Analysis and Correction of Locomotor Dysfunction as It Applies to Autonomic Nervous System Dysregulation

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I propose a mechanism by which Lower Extremity biomechanical loss of Plantar Venous Pump drive translates into loss of not only venous return to the right atria, but also loss of kinetic energy required for locomotion.

Loss of torsional movement decreases fluid drive from the lower extremity to the heart, which increases sympathetic tone, leading to a Neuroendocrine HPA Gonadal response.

(The pelvic-thyroid-adrenal syndrome of Owens)

Once the femoral head loses the ability to internally rotate, the pelvis, by Chapman's assessment, produces hyper congestion, a relative left pH shift, and also increases sympathetic tone.

Biomechanical and immunological stress in the system increases the base line sympathetic tone, and when chronic tends to favor a hypersympathetic state, leading to inhibition of the repair side and producing an increased inflammatory response.

This is the beginning of chronic adaptation that lends itself to development of chronic inflammatory disease processes.
The sympathetic nervous system and neuroendocrine systems are best described as regulators of protection of body tissues during challenges arising internally or externally to the body.

These systems serve to adapt organ functions to behavioral responses in threatening environments, and to perceived stress of any kind.
Tenants of neurological control developed by Dr Loren Rex DO

Ursa Foundation, Ganglia Series-1996 class notes

Remember:

Ganglia - provide good regional information

Plexus - give fine local control
The adaptation of the immune system, control of inflammation and control of the nociceptive system (related to pain and hyperalgesia) by the CNS, all require a sympathetic system which functions in a differentiated way.

During real or impending tissue damage, this integrated protective system organized by the Hypothalamus is strongly activated, leading to protective illness responses including pain and hyperalgesia, and other aversive sensations.

During fast defense, organized by the hypothalamo-limbic system, fast analgesia, mobilization of energy, activation of the sympathetico-adrenal system and activation of the HPA axis occur.

This fast defense is preferentially activated from the periphery by stimulation of nociceptors of the body surface and is accompanied by an increase in arterial blood pressure and heart rate, and an increased vigilance and alertness.

Heart Rate Variability is an effective measure of these changes.

Currently, Neal O’Neal, PT, Director of Research at The Ursa Educational Institute for Manual Therapy, is overseeing a research project to not only measure the changes in sympathetic-parasympathetic balance but also response to manual treatment of peripheral resistance, such as is seen in Lower Extremity loss of fluid drive that leads to aberrant sympathetic tone.
During slow defense, the body switches to recuperation and healing of tissues.

This slow defense system is activated by peripheral signals mainly in afferent nociceptive neurons from deep body tissues (deep somatic tissues, viscera) and from the immune system by cytokines via afferent vagal neurons. It is accompanied by decreased arterial blood pressure and heart rate.

The involvement of cytokines in sensitization of nociceptors during inflammation, is via the terminals of sympathetic fibers and/or the activity of the sympathetico-adrenal axis.
The anatomical evidence showing that the sympathetic nervous system, but not the parasympathetic nervous system, is involved in control of the immune system, is overwhelming. This applies to the primary and secondary lymphoid organs.

Primary and secondary lymphoid tissues are innervated by postganglionic noradrenergic sympathetic neurons.

Varicosities of the sympathetic terminals can be found in close proximity to T lymphocytes and macrophages, as described for other sympathetic target cells.

The SNS also exerts complex control on joint inflammation. Along with the dorsal root reflex, it is a critical proinflammatory component of neurogenic inflammation.

Sympathetic outflow can participate in the **chronic phase** of arthritis and can exhibit both pro- and anti-inflammatory actions.

The duality of this system can be explained in part by the fact that the SNS innervates the joints and the secondary lymphoid organs.

**Not tertiary.**

An example:
The knee joint is densely innervated by sympathetic postganglionic neurons.

This innervation is involved in regulation of blood flow and in regulation of synovial plasma extravasation, either directly or indirectly in association with leukocytes and other cells related to the immune system.

Neuroplasticity of Sensory and Sympathetic Nerve Fibers in the Painful Arthritic Joint.

About 60–70% of ongoing and bradykinin-induced synovial plasma extravasation is dependent, in physiological conditions, on the presence of the sympathetic innervation of the joint (but not on activity in these neurons and not on norepinephrine released by them).

The extravasation is modulated in an inhibitory way by epinephrine released by the adrenal medulla.

Ectopic lymphoid follicles may result from the accumulation of dendritic cells that are activated locally and **congregate at sites of lymphatic drainage.**

This is why “Drain the Tissue” techniques and Chapman’s Neurolymphatic Techniques continue to be powerful tools for patient treatment.

In Dr. Chapman's book, Dr Owens states that a leg that does not internally rotate is a sign of pelvic congestion and therefore a sign of endocrine dysfunction (pelvic-thyroid-adrenal syndrome).

Loss of Lower Extremity minor motion predisposes the patient to increased sympathetic tone.

**And low grade inflammatory sustained response.**

I am proposing a mechanism to explain the formation of tertiary lymphoid tissue at sites of inflammation or infection in normal individuals.

With chronic low grade inflammation, this leads to rerouting of the lymphatic drainage towards newly formed tertiary lymphoid tissues.

As a result, the developing lymphoid tissue is maintained by the continuous influx of antigen, which leads to sustained local immune activation.

This lymphoid tissue is no longer under the HPA-SNS control.

In order to appreciate the lower extremity and pelvis correctly, they need to be considered as a major fluid pumping station of the body, where **torsional movement** is the key.

We need to see the LE/Foot complex as a part of the Upper Extremity driving force.
Think of the Great toe, Midfoot, Ankle Mortice, Femoral Head and Pelvis, as levers attached to a hollow cavity filled with organs, that is aided in fluid drive by the action of certain muscles (Iliopsoas, Diaphragm, and Pelvic floor) as an agitator for fluid drive (electrochemical gradient potential), and immune response and function.
“In many respects, the foot remains the last unknown link in the lower limb chain. We understand the movement of the four major articulations in the foot (the Ankle, Subtalar, Midtarsal and Metatarsophalangeal joints). However, we know little of how the small midfoot joints move and how the musculature within the foot acts.”

Tissues are multifunctional. However, sometimes this is not well recognized, if at all.
So if gait is still somewhat of an ambiguous issue, then applying it to immunology must be downright heresy.

**Motto** – Movement is life and stillness is death, so the stiller you are the deader you are.

Leading to this lecture on biomechanics and immunology: fluid drive, stress reaction and adaption.

**What is the tipping point?**
One of the big questions is whether movement-induced changes (above 1.9 METS) in innate immune function alter infectious disease susceptibility or outcomes, and whether the supposed anti-inflammatory effect of regular exercise is mediated through exercise/movement-induced effects on innate immune cells.

We need to know if movement alters migration of innate cells and if this alters disease susceptibility, both pro and con.

Minor motion loss plays a big part in this mechanism.
What most Allopathic practitioners look at is the **drive side** (arterial) of fluid dynamics. But the return side is what keeps us clean (repaired).

Arteries do not need help, having an elastic wall. Veins on the other hand, need biomechanical assistance in order to drive fluid, not only of venous blood but also to aid lymph returning into the pelvis from the lower extremity.

**Rhythms matter.**
So let's look at this in a new and different light.

Around 1890, F. Lejars was the first to describe a venous pump activated by walking:

The Plantar Venous Pump.

He described large superficial vessels which form a true plantar reservoir. He almost got it right.
Then 100 years later Gardner and Fox proposed a hypothesis which stated that it is the stretching of the medial and lateral plantar veins which, with each step, pushes the blood into the saphenous veins and the deep venous network, and that the pump of the foot and the calf function sequentially.

Now they got it right.

Gardner AM, Fox RH. Peripheral venous physiology. In: Gardner AM, Fox RH, editor

Non-valvular veins of the cutaneous network visible in the totality of the sole of the foot.

This is not the important part.
The pump (shown in green) comprising the plantar veins is polarized and contains 3 parts going from distal to proximal: a suction pole (A), a reservoir (R), and, an ejection pole (C) the calcaneal confluent.

B: relations with the weight-bearing area.

Red circular patch = weight-bearing area on the ground.

Blue circular patch = manual compression area of the plantar veins.
At A, blood enters the pump during raising of the foot into plantar flexion on relaxation of the plantar muscles: Plantar quadratus and abductor hallucis
The intramuscular veins that are part of the PVP system are concentrated in the soleus, medial head of the gastrocnemius, and vastus lateralis. These veins act as a blood reservoir.

Contraction ejects the volume of blood and muscle relaxation allows filling of this reservoir.

But what if the mechanics are dysfunctional, or what about high resting muscular tone?
All the deep veins of the calf join to form the popliteal vein which is the calf pump outflow tract.

The calf muscle pump generates systolic pressures of 200-300 mmHg.

The foot pump also plays an important role in venous return in that it contains a pump powerful enough to propagate a column of blood to the right atrium.
So, in looking at foot biomechanics, not only are we dealing with harnessing kinematic forces on the lateral side, but we are also looking at vascular supply and demand on the medial side. IT Band tightness may have a different etiology, and in fact is only present if the system becomes inefficient.

Loss of foot function leads to increased cost of doing work, which in turn makes us much less efficient, and leads to a reduced recovery potential.

The first metatarsal interspace (1\textsuperscript{st} Ray) \textit{vein} gives rise to the greater and lesser saphenous veins.
Three phases can be described during walking:

The weight-bearing phase:

Contact of the foot on the ground produces direct compression of the reservoir in the sole of the foot between weight-bearing areas.

When a subject goes from the seated to the standing position, under the influence of gravity, the weight of the column of blood exerts a pressure of about 80mm of mercury.
Three phases can be described during walking:

The impulse phase:
Weight bearing on the forefoot with flexion of the toes which fix the foot on the ground, resulting in compression of the pump in the musculotendinous plane by muscle contraction.

After a certain number of steps (about 10 - 25 m), ankle pressure falls to 30mm of mercury.

This decrease is related to mobilization of the volume of blood, due to activation of the different venous pumps in the lower limb during walking.
Three phases can be described during walking:

The suspension phase:

The foot is lifted off the ground, allowing filling of the pump.

During walking, the pump reloads cyclically as the foot is lifted up and empties as weight is applied.
This venous pump of the human foot is the first part in venous return from the lower extremity to the heart.

Flexor digitorum brevis, quadratus plantae, abductor hallucis and the flexor digiti mini brevis are all involved.

Contraction of these muscles compresses and empties the lateral and medial plantar veins of the foot reservoir.

Could loss of this be the beginning of plantar pain, or the beginning of pelvic congestion?
The plantar venous pump is the only one effective up to the calf, where its action is taken over by the calf pump of the soleus muscle.

Its dual action, on both the deep and the superficial saphenous vein circulation, underlines the unimpeded circulation of blood between the two vascular compartments.

In fact, the perforator veins of the foot have do not have any effective valves.

The calf pumping mechanism, produced by contraction of the soleus and of the gastrocnemius, then takes over once the fluid has left the true foot.

The soleus contracts when the center of gravity passes in front of the knee joint.

It arises from the fibular head, 25% of the proximal posterior surface of fibula, and the middle 1/3 of the posteriomedial boarder of the tibia.
The Achilles gets more fibers from the **soleus** than the **gastrocnemius** in forming the actual tendon.

During their descent, the fibers of the Achilles tendon internally rotate 90° so that the posterior fibers of the soleus insert into the MEDIAL aspect of the Achilles footprint, and the gastrocnemius (initially anterior) inserts LATERALLY.

Torque at the foot is maintained by torque at the Achilles.

**This is a spring ligament.**

**1 of 3 in the body.**
Large fat pads are particularly prominent immediately deep to the patellar and Achilles tendons, and hip joint.

They are associated with synovium, are richly innervated and vascularized, and are likely to serve as **mechanosensory organs for tendons** and be implicated in tendinopathies.
Continuing on in this light, let’s discuss hypomobility of the 1st ray complex, not only from the biomechanical perspective but also from how it applies to fluid drive, connective tissue irritation, or muscle injury.
At heel strike loads from below and above are transferred from talus backward to calcaneus, and then forward to navicular through the cuneiforms, the third bearing the most importance, and finally, the 2nd ray.

The talus is a load transferring bone not a load accepting bone.

The third cuneiform then is the keystone for the arch.
The lateral longitudinal arch is related to the **cuboid** and the hip abductors, namely **gluteus medius and gluteus minimus**.

The medial longitudinal arch is related to the **navicular and psoas muscle**.

**Third cuneiform depression - most often dysfunctional.**

The result to the force couple is that the mid row loses minor motion.
Things I keep in mind:

If you have a **gluteus medius problem**, you have a **pelvic floor problem**.

**Pudendal nerve:** Reproduction of pelvic floor pain.

**Obturator trigger points:** Reproduction of lower extremity pain.

**Obturator Internus Muscle:** Can refer dull ipsilateral ache, golf-ball-in-the-rectum sensation, pain to the coccyx, hamstrings, posterior thigh, urethra, vagina, and vulva (important in vulvodynia).

**Obturator nerve:** Pain into thigh.
Posteriorly, the Biceps Femoris long head tendon has a distal **fascial expansion** to the lateral gastrocnemius and soleus muscles.

The action of tibialis anterior and **biceps femoris** are coupled to load the longitudinal fascial network, which serves to store energy and then transmit the energy.

The lateral knee is part of the force drive mechanism, along with Vastus Lateralis.
Deep and posterior to the deep and superficial layers of the ITB, the capsulo-osseous layer emerges from attachments at the lateral head of the gastrocnemius and short head of biceps femoris.

These attach medial and distal to the deep layers of the ITB.
Biceps femoris starts to contract just before heel strike.

At heel strike the fibula drops inferiorly to make the ankle mortice deeper, and loads the biceps femoris **eccentrically**.

The **fibula** is responsible for the **locking and unlocking** of the knee and ankle.

**Posterior motion** unlocks the mechanism and **anterior motion** locks it.

The fibula has reciprocal motion, such that if the top unlocks, the bottom locks, and vice versa. Loss of the syndesmosis creates big problems.
Vastus lateralis contraction pushes iliotibial band laterally, further loading the lateral leg.

This is extremely important for energy conservation and transfer from the lower extremity to the upper extremity force drive.

Also tell me about the venous pump at this time?
Sacrotuberous Ligament is continuous anteriorly with the membranous falciform process which connects with the obturator internus fascia, and gives partial attachment to the piriformis.

Phylogenetically, the sacrotuberous ligament is considered to be the degenerated tendon of the origin of the long head of biceps femoris, however this does not mean that these two structures are entirely continuous.
In the lower thoracic region, fibers of the *serratus posterior inferior* muscle and its fascia fuse with fibers of the deep lamina of the *Tensor Fascia Lata*, and is continuous with the sacrotuberous ligament.

Serratus posterior inferior is not a respiratory muscle (primary).

It functions synergistically with the ipsilateral iliocostalis and longissimus for rotation (unilateral) and extension (bilateral) of the spine, plus fires with QL synergistically.

These are proprioceptive muscles.
The ligamentum teres may play a role in nociception and coordination of movement.

It has type IVa receptors and transmits afferent somatosensory signals. These have been identified in both normal and dysplastic hips.

It is postulated that the ligament may play a role in proprioception in patients with arthropathy and that it acts as a stabilizer to prevent excessive movement.

The ligamentum teres helps distribute synovial fluid within the hip joint by way of the “windshield wiper” effect.
To illustrate, consider the system under an adapted biomechanical situation:

Let’s say the rear foot is non-adaptable, there could be many reasons for this. The centre of gravity will shift lateral and caudad in the spine.

A decrease in tibial advancement will result in a failure of all subtalar joints to engage properly.

This lack of engagement will prevent calcaneal eversion, causing compensatory hyperpronation of the midfoot.

The rear foot will remain in a neutral position.
Accounting for the way the foot has to move, and not being able to accommodate through the midfoot, this produces a failure of the converging axes of the transverse tarsal joints to approximate correctly.

Therefore the self-locking mechanism of the calcaneocuboid joint fails to engage.

This lack of engagement leads to a failure of the windlass effect of Hicks.

Toe off becomes dysfunctional with a subsequent lateral migration of the weight bearing surface of the foot, and so the plantar venous pump system becomes incompetent.
The patient may present with musculoskeletal complaints, in particular non traumatic knee pain.

When the patient has non traumatic knee pain, examine the foot ankle complex and the hip complex.

If no problem is found examine the ninth thoracic vertebra.

The consequences of the above resultant adaptive biomechanical dysfunction leads to increased cost, and subsequently, to inappropriate immune response.
Figure 1. The Neuroimmune Regulatory Network.
This figure shows the interaction of major systemic neuroimmune regulatory pathways with local paracrine-autocrine regulatory circuits. Some key organs are also shown with their major regulatory input. Each organ/tissue has its local circuits, which interact with the systemic network via innervation, hormones and cytokines. Mobile elements of the immune system home to all organs and tissues and participate in physiological and pathophysiological processes. Neuroimmune regulation is fundamental to the development and function of all cells in the body from conception till death.
There is no difference between biomechanical function, fluid drive, sympathetic load, SNS/PNS balance (HRV), and the health of the organism as determined by the HPA system.

I have attempted to paint a picture by which Lower Extremity biomechanical adaptation, then loss of Plantar Venous Pump drive, translates into loss of not only venous return to the right atria, but also loss of kinetic energy required for locomotion.

Once the femoral head loses the ability to internally rotate, the loss of torsional movement decreases fluid drive from the lower extremity to the heart.

The pelvis, by Chapman's assessment, produces hyper congestion with relative left pH shift within the pelvic cavity, and a chronic increase in sympathetic tone, leading to non alternating Neuroendocrine HPA Gonadal response. **(The pelvic-thyroid-adrenal syndrome of Owens)**

Biomechanical and immunological stress in the system increases base line sympathetic tone, and when chronic this tends to favor a hypersympathetic state, leading to inhibition of the repair side and producing an increased inflammatory response.

I will explain this further utilizing the lab sessions this afternoon.

Thank you.