Lymphatics
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The lymphatics are closely and universally connected with the spinal cord and all other nerves, and all drink from the waters of the brain.
--AT Still, Philosophy and Mechanical Principles of Osteopathy, 66.

I had an amazing palpatory experience the other day that I want to share with you.

A 40 year-old woman that I had seen for a decade off and on for multiple sclerosis came back with an acute exacerbation. Usually, she responded to treatment and went about her business for months before she would need to have another treatment for headaches or other complaints that seemed to be rather easily remedied with OMT.

Over the years I had learned to palpate some of her MS lesions that were visible on MRI. They appeared in my palpatory gaze as small islands of limitations of normal fluidity within the brain substance or spinal cord. Although my experience with palpating her lesions was inconsistent, there was always one lesion that I could identify in the cord at about the 7th thoracic vertebra. I came to know how her multiple sclerosis lesions felt because I had a “standardized reference” from visit to visit at T-7. The lesion felt like a firm, foreign object, disc-shaped with the flat surface orthogonal to the vertical axis of the cord and lying to the left of center in the dorsal cord, but moving in harmony with the breathing cord.

On this day, she was nearly unable to ambulate because she was so bent over from pain in her chest that also inhibited her breathing. She displayed a lesion at T-7 that to my palpation was now immobile, separating the left side of the cord into two segments, one above and the other below the lesion, both segments out of synch with each other with regard to primary respiration. The dimension of the lesion was larger in both thickness and circumference than before. I concluded her pain was derived from this lesion, because of its location in the inferior sternum at the level of the seventh spinal nerve.

My first thought was to work with the glymphatic system that had recently been discovered and that I had recently learned about. I thought if I could make the lesion more fluid-like, it might be softened or reduced in size by the glymphatic mechanism that serves the CNS as its lymphatic system and clears it of solutes. The glymphatics rely on “convective bulk fluid flow” according to the literature. I thought, “The authors of these articles are puzzled by the origin of bulk flow they are witnessing in the brain substance. Since I know something about a bulk flow and can work with it directly, perhaps I can enhance the glymphatics.”

Of course, cranial manipulation works with “convective bulk fluid flow,” to use their words. Sutherland called this the fluctuation of the cerebrospinal fluid or the “tide.” I hoped it would soften lesions from MS that result from demyelination.

The next thought that came to me as I palpated her mechanism from her head was the radicular artery of Adamkiewicz since it is located at T-7 in most people. I used my old trustworthy tool, querying the body’s primary respiratory mechanism. The PRM stopped when I silently said: “The radicular artery of Adamkiewicz is functioning poorly.” The reverse statement, “The radicular artery of Adamkiewicz is functioning normally” produced a healthy fluctuation, a “Yes.” I asked about the azygous and hemi-azygous veins that are also located in this region; also about any artery or vein and received a “No” – a cessation of the PRM.

Surprised by this information, I went to the next level of questioning: “The lesion comes from arachnoid adhesions obstructing the flow of cerebrospinal fluid.” Obtaining a “No” to this statement, I posed the reverse statement for confirmation. The response was “Positive.”

Having not discovered the cause of the lesion by asking about two fluids, blood and CSF, I wondered what congestion I was feeling anterior to the lesion in the cord. I said to myself, “Am I feeling the lymphatics?” I immediately received an affirmative response from the fluctuation of fluid. What I was feeling was congested and engorged lymph nodes in the region of the lesion in the cord. As I continued to observe, three nodes began to be defined by palpating a lack of localized motility.

Suddenly, I remembered what Frank Willard, PhD said in his lecture about the lymphatics at the last AAO Convocation. He proclaimed something that really surprised me: the lymph node directly returns 35% of the afferent lymph fluid into the venous drainage through the high endothelial venules within the deep cortex of the node. Dr. Willard stated that collectively the lymph nodes return more lymph back to the venous system than the thoracic duct and lymphatic duct, both of which empty their contents into the left and right subclavian veins, respectively.

Then, my mind went to Dr. Still’s declaration about the lymphatics being closely connected to the spinal cord and all the nerves, “and all drink from the waters of the brain.”
Here's where the palpatory experience comes in. I felt the congested lymph nodes (it felt like three of them) and intended that they should be more fluidic. Soon they began to change in size and character, indeed, becoming more fluid like. As they did so, the spinal lesion simultaneously diminished in density and size. Given a few minutes, the lesion actually disappeared leaving behind fluid that continued to divide the spinal cord where the lesion had been.

My intention now changed to integrate the function of the two segments of the cord into one. Soon, I felt the cord breathing as a unit without any separation where the lesion had been.

In the process of all of this, holding the nodes, the CSF and arteries in my awareness, I noticed the breathing of the cord and the fluctuation of the CSF were harmonious with the arterial fluctuations (Traube-Hering waves)\(^1\) at the usual rate of primary respiration (6-10 times per minute), but the fluctuation of the fluid in the lymphatics and veins was slower, at a rate consistent with the long tide (Mayer waves,\(^1\) 1-2 times per minute). These waves have been reported in the literature.\(^2\)\(^,\)\(^3\)

I said to the patient that she should come back next week for follow up. I expectantly waited for her next appointment to see what had transpired. When she walked in she said she was back to work and felt some pain but at a level that was manageable with gabapentin. She was able to sleep better and was thinking about her next project at work, whereas at her last visit she was contemplating what she was going to do with the rest of her life, feeling unable to work at all.

When I examined her, the lesion was evident but at half the density and size at her last visit and was pliable. Importantly, the whole cord was breathing in synchrony with PRM. The lesion responded more readily than the last treatment and resolved again.

Then, my attention went to the whole lymphatic system. Now I knew what the chain of nodes in front of the aorta felt like having experienced their position and consistency at her last visit. I could follow them up the chain to the thoracic nodes and down to the iliac nodes. I could even follow them to the cervical nodes and on into the skull to the dural lymph channels. These lymph channels are found adjacent to the venous sinuses and drain into the deep cervical lymph nodes. I felt a decongestion of the lymph channels in the dura of the tent and falx. This whole chain of nodes and channels drained in sequence and I was privy to it, observing all of this from the head.

What came to my mind was that my palpatory experience showed me that congested lymphatics were responsible for poor drainage of the parenchyma of the CNS and the formation of the lesion within the substance of the cord. Once the lymphatics decongested the lesion immediately responded becoming more pliable and less injurious. I thought, "What is the histology of this mechanism?" A recent article helped answer this question.\(^4\)

I also found two separate reviews of the glymphatic system.\(^5\)\(^,\)\(^6\) In their reviews, we learn that the external surface of the parenchyma of the central nervous system is lined with end feet of astrocytes. As the arteriole penetrates the brain surface from the subarachnoid space, pia mater remains as a coating of the vessel. It forms the interior of the paravascular space while the astrocyte end feet lining the brain surface form the exterior wall. Together, the pia and astrocyte end feet form the blood-brain barrier. As an extension of the subarachnoid space the paravascular space is filled with CSF. The astrocyte end feet contain aquaporin channels that regulate the flow of water into the brain from the paravascular space.

As the arteriole narrows and becomes a capillary the pia and muscular layers of the arteriole are abolished allowing the interstitial fluid (ISF) from the brain parenchyma to mix with the CSF from the subarachnoid space. Capillary permeability permits the exchange of the CSF into the brain parenchyma where it serves its "glymphatic clearing" function. Once the fluid enters the parenchyma, it clears the brain of debris by convective bulk fluid flow and drains into the paravascular space surrounding the veins where it eventually ends up in the deep (retropharyngeal) cervical lymph nodes. If we consider the spinal cord, the same function exists with the glymphatic fluid draining into the nodes near the spine.

The major lesson for me in all of this is that lymph nodes are responsible for receiving the clearing fluids that drain the central nervous system by bulk fluid flow. This bulk fluid flow is a mystery to the physiologists who are not ready to accept Sutherland's hypothesis that bulk fluid flow is generated by the breath of life. If they can't measure it they will not advocate for it. The breath of life is not measurable; bulk fluid flow is. What is the mechanism of this interface? Spirit influences the physical movement of water and transmits information via the water that is critical for health.

**References**

An Osteopathic Approach to Ophthalmic and Optometric Disorders: Part 8

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Upon history taking of Patient 48, a 55-year-old female RN, concerns of possible Multiple Sclerosis (MS) surfaced because of having the recent onset of vague, wandering, episodic neuralgic sensations, including a hard-to-describe, abnormal left periorbital sensation. A left thoracic spine/rib/rhomoid dysfunction also preceded onset of the eye sensation. The OD globe, with hard contact lens left in place, was found to be medial superior CCW, while the orbit and membrane were also CCW. The projection was medial inferior with the Periorbita (bulbar fascia) restricted at 4 and 8 o'clock. Intraoral OMT addressed the ipsilateral zygoma, palatine, maxilla, SPG, frontosphenoidal suture and great/lesser wing intrasosseous strains. Lastly an occipital CV4 followed, due to the regional cranial and systemic nature of the symptoms, and for reasons discussed in preceding articles in this series. Also, recall the nutritive effects of CSF on all tissues including the perineural channels it traverses, for the good of the entire body. “With that consideration we can go back to Dr. Sutherland’s statement: ‘Regardless of the problem, if you do not know what else to do, apply compression of the fourth ventricle.’”217 (For all osteopathic findings, unless otherwise specified, the technique approaches herein involved directly engaging restrictive barriers, the rationale and specifics having been described in earlier parts of this series of articles, as well.) Hypothyroid treatment with whole desiccated thyroid was reinstated. Seen four days later, she stated her left eye felt 60% better. In addition to addressing left thoracic lesioning, her OS globe was now medial CW, orbit CCW and the (suspensory) membrane CW. The orbital projection was medial superior while the periorbita was restricted at 2 and 12 o’clock. The visualy relevant intraoral OMT, as per prior case studies, followed. At three days following this, and improving, her OS globe was now CW while the orbit and membrane were CCW, projection medial superior, and the periorbita was restricted at 3 and 12 o’clock. Intraorally accessed structures were addressed also, in addition to cervical, thoracic and pelvic regions. Around two weeks later her eye pain was 90% improved. Her OS globe was now CCW, membrane and orbit CW, and the periorbita was restricted at 1 and 2 o’clock. The intraoral OMT followed. At the next visit around a week later, she reported total relief of left eye pain. She also reported that a neurological consultation with cervical and brain MRI performed in the interim proved negative for MS. Her ongoing left rib, cervical and thoracic symptoms, improving as well, were addressed with OMT. Total relief of the symptoms followed and she has remained asymptomatic for over two years now.

After her smart-phone camera slipped, Patient 49 (previously Patient 21), a 56-year-old bookkeeper, inadvertently looked at an eclipsed sun with her naked eyes. She presented the next day, with left greater than right frontal area and eye burning pain and visual blurriness, and bilateral eye globe redness. Both eyes appeared erythematous. This was in spite of a dose of homeopathic Phosphorus 200C via phone consult the day of the visit before arriving. After the diagnosis of bilateral photokeratitis was made, local osteopathic evaluation and OMT were performed on the more painful eye (with the aim of reflexively influencing the other eye). Regional techniques to encompass the other eye also followed. (Can include cranio cervical, cranial base and vault area, the region adjacent to the facial globe and orbit, which is referred to as local treatment in these writings.) Her bilateral palpatory scan revealed the left globe was protruded with a decrease in CRI, confirming and prompting attention to the left eye, in light of time constraints. The OS globe was lateral inferior CCW, orbit CCW, membrane CW, projection superior lateral anterior, and the periorbita restricted at 3 and 4 o’clock. OMT for the ethmoid and intraoral visual – related accessible structures was next. After this, she reported her symptoms already improved. An abbreviated VST and occipital CV4, followed by a frontal lift and spread, were performed because of the patient’s concerns from the then reported continuing frontal pain. Sunglass use was advised. Patient concerns over losing vision, a common fear for all eye symptoms which prompts more immediate medical attention, also prompted her to return two days later. After