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Chronic Problems Related to Gall Bladder Dysfunction / Disease, Part 3

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In Part II of this series I invited you to consider how the progression of dysfunction / disease of the gall bladder may:

a. participate in the progression toward diabetes

b. reduce the efficiency of the gastrointestinal processes of assimilating nutrients and eliminating waste by effecting the physiologic functions of the pancreas and the small and large bowel

c. slow or restrict venous and lymphatic drainage back to the heart creating a bog of congested fluids beneath the respiratory diaphragm(1).

My clinical experience suggests that progressive venous and lymphatic congestion is a significant contributing source of our clients' various somatic complaints and pain, especially when these have become chronic. Facilitating the rate and volume of these fluids in their return to the heart is a significant contribution to the healing process of every client. Understanding the web of why, where, and how the body typically adapts is the purpose of the Inside-Out Paradigm.

Many years ago Deane Juhan offered our profession an elegant analogy; conceive of the fluids below the diaphragm muscle as the Agricultural South returning blood and lymph to the Industrial North for reprocessing. In the ecology of the body venous blood and lymph are necessary raw blood products for the cardiopulmonary system to generate newly oxygenated and nutritious blood (2). If the rate of flow or the volume of these fluids is insufficient to the demand, what's the body going to do?

The body will either endeavor to find alternate routings back to the heart or it will endeavor to push the blood through the thousands of miles of arteries and veins with more push and speed to maintain an adequate supply. The rate of return and the volume of return (the amount of fluid available for processing) are both significant variables for the cardiovascular system to maintain its efficiency. "The important factor determining the amount of blood pumped by the heart is still the rate of entry of blood into the heart."(3)

The heart typically receives most of its venous blood from below the diaphragm muscle via the inferior vena cava while venous blood from above the diaphragm flows into the superior vena cava. However, when the portal or the caval system below the diaphragm is impeded, as would be the case with an inflamed gall bladder, the body selects to route its southern venous return through the superior vena cava.

Most of what has been written about alternate venous routings back to the heart is based on pathological conditions, liver disease such as cirrhosis or cardiac failure. References use the language of impeded or obstructed blood flow of the portal vein and/or the inferior vena cava. (4,5) Under normal circumstances the entire abdominal gastrointestinal tract drains toward the liver by means of the portal system and then into the inferior vena cava(4). Given the portal vein's intimate juxtaposition and fascial connection to the gall bladder it is exquisitely vulnerable to any distention or inflammation of the organ.

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The alternative venous return pathways that nature has provided are both deep and superficial. The deeper alternative pathway includes the azygous, hemiazygous, accessory hemiazygous veins, and the vertebral venous plexuses(5). The more superficial routing includes, a complex anastomosis (connection of two or more vessels) between the upper abdomen including the lower esophagus, the pelvic floor, those associated with the veins of the abdominal wall that communicate into the bilateral thoracoepigastric venous channels. These eventually drain into the axillary veins which become the subclavian veins(4,5).

In this article we will explore the deeper venous pathway depicted by Figure 1 and 2. Additional pictures of these routings to more fully appreciate their significance are listed in the reference section of this article(6).

In viewing Figure 1 which depicts the deeper alternate routing for venous return from the "inside-out" I encourage you to consider that congestion within this internal scaffolding is a contributing factor to the back pain and stiffness of your clients. Allow your kinesthetic memory to engage Figure 1. Reflect on how frequently you palpate muscular contracture and spasm along the erector muscles on a daily basis. It's pretty amazing how it is a mirror image of where many or most of our clients report that they experience somatic difficulties a fair amount of the time.

Next relate Figure 2 to the effects of congestion of the azygous vein to the frequency you palpate muscular tension associated with the upper right quadrant of the back and the right shoulder. Please consider from this moment forward to begin translating your palpation of external soft tissue tension as an indication of internal fluid congestion.

The deeper venous pathway drains into the superior vena cava located in the upper right quadrant of the thoracic cavity. The azygous vein's typical anatomical routing is through the aortic opening of the diaphragm into the chest cavity traveling up along the right side of the spine to the heart. The thoracic duct also typically passes through the aortic opening moving lymph upward toward the left subclavian vein.

In contrast, nature's plan for the hemiazygous vein has selected for it to find its way into the thoracic cavity by piercing the right crus of the diaphragm and traveling up the left side of the spine with its venous return crossing the midline to join the azygous(4). The accessory hemiazygous vein originates to the left of the vertebral column in the upper thorax and descends downward draining blood from the 4th - 8th intercostal spaces and sometimes the bronchial veins. Commonly it crosses between anterior to the 7th – 9th thoracic vetebrae and also contributes its fluids into the right-sided azygous vein(5).

What's crucial for us to consider is that this venous complex already has a primary job. This is to drain the posterior body wall above the diaphragm via the intercostal veins, vertebral plexuses and sometimes, some or most of the lumbar veins. Consider the possible contribution to the back pain (upper, middle, or lower) of your clients when these vessels are doing double and triple duty. Let us remember that pain receptors are primarily activated when soft tissues are stretched or compressed. Internal congestion equals external tension and often pain.

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An implication to heart function emerges when one considers that the descending aorta typically shares the aortal opening of the diaphragm with the azygous vein and the thoracic duct. It is a reasonable speculation to wonder about the possible effects of progressively increasing venous and lymphatic congestion upon the flow of arterial blood through the aorta. Appreciating the incredible distensibility of the venous and lymphatic vessels there certainly is a threshold beyond which the body's homeostatic responsiveness would trigger the heart to pump with more force to overcome this initial resistance.

Let us now consider the possible effects of an inflamed gall bladder upon the lymphatic system in the cisterna chili and its superior tributary, the thoracic duct. The same variables apply, rate of flow and volume, and the vulnerability of the confluence of the inferior lymphatic trunks to gall bladder distention or inflammation. Another interesting correlation exists as the thoracic duct crosses from its right-sided vertical routing along the spinal column to the left side of the column somewhere between the 5th & 3rd thoracic vertebrae almost dead center between the scapulae. Consider how often your clients report tension, discomfort, or pain between the shoulder blades. Internal congestion equals external tension and often pain.

I have wondered for years why nature had selected for 80% of the body's lymphatic fluids to drain into the left subclavian vein. It is my discovery and postulation that there is a relationship in symmetry and just plain functional hydrostatic balance since the superior vena cava may be called upon to receive potential overflow from or, become the primary alternate routing from the portal and inferior venous systems on the right side.

Now, let's briefly consider the other obvious alternative for the body to assist venous drainage which is to drive the blood more forcefully from the heart in order to make it through the "60,000 miles of vessels." These are quotes from National Geographic. "The human heart beats 100,000 times a day, propelling 6 quarts of blood --- 20 times the distance across the US coast to coast" (7). It is the sympathetic division of the autonomic nervous system that stimulates the heart to function as a stronger pump. It also stimulates the peripheral vessels to contract (narrow) thus, increasing the speed of blood flow. The bad news is that it also increases the resistance to venous return. (2)

It's long been known that various heart ailments are accompanied by the enlargement of the left ventricle in response to working harder to push arterial blood more forcefully from the heart. With even more acclaim, high blood pressure is now considered to be the axis of evil. However, let's consider that what starts out as a short-term adaptation to periodic or episodic stress becomes a necessary evil to keep the overall system flowing. This is when the grinding and degrading effects may subtly and perniciously trigger progressions toward pathology. An additional learning emerged in researching this article that the Tricuspid valve of the heart acts as a primary feedback regulator of pressure for the heart(3). The tricuspid valve is normally found just below the lower right end of the sternum close to the attachment of the 5th rib. It receives the blood from the inferior vena cava into the right atria. This relationship was very validating of my clinical experience with clients as so often when palpating the degree of thoracic pressure along the intercostal space between ribs 5 and 6, there is line of horizontal tension that bridges across the mid-line of the anterior chest wall. I have come to correlate a softening of this tensional line coupled with greater ease of compression throughout the chest wall as therapeutic indicators that venous return has been facilitated. This also brings us back to

the importance of assisting the body to maintain appropriate pressure differentials between the thoracic, abdominopelvic, and cranial cavities (8). It is intention that matters, not technique.

Next, let's explore the possible relevance of gall bladder/pancreatic dysfunction and venous congestion to the high frequency of hiatal hernias and gastric reflux problems. The most common types of hiatal hernias are sliding and para-esophageal hernias. They both occur on the left side of the hiatal junction between the esophagus & the stomach. The common reason stated is that right-sided hernias do not occur is because the weight and presence of the liver essentially protects this side. (4)

What first became curious to me was noticing that the right crus (leg) of the diaphragm muscle crosses the body's midline and wraps around the hiatal junction. Over many years my hands have correlated gall bladder difficulties and contracture of the right hemi-diaphragm. I began conceiving of this junction as a gasket seal that, when pulled to the right by the crus of the diaphragm, would disturb its competence to close properly (re-sealing the gasket), thus becomes a possible contributing variable of gastric reflux. This seemed to make sense all by itself, then I realized that there were probably additional variables at play.

One variable that has clinically shown itself to be related has been contracture of the lessor omentum which crosses the mid-line from left to right(9). These distensible ligaments have a fan-like appearance with their moorings attached between the stomach, the liver and gall bladder, and the duodenum. It is my postulation that inflammation of the gall bladder or pancreas as was described in the last article may stimulate a contraction of these fibers not only to the right side but also downward, further weakening the upper left portion of the hiatal junction(1). Let's remember that the duodenum cradles the head of the pancreas while the curvature of the stomach cradles its tail.

The most significant variable to hiatal incompetence is to recognize that when either the portal system or the inferior vena cava flow is compromised, the esophageal veins and the left gastric vein participate in both the deep and superficial alternate routings for venous return. The bottom line is x if it can't get through, it backs upx and it does so through the hiatal junction. I speculate that such congestion additionally weakens its capacity to function as a competent gasket. This is similar to my earlier speculation regarding the azygous vein and thoracic duct sharing the opening with the descending aorta.

In summary, please remember that these descriptions, based on my clinical experiences of what has helped my clients most consistently, are not immutable facts. Anatomy reflects the full spectrum of the genetic continuum.

Yet, do consider the possibilities postulated here: that one of the major sources of our clients' chronic somatic complaints may indeed be related to the subtle & insidious effects of progressively increasing venous and lymphatic congestion; that we may contribute at a much more foundational level to our client's health than we realize by intending to facilitate the systemic return of these fluids; and that gall bladder dysfunction/disease may be a possible tipping point in the progression of many disorders.

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The essence of anecdotal clinical observation is to extrapolate possible connections & relationships not necessarily to confirm specific conclusions. To hold the many possible relationships between physiologic function, anatomical structure, and human consciousness simultaneously is the essence of the Inside-Out Paradigm©.

In the final installment, we will connect these relationships to headache patterns including migraines and will speculate on how the existential questions of life may be contributing factors to the progression of gall bladder dysfunction/disease.

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