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Chronic Problems Related to Gall Bladder Dysfunction / Disease, Part 2

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In Part I of this series I encouraged you to hold in the forefront of your awareness the implications of the gallbladder's anatomic centrality, its tendency to become chronically inflamed, its capacity to subtly progress in dysfunction toward disease over many years, and to refer your clients when you suspect its relevance to their somatic complaints. In this article you are invited to take a more speculative leap into functional physiology.

What is so important about gall bladder dysfunction along its continuum of progression is that it potentially can simultaneously reduce the efficiency of the gastrointestinal tract and slow the venous and lymphatic drainage back to the heart from all parts south of the diaphragm muscle.

Nature has provided the human gastro-intestinal (GI) tract with two important aids to assist its crucial activities of digestion: assimilation of nutrients and the excretion of wastes. These aids are bile from the liver/gall bladder complex and pancreatic juices and enzymes. These fluids share a tube, the Common Bile Duct, which ends in the duodenal portion of the small intestine. I encourage you to seek out an anatomical drawing of this relationship between the liver/gall bladder, pancreas, and duodenum. (1)

Consider what might happen down-line within the rest of the length of the small and large intestine should these digestive aids be omitted or significantly reduced. What effect might this have on our ability to assimilate nutrients and eliminate waste? Also, consider what may happen to the functioning of the pancreas should its juices back-up within the organ.

In most medical textbooks, you will find the two most correlated conditions associated with pancreatitis are the progressive effects of alcoholism and the presence of bile sludge or small gall stones that occlude or reduce the capacity for bile and pancreatic juices to reach the duodenum through their common opening, the sphincter of Oddi. (2)

When I first read this many years ago, my hands tingled in a composite kinesthetic memory of thousands of clients and a central question emerged: what effect might the backflow of pancreatic enzymes have on the overall function of that same organ? Most of the clients that I remembered by face had reported blood sugar irregularities including some officially diagnosed with diabetes. Checking my clinic notes of these individuals there appeared a constellation of impressions of what might happen inside the small intestine and the large bowel as food transits the GI tract with little or no bile and pancreatic enzymes.

Immediately, I began to wonder how this might be related to what I had repeatedly experienced as a generalized congestion within the abdomino-pelvic cavity: food going through and down while simultaneously blood and lymph needing to return upward to the heart. There seemed to be a connection.

Researching further I was gratified to discover that the backflow of pancreatic enzymes into the organ had been identified in the general medical literature as one of the contributing factors in the emergence of diabetes mellitus. The literature also confirmed my speculation that chronic pancreatitis occurs more commonly than most practitioners realize. (4,5) Pancreatic stone formation is also possible as a result of such chronic inflammation (5). I would further propose that chronic inflammation can eventually spreads throughout the remaining length of the GI tract.

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Let's remember that all itis's infer inflammation. When an organ is inflamed it occupies more space and may influence the rate of flow not only within its own vascular tubes but also its neighboring low-pressure lymphatic and venous vessels. It is also possible for the entire pancreas to go into relative states of contracture or even spasm.

We would all feel an acute spasm, yet nature did not endow the smooth muscle of the cardiopulmonary organs, gut tube or the urogenital organs with the same broadband of sensory nerves as it did for the musculoskeletal system and the skin. This is the basis for my postulation that many organ dysfunctions, along the continuum of progression toward pathology, often go unnoticed until a critical threshold is reached.

"Many visceral ailments cause no other signs except referred pain. The brain does not know from firsthand experience that the different organs exist and therefore, any pain that originates internally can be localized only generally." (2) 'There is no high-grade sensibility in smooth muscle fibers and inflamed abdominal viscera are not necessarily tender on palpation.' (3)

I propose that the body uses its complex neural net to both express and distribute its internal tensions to the musculoskeletal system as has been described in previous articles. The body is signaling from the inside-out that something needs attention. Let's consider that many of our clients who come to us with chronic somatic complaints reflect the early stages of such physiological progressions long before they could be identified clinically by medical testing procedures.

Continuing with the same example of a partial or complete obstruction of the sphincter of Oddi which prevents both pancreatic and bile fluids from reaching the duodenum in a timely fashion, what are the implications for small intestine's absorption of nutrients? Might this be correlated with the commonly seen swelling along the length of this organ within our bellies as the small intestine attempts to create more surface area in order to do its job of assimilating nutrients without the needed raw materials for digesting fats, proteins, and carbohydrates?

My research also revealed that bile salts are considered to have bacteria reducing properties, thus "bacterial concentrations in the small bowel increase with lack of bile salts."(5) This may be one of the mechanisms by which inflammation spreads throughout the rest of the GI tract.

Many clients have brought me their films of Barium swallows from upper and lower GI medical testing. In composite, it is common to see portions of the small intestine dramatically narrowed while others are expanded along the many feet of its length. What is happening neurologically at these narrowed portions of the tube? What are the effects of a substantially reduced rate of flow? What are the effects upon the complex web of physiology and internal homeostasis? Might seemingly unexplained weight loss be correlated to this or, for others, part of their subconscious drive to grab for more food, resulting in additional weight gain further straining the function of the pancreas? Many nutritionists have proclaimed we are a nation of malnourished individuals despite our agricultural plenty.

My next speculation proposes that as the small intestine struggles to assimilate nutrients, its normal rate of transit becomes variable influencing the timing and full expression in amplitude and force of peristaltic waves is effectively reduced within the large intestine from the cecum to

the anus. The timely discharge of waste also becomes variable instead of regular. Swinging between constipation and diarrhea is much more of a weekly reality than most people are willing to notice, much less admit.

Now, let us shift our attention more specifically to the venous return system from the lower extremities and the pelvic floor. According to Dr. Barral, the developer of the Visceral Manipulation approach, the venous blood returning from the left lower extremity has a slightly longer routing back to the heart than does the right side venous return. (4)

My clinical experience over 20 years since first beginning my study with Dr. Barral corroborates this vascular asymmetry and has correlated left leg/foot edema, left hip problems and the presence of hemorrhoids as part of the profile for the progression of gall bladder dysfunction/ disease (5). In fact, I've lived it.

Two years ago I experienced a gall bladder episode in response to multiple family illnesses only to look back over the previous year to remember that I had had the first hemorrhoid in my life six months prior to this episode coupled with occasional bouts with left hip problems and minimal edema in my left calf over many years. My more acute episode reflected Dr. Barral's additional clinical postulation that the gall bladder is the most reactive organ to emotionally charged events outside of the brain and spinal cord. (4)

In researching anatomy books to explore the venous drainage of the pelvic floor there is common agreement that "any obstruction in the return flow toward the portal system causes veins to become varicose" contributing to the development of internal & external hemorrhoids. Another anatomical fact is that many of the rectal veins do not possess the valves that most other veins do thus they are more susceptible to the effects of muscular straining during defecation (6).

However, it is my postulation that relative states of visceral contracture and/or chronic inflammation as they relate to the sigmoid and transverse colon, the small intestine, the pancreas, and the gall bladder have a cumulative effect that contributes more significantly to the development of hemorrhoids. Muscular straining just pops the already overfull and overstretched venous vascular walls.

Let's remember that the gallbladder sits in fascial communication with the portal vein of the liver, just anterior to the transverse colon and in approximation to the abdominal confluence of lymph trunks, often referred to as the cisterna chili. The gall bladder is the gatekeeper to venous and lymphatic drainage back to the heart. The net effect below the muscular diaphragm is the creation of a bog of swollen and constricted tubes, a damming of venous and lymphatic fluids.

Does everyone have gall bladder dysfunction that will lead to a diseased state? No. However, I have palpated its inflamed state, and contracture of the common bile duct, pancreas, small intestine, and large intestine across the human life span from babies to clients in their eighties. It is one of the most common progressions I have experienced in 27 years, not the only. It is the linkage between these organs and the anatomic centrality of the gall bladder that implicates its participation in so many disturbances of functional physiology. When one considers that some estimate that "more than half of our blood, 65%, may be in our veins" (6) on a moment-by

moment basis, the clinical importance for us to attend to assisting its systemic return to the heart becomes clear.

I would propose as a life-long student of biology that nature does prioritize physiologic function in its survival orientation. Specifically, that the assimilation of nutrients is its primary task to keep on trucking with the prime directive of perpetuating the species. The neural priority that the brain gives to recognizing the body parts involved in locomotion over the internal organs derives from the same priority. Difficulties with elimination and venous and lymphatic drainage are simply collateral strains that often lead to unintended damage and the reduction in the quality of our lives.

"We are perfectly adapted to a world that no longer exists." (7) I would propose that elements of consciousness and identity consolidation form the modern foundation of what drives our nervous system. To date, 100% of the clients who have come to me with reported or medically diagnosed anxiety problems, all have some degree of gall bladder and other visceral involvement described in this article.

Survival vs. quality of life is an ongoing paradox that confronts our species with all of its diversity. Nature's endowments of fight, flight, and freeze are the shadow elements of our collective need to create ways to cooperate with one another (8).

In order to be thorough, I have decided to extend this series beyond the proposed three. In the next installment, we will explore the relationships between gall bladder/pancreatic dysfunction to how alternate routings of the venous return system may influence cardiac efficiency, blood pressure, and the competence of the hiatal junction.

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