Osteopathic Manipulative Treatment as an Integral Component of the Care Plan for the Patient Whom Suffers from Opiate Addiction

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The Plain Facts
Pennsylvania At-a-Glance
• In 2009, the rate of drug-induced deaths in Pennsylvania was higher than the national average.
• Approximately 7.99 percent of Pennsylvania residents reported past-month use of illicit drugs.
• As a direct consequence of drug use, 1,812 persons died in Pennsylvania in 2007.
• Pennsylvania drug-induced deaths (14.6 per 100,000 population) exceeded the national rate (12.7 per 100,000).
• Prescription drug abuse is the fastest-growing drug problem in the Nation.
• Source: National Survey on Drug Use and Health (NSDUH) 2009-2010
The Plain Facts
from the Weekly Newsletter of St. Sen. Bob Mensch
Pennsylvania 24th Congressional District - 10/27/2016

- On average at least ten Pennsylvanians die every day from a drug overdose, with over 3,500 overdose deaths in Pennsylvania in 2015.
- According to a National Survey of Primary Care Physicians, nine out of 10 doctors reported prescription drug abuse as a moderate to large problem in their communities, and 85 percent believed that prescription drugs are overused in clinical practice.

The Pathophysiology of Drug Addiction

Drug addiction is a chronic disease that results in changes to both brain structure and function. Addictive drugs provide a shortcut to the brain's reward system by flooding the nucleus accumbens (the brain's pleasure center) with dopamine. Repeated exposure to addictive substances causes nerve cells in the nucleus accumbens and the prefrontal cortex (the area of the brain involved in planning and executing tasks) to communicate in a way that couples doing something with wanting it, in turn driving the sufferer to go after it. That is, this process motivates the brain to take actions to seek out the source of pleasure. Addictive drugs, for example, can stimulate the release of 2 to 10 times the amount of dopamine that natural rewards do, and more quickly and more reliably. In a person who becomes addicted, brain receptors become overwhelmed. The brain responds by producing less dopamine or eliminating dopamine receptors. The hippocampus records earlier memories of a sense of satisfaction, and the amygdala responds by creation of a conditioned response to stimuli, e.g. a pill as a means to pleasure.

The Pathophysiology of Drug Addiction

Repeated exposure to the drug results in physical change to the brain's structure, the dopamine receptor, and the function of the transmitter, i.e. dopamine has less of an impact on the brain's reward center. The chronically ill patient no longer has the capability to obtain satisfaction from the drug, however, now struggles with the memory of the desired effect and the need to re-create it.

As the brain goes through these changes, beginning with recognition of pleasure and ending with a drive toward compulsive behavior (to get pleasure) in time, the desired substance no longer gives much pleasure. The pleasure associated with an addictive drug or behavior subsides—yet the memory of the desired effect and the need to recreate it (the wanting) persists.
Can Osteopathic Manipulative Treatment (OMT) be utilized as an integral component of the total treatment program for patients who suffer from opiate addiction? Current treatment protocols include:

- **Drugs** – Aimed to address the depletion of the transmitter dopamine, and symptomatology include: Alpha blockers, anti-depressants (selective serotonin re-uptake inhibitors with dopanergic properties), and cortical stimulants. Typical drugs used for treatment also are not uniform on all formularies, e.g., Subutex, Suboxone, Methadone, Narcan, etc.
- **Cognitive Behavioral Therapy** – A discussion between a patient and a therapist to raise patient awareness of the life situation both now and before the use of drugs.
- **Manual Medicine (OMT)** perhaps used to address discontinuity in circulation, and nerve transmission, as well as localized areas of pain (Somatic Dysfunction).

Somatic Dysfunction

OMT is used to normalize somatic dysfunction as defined below...

Impaired or altered function of the somatic framework (skeletal, arthrodial, or myofascial) which can lead to altered vascular, neural, lymphatic, or metabolic function.

Anatomically consider that “*Through fascia, organ systems communicate via the blood vessels, neural tissue and lymphatic vessels that pass through it.*”

Treatment Goal of Osteopathic Medicine

To maintain “normal” relationships between structural and functional units of the body through:

- A. Connective tissue relations (fascia, ligaments, tendons).
- B. The Vascular and Lymphatic System
- C. The Central and Peripheral Nervous System
- D. The axial and appendicular skeleton.
- E. The Metabolic, Endocrine and Exocrine Systems.
Basic Premise

- 1. The Body is a Unit.
- 2. The body is a self-regulatory mechanism.
- 3. The body has the inherent capacity to heal itself.
- 4. Structure and Function are reciprocally interrelated.

The spinal facilitation concept
...from the works of Denslow, Korr, et al

In principle, the facilitated segment was described as a specific area of the spinal cord that was capable of organizing disease processes. It was a very simplistic model. It had two input and two output routes. The input routes were sensory from musculo-skeletal and viscera. The output routes were the motor efferents to muscle and autonomic motor to sweat glands, blood vessels and viscera. Inside the spinal cord it was suggested that abnormal activity in one area of the spinal cord could spread to adjacent areas. The facilitation process would be initiated when aberrant sensory information from an area of damage or pathology (muscle or visera) was conveyed via the afferents to the spinal cord. This would alter the neuronal activity at the same segmental level and might spread to adjacent areas of the spinal cord affecting spinal centres not directly related to the original injury. For example, a musculoskeletal injury could reach the spinal cord through its afferent connection causing spinal facilitation or sensitisation to take place. Because of the anatomical proximity of the motor and autonomic spinal centres, this spread of excitation would eventually involve these lateral centres. This in turn would alter the segmental autonomic activity leading to changes in vasomotor, sudomotor and visceral activity. The reverse could happen too; through the same neurological mechanisms a pathological condition in the viscera could end up affecting skeletal muscle activity.

The CSF and Osteopathy in the Cranial Field

One of the aims of cranial therapy is to normalize CSF and venous sinus flow to clear metabolites and toxins from the central nervous system. In essence, the CSF circulates throughout the central nervous system, and serves to both support neural metabolism and byproduct elimination.
The Cerebrospinal Fluid

The cerebrospinal fluid (CSF) is produced within all four ventricles. After production, through inherent motion of the brain, and reciprocal tension produced by the surrounding dura, CSF is propelled to the fourth ventricle for circulation to the spinal cord. Through motility of the spinal cord, mobility of the sacrum, and taughtness of the dura, CSF is returned to the brain, and transported via the arachnoid villi to the venous sinuses for drainage into the blood and lymph. This mechanism is supported by bony movement, membranous movement, CSF production, and respiratory motion. In essence, the CSF circulates throughout the central nervous, and serves to both support neural metabolism and byproduct elimination.

The Cerebrospinal Fluid

- The CSF circulation can be restricted by soft tissue tensions, and can be manipulated in situations where the patient’s CSF flow is compromised, to foster more efficient toxin elimination, and restoration of a normal primary respiratory mechanism.

Suggested Pathway for CNS Facilitation or Central Sensitization
Research suggests that patients with chronic pain syndromes possess a regulatory dysfunction with the release and maintenance of excitatory substances, e.g., substance P, excitatory amino acids, and nitrous oxide.

Facilitated Synapse

Pharmacologic Approaches to Treatment
Central Sensitization

• Current understanding of the pathophysiology of chronic pain syndromes, e.g. fibromyalgia is that the disease is a disorder of central pain processing or a syndrome of central sensitivity. The mechanism for opiate addiction is comparable with similar chemical changes over time.

• Central Sensitization has been described as a diffuse problem of sensory volume control that alters the patient's threshold to pain and to other stimuli, such as heat, noise, and strong odors.

• Much like the facilitative process associated with chronic viscero-somatic disorders, the inhibitory mechanism, e.g. the inhibitory effect of serotonin on afferent pain signals, is self-perpetuated with greater sensation of pain and decreased inhibition of pain signaling.

Central Sensitization cont.

• It has also been suggested that patients may have hypersensitivity because of neurobiologic changes that affect the perception of nociceptive pain or because of expectancy or hypervigilance, which may be related to psychological factors, and physiologic changes to the inhibitory mechanism.

• N-methyl-D-aspartate (NMDA) subtype glutamate receptors (within the dorsal horn) react secondary to afferents like substance P.

• However, more recent evidence may suggest that suppression of the normal activity of dopamine-releasing neurons in the limbic system is the primary pathology in fibromyalgia.

• Indications are, that fibromyalgia syndrome does represent a complicated dysregulation of pain neurotransmission without any obvious physical injury.

Probable Pathogenicities of Fibromyalgia & Similarities to Chronic Pain Syndromes

• Central Sensitization
  – Decreased Serotonin Levels
  – Increased Substance P
  – Increased Release of Excitatory Amino Acids
  – Low Levels of ATP
  – Decreased Growth Hormone
  – Hypothalamic-Pituitary-Adrenal axis dysfunction
  – Increased Allostatic Load
  – Increased Release of Nerve Growth Factor

• Failure to Maintain Restful (Delta) Sleep Periods

• Genetic Predisposition
Pathogenesis
“What is Known”

- Currently known abnormalities include:
  - Low serum levels of serotonin
  - 4-fold increase in nerve growth factor
  - Elevated levels of substance P
- All of these abnormalities lead to a whole-body neural dysregulation to pain and suggest a condition of abnormal central processing of nociceptive pain input or central sensitization.

Serotonin

- In fibromyalgia, the most widely acknowledged biochemical abnormality is abnormally low serotonin levels.
- Serotonin is a neurotransmitter linked to sleep, pain perception, headaches, and mood disorders.
- A low platelet serotonin value is believed to be the cause of the low serum levels, which have been correlated with painful symptoms.
- Serotonin levels in the CNS are thought to be low because of low levels of tryptophan (amino acid precursor to serotonin) and 5-hydroxyindole acetic acid (metabolic by-product) in the spinal fluid.
- Investigators have proposed a link between low serotonin levels and symptoms of fibromyalgia.
- Moreover, many propose that low serotonin levels may propagate fibromyalgia in part, because of its role as an inhibitor of ascending pain signals.

Substance P

- Substance P elevations measured as high as 2-3 times the normal.
- Substance P, the neuropeptide in spinal fluid, is a neurotransmitter that is released when axons are stimulated.
- Increased levels of substance P sensitize the receptors within the dorsal horn to propagate and heighten awareness of pain, which may cause “normal stimuli” to result in exaggerated nociception.
Adenosine Triphosphate

• Some research suggests low levels of adenosine triphosphate (ATP) in red blood cells and platelets of patients with fibromyalgia.
• If ATP is necessary to move and then hold serotonin in platelets, then is it significant to suggest that low platelet serotonin levels can be explained if platelet ATP levels are also low.

Dysfunction of the HPA axis

• Some have studied the neuroendocrine aspects of fibromyalgia and found dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis.
• The HPA axis is a critical component of the stress-adaptation response. In a normally functioning system, corticotropin-releasing hormone (CRH) stimulates the anterior pituitary to release adrenocorticotropic hormone (ACTH).
• ACTH then stimulates the adrenal cortex to produce glucocorticoids, which are powerful mediators of the stress-adaptation response.
• Circadian regulation and the stress-induced stimulation of the HPA axis are, in part, regulated by serotonin. Perturbations in serotonin metabolism (as well as premorbid abnormalities of the HPA axis) may explain the abnormalities of the HPA axis in fibromyalgia.

Dysfunction of HPA axis cont.

• Dysfunction of the HPA axis may exaggerate the effects of abnormal serotonin metabolism. Hypoactivity of the HPA axis may cause low central serotonin levels.
• Some authors have noted that 5 main measurable neuroendocrine abnormalities are associated with dysfunction of the HPA axis:
  − (1) low free cortisol levels in 24-hour urine samples
  − (2) loss of the normal circadian rhythm with elevated evening cortisol level (when it should be at its lowest level)
  − (3) insulin-induced hypoglycemia associated with an overproduction of pituitary ACTH
  − (4) low levels of growth hormone
  − (5) stimulated ACTH secretion leading to insufficient adrenal release of glucocorticoids
Dysfunction of the HPA Axis...cont.

- SPECT studies of the brain have noted **regional areas of decreased blood flow**, perhaps secondary to the variable changes to vascular tone.
- Patient subjective complaints of palpitations, peripheral hypo/hyperesthesias are perhaps secondary to HPA dysfunction.
- Associated IBS, urethral irritability and headache, common Fibromyalgia Syndrome complaints are perhaps secondary to HPA dysfunction.
- Nocturnal **elevation of cortisol levels** (compared to normals) perhaps are related to the described *sleep disorder.*

Growth Hormone

- Growth hormone, produced during delta sleep, is involved in tissue repair.
- Therefore, disrupted stage 4 (delta) sleep associated with fibromyalgia may account for low levels of growth hormone.
- Growth hormone stimulates the production of insulin-like growth factor 1 (IGF-I) in the liver.
- Some authors have found that most patients with fibromyalgia have low levels of IGF-I and that *low levels are both specific and sensitive for fibromyalgia.*

Nerve Growth Factor

- In some studies, nerve growth factor was 4 times higher in the spinal fluid of patients with fibromyalgia than in others.
- This factor is important to the pathophysiology of fibromyalgia because the process **enhances the production of substance P** in afferent neurons, increasing the person’s sensitivity or awareness to pain.
- Nerve growth factor also may play a role in spreading or redistributing perceived pain signals.


Allostatic Load

- **Allostatic load** is chronically elevated sympathicotonia with physiological costs of chronic exposure to the neural or neuroendocrine system.
- Allostasis is the initial physiologic adaptation in the face of stressful situations and its stimulation involves activation of neural, neuroendocrine and neuroendocrine-immune mechanisms.
- Often unresolved stressors will maintain the allostatic load, e.g. trauma, infection.

Allostatic Load cont.

- The main hormonal mediators of the stress response, cortisol and epinephrine (adrenaline), have both protective and damaging effects on the body.
- Fibromyalgia patients have increased allostasis due in part to
  - Repeated frequency of stress responses to multiple novel stressors.
  - Failure to habituate to repeated stressors of the same kind.
  - Failure to turn off each stress response in a timely manner due to delayed shut down.
  - Inadequate response that leads to compensatory hyperactivity of other mediators.

The Role of Fascia

“A Role for Osteopathic Manipulative Treatment”

- Fascia supports and lubricates muscle and organs, compartmentalizes the body, and stabilizes the body’s skeletal structures. Fascia maintains the “FORM” of the body.
- **Through fascia, organ systems communicate** via the blood vessels, neural tissue and lymphatic vessels that pass through it.
How Can The Properties of Fascia Serve a Therapeutic Use for Visceral Disease?

- 1. **Fascia follows Hooke's Law, and Wolff's Law.**
- 2. **Fascia is communicative through attachment and investment.**
- 3. **Release of fascial restriction can ease restrictions to motion and enhance biologic function and communication.**

The Respiratory-Circulatory Model Proposed by Gordon Zink, D.O.

- 1. For **“health”** there must be good circulation of the body fluids.
- 2. For good fluid circulation, there must be efficient function of the respiratory processes, “cranial mechanism” and “pulmonary mechanism”.
- 3. The primary respiratory mechanism requires freedom of movement of both the cranial and pelvic diaphragms.
- 4. The secondary respiratory mechanism (thoracic inlet and outlet), is essential to venous and lymphatic flow, and is supportive of the primary respiratory mechanism.
- 5. The alternate **“twisting”** of the four diaphragms of the body is essential for positive fluid flow.

Sequence of Myofascial Lymphatic Treatment by the Zink Concept

- 1. Rib raising to enhance mechanical respiration and inhibit hypersympathetic activity.
- 2. Fascial release of thoracic inlet to decrease restriction of thoracic duct.
- 3. Relax and balance the thoracic outlet (thoraco-abdominal diaphragm), to foster pressure differentials between the two compartments, i.e. enhance lymphatic and blood flow between compartments.
- 4. Balance the pelvis (pelvic diaphragm) to promote lymphatic flow.
Venous Sinus Drainage Technique

CSF Motion through 4th Ventricle

Application of the CV4 Hold to Enhance Lymphatic flow, and Diaphragmatic Motion

Patient is supine with operator facing the patient’s vertex. Palms are crossed over midline of the occiput with left hand over left landmarks of patient and right on right. The patient is asked to inhale, ed fraction is placed on the inferior nuchal line of the occiput. Position is held until release is felt. Promotes CSF flow and parasympathetic expression.
Myofascial Release of Suboccipital Triangle Muscles

Suboccipital release

1. The pads of fingers 1, 2 and 3 of each hand are placed on the dorsum of the neck, immediately inferior to the occipital poles.

2. The patient is asked to lay the back of the head upon the fingers, and allow the weight of the head to exert the total force on the tissues.

3. The physician maintains the position, as the muscles fatigue, and relax, while the fingers palpate deeper fascial (dural) layers.

4. The endpoint of the treatment is a sense of vibration, followed by heat release, felt by the physician, and sometimes by the patient.

Benefits include: Normalization of autonomic tone, drainage of the 4th ventricle, normalization of sacral motion.

Suboccipital release

Benefits include: Normalization of autonomic tone, drainage of the 4th ventricle, normalization of sacral motion.

Cervical Myofascial Release

• 1. Modality is often utilized to treat headache, cervicalgia, sinusitis, and diaphragmatic dysfunction. Addresses thoracic inlet restriction.

• 2. Directly, a steady force is applied longitudinally, until creep is felt.

• 3. Indirectly, the involved segment(s) is/are placed in a point of physiological ease to allow fascial “memory” to find original “form.”

• 4. Treatment directed at C3,4 addresses diaphragmatic dysfunction.

• 5. Treatment directed at OA addresses vagus nerve expression (note proximity to ganglion nodosum).

Myofascial Release of Thoracic Inlet

Improves venous and lymphatic drainage (pre-load) and improves upper rib motion.
Rib Raising

Rib Raising Supine
1. Patient supine with operator standing or sitting at the side.
2. Operator's hands slide under the patient with fingers contact medial to the rib angles and with forearms in contact with the table (Fig. 7.31).
3. Operator's fingers pull lateral traction on the rib angles while lifting the rib cage by pushing down on the forearms. Do not try to lift the rib cage by wrist flexion.
4. Move up and down the rib cage on both sides. If a second operator is available, bilateral rib raising can be done.


Doming of the diaphragm in the respiratory compromised patient is necessary to normalization of the thoracic cage. In the compromised patient functional techniques are best tolerated. Note hand placement by the operator around the anterolateral borders of the diaphragm.

Dome Abdominal Diaphragm

"Doming the diaphragm" is a term used to refer to releasing the resting state of the abdominal diaphragm (or the pelvic diaphragm). If the diaphragm can be completely relaxed and well-defined, its contraction and relaxation produce greater pressure gradients between the thoracic and abdominal cavities, along with the one-way valves, and promote better lymphatic drainage back into the systemic circulation. The previous gesture acts as an extrinsic pump for the lymphatic system. The doming techniques may also directly engage the inferior surface of the diaphragm and augment its excursion during expiration.

Application of Myofascial Release to treat sacral dysfunction (parasympathetics)

SACRAL REGION/MYOFASCIAL, RELEASE

Indication: Somatic dysfunction associated with lower sacral and pelvic pain, muscle spasm, constipation, diarrhea, entero-colitis, and other visceral dysfunctions.

Procedure: 
1. Place flat hands on the sacrum with the bottom hand just above the Douglas pouch noticed.
2. Ask the patient to take a deep breath and then exhale slowly and fully.
3. At the same time slowly lower the sacrum by pushing the pubic symphysis anteriorly.
4. Return to the starting position.
5. Repeat a few times.
6. Evaluate response by noting the sacral fascial pliability and movement of the lower lumbar region.
7. Check that the sacral body is in the position of body weight and its motion is relative to thinness.
8. Guide the sacrum to the direction of less tension and apply steady tensor and sacral motion.

Note: Patient can also be in the supine position, and exerting the force against the physician.

Note: The physician can also incorporate the respiratory drive, and its relation to sacral motion.

Summary

1. The applications of Osteopathic Principles presented here are intended to support the neurophysiology of the central nervous system.

2. The Respiratory-Circulatory Model as presented by Gordon Zink, D.O., FAAO, is a systematic approach to normalize the fascial, lymphatic and autonomic functions of the neurovascular system.

3. Myofascial Release can be used to diminish restrictions in physiologic range of motion.

4. Myofascial Release can be used to ease tensions within sheets of fascia that envelope communicative structures, e.g. blood vessels, to improve system function.

5. Fascial balance and balance of the dural membranes promote efficiency of the primary respiratory mechanism, the venous, lymphatic and arterial circulation as well as the neuroendocrine circulation.

6. Myofascial release is a gentle technique that can be both direct and indirect relative to the motion barrier.

7. Myofascial release and OMT in the Cranial Field are indicated for the patient, whom is immobile, and unable to either actively participate in treatment, or tolerate extremes of treatment positions.

8. Rib Raising is an inhibitory technique intended to normalize sympathetic efferent expression.