins, i.e., regulatory proteins of the nervous tissue, which are synthesized in its cells (neurons and glia). In the early1950s, Rita Levi-Montalcini and Victor Chamburger showed that death or survival of neurons in vitro and in vivo depended on target tissues innervated by them.[30] Neurotrophins act locally in the area of release, and especially intensively induce branching of dendrites (arborization) and growth of axons (sprouting) in the direction of target cells [30, 31] Neurotrophins are a family of regulatory proteins of the nervous tissue, which are synthesized by neurons and the cells of microglia and glia, and favor proliferation, differentiation and maintaining of the vital activity and functioning of peripheral and central neurons. For this, autocrine and paracrine regulation mechanisms are used.[31, 32] Synaptic sprouting provides "re-intensification" of existing neuronal currents, the formation of new polysynaptic connections[33, 34] and plasticity of the neuronal tissue, and forms mechanisms that participate in the recovery of disturbed neurological functions.[35] Growth factors maintain the life neurons, which it cannot live without .[30-35] In the mature nervous system, neurotrophins regulate both short-term synaptic transmission and long-term potentiation, thus, participating in the provision of nervous system plasticity required for its normal functioning.[35] It has been revealed that neurons long to obtain nerve growth factors.[36] Today the family of neurotrophins consists of the following representatives: nerve growth factor (NGF), brain derived neurotrophic factor (BDNF), neurotrophin-3 (NT-3), neurotrophin-4/5 (NT-4/5), neurotrophin-6 (NT-6) and neurotrophin-7 (NT-7).

Understanding the role of modern theoretical and practical knowledge on trophic factors is of special interest for osteopathic practice. This knowledge may serve as a useful vector for developing the new methods of osteopathic treatment. The point is that NGF (nerve growth factor) intensifies growth and maintains the vital activity of sensory minor cells of spinal ganglions and postganglionic (effector) sympathetic neurons. Thus, osteopathic techniques on the VNS are involved in controlling the incoming (sensory) and outgoing (motor) signals. Furthermore, these chemical neurotrophic factors (i.e., manipulations of the osteopath) provide the interaction between numerous nerve and non-nerve cells, i.e., control homeostasis. Light osteopathic techniques activate the nerve cells, making them form new connections and "pump out" neutrophins, i.e., the substances activating the starting mechanisms of intracellular mobility, prompting the intracellular molecular machines (dyneins and kinesins) to activation. Neurotrophins stimulate the activity of the nerve cells, not only in the VNS but also in the nervous system as a whole, providing normalization of circulation, consequently, a burst of energy in the brain. All osteopathic manipulations on the VNS favor the improvement of blood microcirculation and intensify lymphatic drainage. As a result, osteopathic therapeutic influences saturate with oxygen and recover the connective tissues of the organism, increase the somatotropic hormone content in the organism, suppress pain sensations, and promote the production of serotonin and neurotrophins stimulating brain function.

In conclusion, in connection with the above and what I would like to emphasize especially, I tried to answer a question of vital importance—why do positive therapeutic (including psychotherapeutic) successes of osteopaths throw the European physician into confusion, sometimes causing his irritation, negative reaction and occasionally the loss of belief in his forces and possibilities? Osteopaths (without medicine, in contrast to generally accepted medical practice) achieve therapeutic effects with the help of visceral, structural and craniosacral techniques, which are directly and indirectly aimed at the recovery of natural regulatory interactions between all elements of the human organism.

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