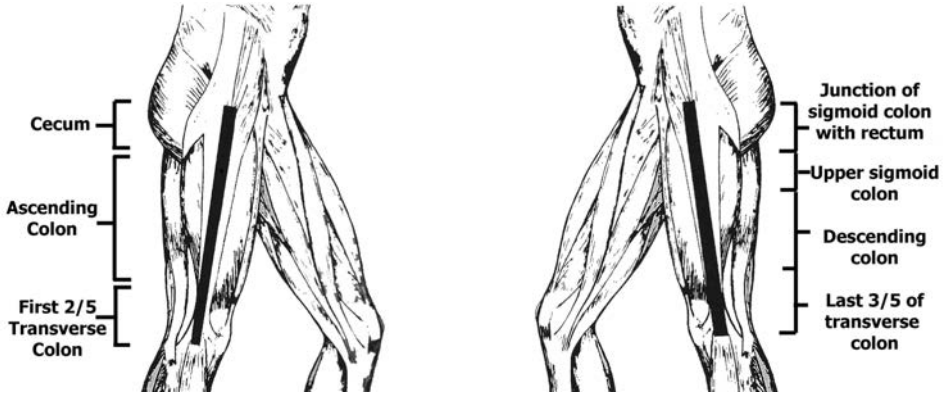

CHAPTER SIX

Applied Kinesiology and Systemic Conditions of the Lower Body

Introduction

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Differential diagnosis of systemic disorders of the pelvic region and lower extremities can be extremely complex. It calls on all the skills a clinician has to draw on in order to combine observation and examination together into a coherent whole. Always consider that dysfunction or pathology from any part of the body can refer disorders and pain into the lower body. It should be recognized also that the pathoanatomical model of lower body dysfunction is frequently inadequate. Pain in the lower body often exists in the absence of major findings on diagnostic biomedical tests (CT scan, blood tests, nerve conduction tests, X-ray), and injured tissues can be identified in people who experience no pain. **(Nachemson, 1999)**

Melzack, **(2001)** in his *Neuromatrix Theory of Pain*, echoes Goodheart by showing that it is often necessary to treat the entire person, and not just the painful parts. In evaluating patients with systemic conditions in the lower body, it must be remembered that biomechanical adaptation may have advanced to a stage where neural, lymphatic, articular, muscular, biochemical and psychological interactions are synchronistic and plentiful. Consideration must additionally be given to the medications a patient is taking, as well as to the symptoms such medications may create, so that the physician can refer the patient back to the drug-prescribing physician should the symptoms be suspected of being linked to the medication(s). A Physician's Desk Reference, which is current and easily searched, is a necessary asset.

The scant or absent hair of the lower leg in patients with arteriosclerosis, especially due to diabetes; **(Veves et al., 2002)** the fine, long leg hair of the anorexic patient; the patient with white flecked nails due to chronic zinc deficiency; **(Shils et al., 1999)** the unilateral leg edema of deep vein thrombosis, thrombophlebitis, pelvic tumor, or cellulitis;

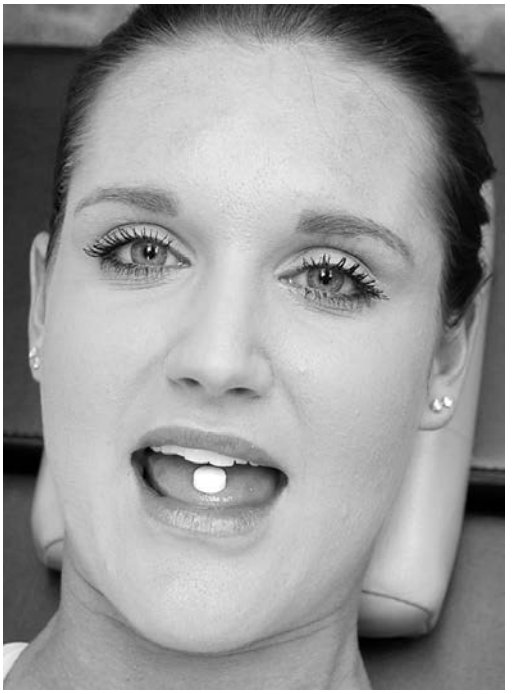
the impaired perception of deep touch, pressure, and vibration, anesthesia, paresthesias, diminished or absent deep tendon reflexes, pathological reflexes, paresis, and ataxia of pernicious anemia; **(Guyton & Hall, 2005)** the dyspareunia due to post-surgical pelvic adhesions, regional ileitis, or pelvic inflammatory disease; **(Chaitow & Jones, 2012)** the left lower quadrant pain of diverticulosis - these and all of the possible differentials make for the high art of clinical practice.

Our purpose here isn't to cover all of that ground, but rather to present tools from applied kinesiology that contribute to distinguishing and resolving systemic disorders that can plague the pelvic region and lower extremities. Often the patient presenting with complex clinical patterns can defy our best efforts at a definitive differential diagnosis even when our tools of history, physical exam, laboratory findings, and imaging have been brought to bear. Visceral hyperalgesia and central sensitization may be predominant features of chronic lower body pains. **(Giamberardino et al., 2010)** Long-term physical, emotional, or biochemical stress may drive long-term potentiation of the CNS so that an individual becomes vulnerable to perceiving sensations that would not normally hurt other individuals. **(Rygh et al., 2002)** In these cases AK (by focusing on the triad of health as it manifests in a particular patient's functional ensemble) can be useful by both helping to integrate and contextualize our existing findings as well as suggesting further orthopedic, neurological, physical exams, laboratory testing, and imaging that may help further establish diagnosis.

The field of clinical nutrition has wide application in treating and preventing systemic disorders, and the evidence supporting this mode of therapy in clinical practice continues to grow. **(Gaby, 2006; Shils et al., 1999)** Applied kinesiology can be useful in adding to the information gathered from history, physical exam, and laboratory findings when applying clinical nutrition for a particular condition. All possibilities for clinical nutrition will not be considered here. The clinical nutrition applications included will be those classically used in applied kinesiology along with the 5-Factor manipulative techniques. **(Goodheart, 1998-1964; Schmitt, 2005, 1981, 1979; Force, 2003; Maffetone, 1999; Boormann, 1979; Deal, 1974)**

Testing Biochemistry with Applied Kinesiology

Ingesting nutrition targeted to a particular organ has been found to more reliably improve muscle strength using AK MMT than placebo, supporting both the organ-muscle relationship model of applied kinesiology and the usefulness of nutrient testing in AK, especially when the information is incorporated with other clinical observations. (Evidence for the AK approach to nutrition is expansively supported in ***Applied Kinesiology Essentials)*** Testing nutrients without ingestion, a method not approved by the International College of Applied Kinesiology, is unreliable. **(Hambrick, 2007; Goodheart, 1998)** AK nutritional testing reflects the nervous system's efferent response to the stimulation of the gustatory and olfactory nerve receptors by various substances. If these receptors are not stimulated (as for instance when a patient holds a pill or has it placed upon the body), the neurological rationale, plausibility and the reliability of sensorimotor pathways for AK testing disappear.



Direct stimulation of the gustatory receptors

AK MMT appears to be a useful method of observing the biochemistry of the organism in that it has shown impressive accuracy for determining food allergies (90.5%) and immunological status when compared to lab tests, though the outcomes of studies have been conflicting. **(Schmitt, 1998; Conable, 2006)** Walther **(2000)** and others have written extensively about this phenomenon; more complete descriptions are beyond the scope of this chapter. However nearly 1,000 case and case series reports over the past 35 years have been given by the members of the ICAK around the world regarding the successful use of nutritional therapies using AK diagnostic means, and are available from several platforms. **(ICAKUSA, 2012; ChiroACCESS, 2012)**

Viscero-somatic Reflexes and Muscle-Organ-Gland Relationships

Viscero-somatic reflex phenomena have been extensively explored in the research **(Pollard, 2004; Masarsky & Masarsky, 2001)** and in the text ***Applied Kinesiology Essentials.*** It has long been established that visceral pain can be referred not only to skeletal muscles but also to the skin, ligaments, and bone. **(Sinclair et al., 1948)** Travell & Rinzler **(1952)** have shown that pain in the pectoralis muscle can accompany coronary infarction, and

this finding has been confirmed. **(Nicholas et al., 1987)** Irritation to specific organs has been observed to result in muscle inhibition, primarily of the muscle(s) associated with the irritated organ and secondarily and to a lesser degree with other muscles of the body. This outcome supports to the muscle-organ relationship model in applied kinesiology. **(Carpenter, 1977)** This finding is further buttressed by research that has shown that stress to an internal organ can result in a viscero-somatic reflex inhibiting both motor and sensory nerves in AK practice. **(Shafton, 2006; Palomar, 2006)** Visceral inflammation has been shown to produce reflex cutaneous leukocyte extravasation. **(Wessellmann, 1997)** Korr presciently observes that viscerosomatic reflex activity may be observed before any symptoms of visceral change are evident and that this phenomenon therefore is of important diagnostic value. **(Korr, 1976)** Finally, Beal **(1985)** elegantly summarizes the research about the "body language" of viscerosomatic disorders by stating that "somatic manifestation is an integral part of visceral disease."

Somato-visceral and viscero-somatic reflexes appear to be interdependent as visceral afferents inhibit the effects of cutaneous afferentation, and cutaneous afferents inhibit the effects of visceral afferentation. **(Pomeranz, 1968)**

Somatoautonomic Nervous System, Somatovisceral Reflexes and Manual Therapies

> *"The term 'autonomic' is a convenient rather than appropriate title, since the functional autonomy of this part of the nervous system is illusory. Rather its functions are normally closely integrated with changes in somatic activities, although the anatomical basis for such interactions are not always clear…A more realistic notion is that these sets of neurones represent an integrated system for the coordinated neural regulation of visceral and homeostatic function…Rises in blood pressure and pupillodilation may result from the stimulation of somatic receptors in the skin or other tissues."* ***(Gray's Anatomy, 2004)***

The neurosciences are rapidly compiling evidence that manual therapies produce distinct and clinically meaningful effects on visceral functions through somato-visceral reflexes. For a better understanding of this research, we recommend the recent impressive synopses offered by Rome. **(Rome, 2010, Rome, 2009)**

Mechanoreceptors and cutaneoreceptors appear to cause somatovisceral reflex responses mediated by both the parasympathetic and sympathetic nervous systems. **(Sato, 1992)** Soft tissue manipulation to the suboccipital region has been shown to increase upper extremity digital blood flow through down-regulation of sympathetic tone (sympathetic dampening). **(Purdy, 1996)** Stimulation to both cutaneous and subcutaneous afferents has also shown somato-visceral effects. **(Sato, 1995)**

In accord with these research findings, soft tissue reflex techniques such as the neurolymphatic (Chapman's) reflexes and acupuncture meridian stimulation have growing and in some cases impressive support in the literature and appear to be useful, both diagnostically and therapeutically. **(Caso, 2004; Moncayo, 2004)** Soft tissue manipulative techniques for the diaphragm, including neurolymphatic (Chapman) reflexes, have shown increased forced vital capacity. **(Lines, 1990)**

Emerging evidence supports the long-term clinical observations of the chiropractic and osteopathic professions that aberrant spinal mechanics can have an adverse effect on autonomic and visceral function and that spinal manipulation has a modulating effect on autonomic and visceral function. This phenomenon appears to have a sound neurophysiological basis. **(Karason, 2003; Haldeman, 2000; Budgell, 2000; Kimura, 1997; Sato, 1992; Jinkins, 1989)**

We might make reference in particular to the chiropractic profession's heritage of removing interference with normal nerve control of the body. Chiropractic has been successful in developing techniques to evaluate abnormal structure and return it to normal, thus improving both peripheral nerve function and autonomic nerve function. There is, however, a phase of nerve control which the profession has failed to adequately observe and master. This is the neurohumoral control of the autonomic nervous system. The allopathic physician has made considerable investigation into neurohumoral control; in fact, many medications are based on this principle. **(Wilson et al., 1998)** Unfortunately the medications are fraught with side effects, as are most procedures with which we try to control the body rather than returning it to its own natural control. Neural control of visceral and neurohumoral function is a unique coordination of somatic and autonomic motor nervous systems; sensory information and motor control are supplied by both visceral and somatic sensory and motor fiber systems. **(Enck & Vodusek, 2006)**

The most frequent neurohumoral imbalance of the autonomic nerve system is caused by relative hypoadrenia. Norepinephrine from the adrenal medulla is necessary for cholinergic activity in the neurohumoral balance of the autonomic nervous system. The adrenal medulla can be dysfunctional from abnormal nerve supply as a result of a spinal subluxation-fixation, or from prolonged stress as described by Selye. **(1978)** When the general adaptation syndrome (GAS) approaches the third stage of exhaustion, the adrenal is incapable of meeting the demands placed upon it. If stress – physical, mental, thermal, and chemical – is the cause of the relative hypoadrenia, nothing but removal of that stress will bring the patient back to normal. On the other hand, the adrenal can become dysfunctional because of nutritional deficiencies which in themselves are a form of stress. It is, without question, the physician's responsibility to return the nervous system to normal function; however, the correction must be applied to the basic underlying cause of the nervous system's imbalance.

This review of the neurological phenomena underpinning applied kinesiology is not complete, but is presented as a basic context for the applied kinesiology approaches to systemic disorders of the pelvis and lower extremity that follow.—

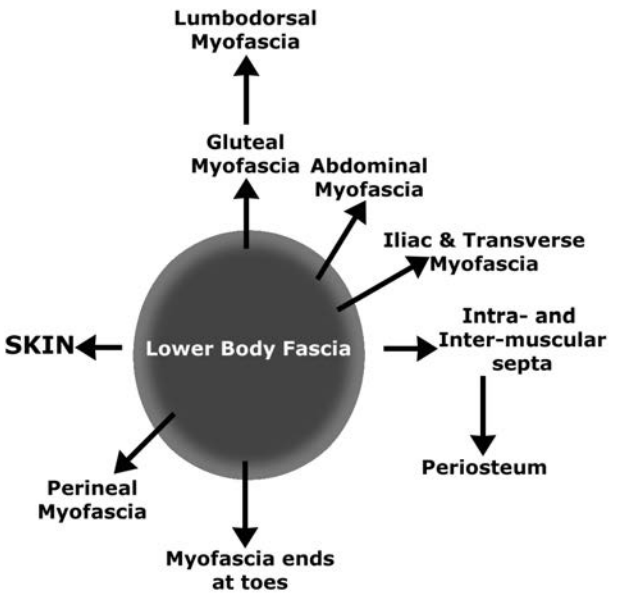
Myofascial release and somatovisceral effects

The manual therapy called myofascial release technique in applied kinesiology exerts somatovisceral reflex effects as noted by Goodheart, who reported that myofascial release technique to the teres minor resulted in increased axillary temperature. It is hypothesized that this increase in axillary temperature is due to a thyroid mediated increase in metabolic rate resulting from the myofascial release treatment of the teres minor muscle, associated with the thyroid gland. **(Goodheart, 1978)** A number of studies have confirmed this somatovisceral hypothesis. **(Moncayo & Moncayo, 2007; Reuters et al., 2006; Moncayo et al., 2004; Bablis & Pollard, 2004; Duyff et al., 2000)**

Myofascial release technique appears to be a method of systematically modulating visceral function through therapy to myofascia. The usefulness of this application for other organ-muscle reflex patterns besides the teres minor-thyroid pattern has been observed. **(ICAKUSA, 2012)**

Muscle and fascia have been shown to be anatomically and functionally inseparable. Electron microscopy has even demonstrated that fascia has smooth muscle actin cells imbedded within it. **(Schliep et al., 2005; Barnes, 1997)** As muscular dysfunctions are being corrected, it is necessary to understand that fascial structures are also being reorganized and fortified. Myofascia is the sum of connective tissues throughout the body. It provides structural and functional support to the soma and viscera. Fascial adhesions have been a recognized cause of musculoskeletal pain and associated with gross anatomical and histological changes since the early to middle 20th century. **(DeJarnette, 1939; Murray, 1938)**

After joint and muscle spindle input is taken into account, the majority of remaining proprioception comes from the fascial sheaths. Acquired abnormalities in elasticity and afferentation of myofascia due to mechanical strain, trauma, surgery, inflammation, infection, or systemic biochemical stresses can result in aberrant biomechanics and neurological disorganization. **(Lewit, 2004)**



Myofascial continuities in the lower body

It has been proposed that a limited mechanical model of myofascial adhesion is inadequate to fully explain the inelasticity of myofascial adhesions found upon palpation and the immediate increase in elasticity following manipulation. A neurological model of myofascial manipulation that restores optimal mechanoreception and, thereby, modulates sympathetic tone and myofascial contractility may more fully explain these observations. **(Schleip, 2003)** It has been shown that fascia's gel-like ground substance which invests its collagen and elastic components may be altered to a more liquid state by the introduction of vibration, percussion, heat, movement or manipulation of the tissue, such as that applied in percussion and other soft-tissue techniques used in AK.

Percussion and myofascia

Fulford introduced mechanical percussion into osteopathic soft-tissue therapies in the 1950s. **(Comeaux, 2011)** Percussion alone will frequently remove the Travell and Jones trigger and tender points. **(Cuthbert, 2002)** The "glued" myofascia that Ida Rolf described **(Rolf, 1977)** is also effectively mobilized and corrected by percussion.

Percussor instrument

The immediate effect of percussion is to modify the physical nature of the myofascial matrix. **(Comeaux, 2011)** The greater fluidity in the matrix will allow an improved fiber density, direction, and movement pattern. The fascial sheets will begin to glide across the mobilized and "smoothed out" connective tissue matrix and intramuscular areas. When inner and outer layers of muscle and fascia are in proper balance, the tissue will have proper "tone," the way a piano string that is in perfect tension has perfect pitch. Elasticity of connective tissue between structures

is essential for an effective relationship between deep and superficial (intrinsic and extrinsic) muscle layers.

Under conditions of decreased mobility and inadequate tissue fluid dynamics (lymphatic or vascular), collagen undergoes polymerization and other chemical changes, causing shortening or decreased elasticity in myofascial tissues. These changes can sometimes occur over an extended period of time.

The colloids in connective tissue are not rigid; they conform to the shape of their container and respond to pressure even though they are not compressible. Under conditions of accelerated tissue fluid dynamics and increased demand for mobility created by the percussor instrument, the collagen in the myofascia "depolymerizes" and becomes longer and more elastic. A treatment that depolymerizes collagen essentially means that we reduce the molecular size of a colloidal structure. Depolymerization also implies an increase or improvement in the biological activity of the tissue. A fascial matrix with heavy concentrations of fat and gristle and glassy, knotty concentrations in it will have its physical nature modified, smoothed-out, ironed-out, and softened by percussion treatment.

Myofascial tissue that has become short in compensation to a lack of movement elsewhere in the body can be thought of as tissue with a low potential energy and conductivity. Because of the inherent gelosis and tension in these tissues, very little activation energy is required to start the exothermic process of releasing them.

Percussion may also press fluid from the nuclear bag of the muscle spindle cells, reducing the tension in the capsule of the spindles. Positive TL to neuromuscular spindle cells and Golgi tendon organs may also be eliminated with percussor treatment.

Therapeutic manipulation of visceral myofascial adhesions

There are a number of approaches for resolving visceral adhesions and AK MMT is effective in determining the need for a given technique and for the effectiveness of therapy. Visceral manipulation is usually best done in brief therapeutic sessions and re-evaluating and reinforcing treatment on follow-up sessions as needed.

There is a basic rule of thumb in tissue pathology: once a tissue is injured, it often heals by downgrading into a simpler, less specialized form. Early in this century, Dr. Louisa Burns, **(1948)** an extraordinary osteopathic researcher, showed that "somatic dysfunctions" are accompanied by microscopic effusions of blood, with edema and inflammation occurring in the connective tissues of the affected joints and muscles. This "extravasation of blood" resolves over time as these connective tissues thicken. Dr. Burns believed that these structural thickenings of the connective tissues found throughout the body were responsible for the restriction of mobility in joints and for the accompanying pain. The literature of Structural Integration (Rolfing) and massage exhaustively describe these phenomena.

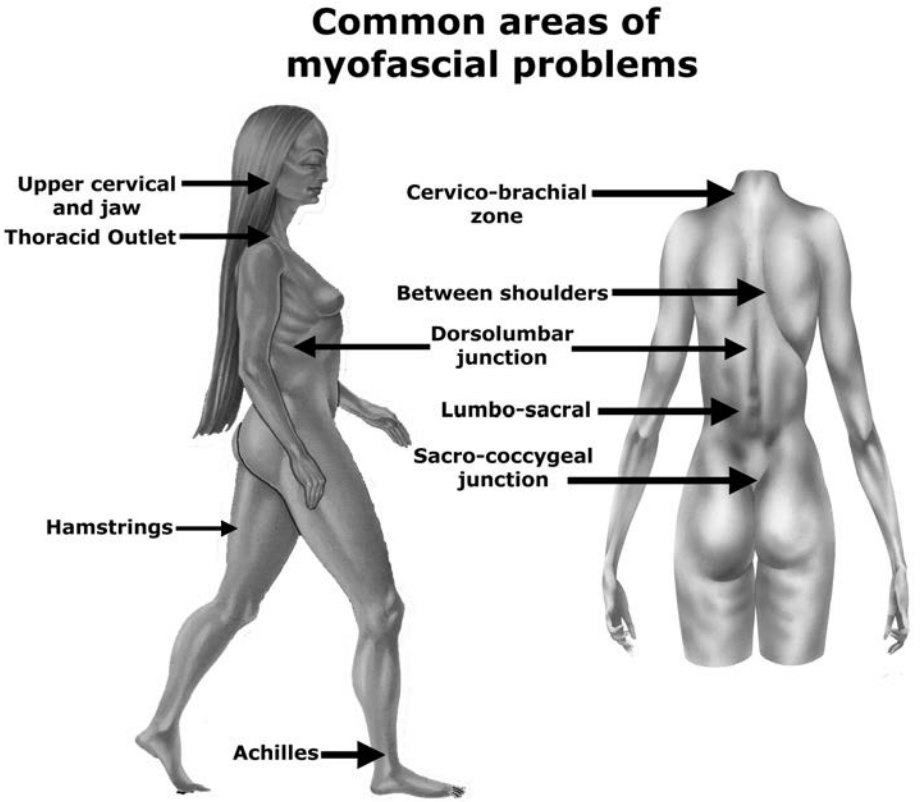
What begins as changed muscle tone as the body adapts as stress progresses to structural changes in the connective tissue elements that surround and supplement the muscle fibers involved in the adaptation. This produces changes in the material properties of the collagenous and elastic connective tissues, including reticular and ground substances that compose the tendons, ligaments, and fascia, as well as the colloidal interstitial fluids. These material tissue changes involve molecular and structural alterations. Embryologically, the entire mesoderm can be caught up in this adaptive response of the body to stress. This is the "body language" that we commonly attempt to read.

In normal connective tissue, the fibers – whether elastin or collagen – are oriented parallel to the forces ordinarily exerted through the tissue, in conformity with Wolff's law. **(Wolff, 1892)** "Every change in the function of a bone is followed by certain definite changes in internal architecture and external conformation in accordance with mathematical laws." When connective tissue is injured or chronically shortened or stretched, there will be an increase in fibroblasts. These new cells produce additional fibers laid out in random matrix fashion, rather than along the original lines of force.

The hard ridges and folds in myofascia that can be felt in patients with myofascial disorders come into being much the way pulling on a corner of a sheet creates deep pleats in the fabric. Similarly, pulls across the back of the upper pelvis and the sacrum give rise to tendon-like structures across the lower back. These ridges, folds, and thickenings of the connective tissue can literally feel like small ropes or cables under the skin. In some patients it feels as though these ropes have knots in them. The structure may even look like a tendon in dissection; it definitely feels like a tendon under the skin. But in anatomy books, no tendons are described in that location.

Common Areas of Myofascial Problems

- Along McGregor's line (from skeletal radiology), which is across the pterygoid and masseter muscle masses and extends back from the jaw to the suboccipital area; the so-called cranial stress bands and switchboards.
- The attachment of the levator scapulae to the upper cervical vertebrae; the upper trapezius and other cervical extensors to the occiput; as well as the ligamentum nuchae.
- The crossover point of the levator scapulae and the supraspinatus at the upper medial point of the scapula (medial thoracic outlet); "the cervicobrachial syndromes."
- The trapezius as it crosses the top of the shoulder near the acromion; the lateral portions of the thoracic outlet.
- The trapezius where it attaches to the scapula below the scapular spine. "It hurts me between the shoulder blades."
- The junction between the lower trapezius and latissimus dorsi behind, and the psoas and diaphragm in front; the dorsolumbar fixation.
- The tight pad created at the lumbosacral junction, and across the top of the pelvis. This



Common Myocascial areas of trouble

myofascial disorder across the sacrum is a major contributor to chronic lower back syndromes. The founder of cranial osteopathy suggests that "sacral sag and fascial drag create old rags."

- The sacro-coccygeal junction.
- The hamstrings.
- The Achilles tendons.

Somatic Fascia

The fascia directly supporting the musculoskeletal systems of the body is the somatic fascia. The somatic fascia includes the subcutaneous structures and the connective tissues surrounding muscles, groups of muscles, blood vessels, and nerves and binds all these structures together into a functional whole.

It has been proposed that the deep fascia of the body depends on the production of hyaluronic acid (HA) within the fascia by fibroblast-like cells (fasciacytes) to maintain free and unrestricted sliding motion between adjacent fibrous fascial layers. Alterations in HA conformation may lead to changes in muscle dysfunction and pain. **(Stecco, 2011)** This is one of the underlying processes creating areas of increased density and decreased mobility found upon palpation that are commonly referred to as fascial (or myofascial) adhesions. Nutritional factors also influence fascial function and structure directly. "Many of the results of deprivation of ascorbic acid [vitamin C] involve a deficiency in connective tissue which is largely responsible for the strength of bones, teeth, and skin of the body and which consists of the fibrous protein collagen." **(Pauling, 1976)** Normally, connective tissue cells are surrounded by a stiff jelly-like or cement-like substance (the ground substance), which in animals deprived of vitamin C becomes watery and thin, powerless to support the cells. It is possible that in cases where there is a failure of the intercellular substances to set to a gel after myofascial treatment, that vitamin C supplementation may provide a solution in many cases. Connective tissue protomorphogens may also be helpful. **(Cuthbert, 2002)** Protection against myofascial tissue damage is also provided by super oxide dismutase (SOD). **(Moncayo & Moncayo, 2007)**

Travell & Simons are even more emphatic that nutritional imbalances must be corrected if myofascial pain is to be adequately resolved: "Nearly half of the patients whom we see with chronic myofascial pain require resolution of vitamin inadequacies for lasting relief… nutritional factors must be considered in most patients if lasting relief of pain is to be achieved." **(1999)**

Over-use syndromes, trauma, surgery, infection, and chronic inflammation may all play a role in histological changes in myofascia that leads to altered myofascial kinetics. **(Stecco, 2008)** These changes in myofascial histology and kinetics may lead to the "propagation of a nociceptive signal even in situations of normal physiological stretch." **(Stecco, 2006)** Many sensory receptors and their axons have a lower tensile strength than the tissues in which they are embedded. Physical trauma to myofascia and their mechanoreceptors and axons may result in localized proprioceptive loss. **(Sharma, 1999)**

Besides the structural role of fascia, it provides

proprioceptive afferentation through Ruffini and Pacini corpuscles, and if normal elasticity of myofascia has been altered the resulting aberrant firing of the afferent receptors may result in abnormal biomechanics and extra-articular pain. **(Stecco, 2009)**

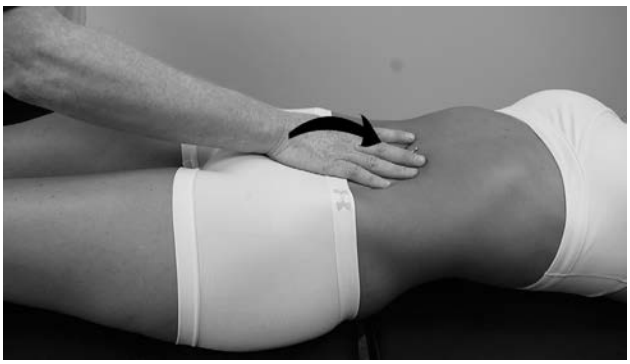
Applied kinesiology includes a number of techniques that address these aspects of myofascial dysfunction. These are fascial release, myofascial trigger point treatment, muscle spindle cell and Golgi tendon organ, percussion and myofascial gelosis techniques. These are outside the scope of this discussion with the exception of the somatovisceral effects of myofascial release technique.

Visceral Myofascia

The visceral myofascia wraps the organs of the body and suspends them. It is less elastic than somatic myofascia. Organ prolapse/ptosis may result from laxity of visceral myofascia, while hypertonicity may limit organ motility.

Hypertonicity, diminished elasticity, or limited shear between planes of visceral myofascia is called myofascial, or visceromyofascial, adhesion. For our purposes here, we will be referring to adhesions of the peritoneum (peritoneal adhesions) which are a relatively common outcome of surgery, injury, and systemic or local inflammatory processes. These often result in intestinal obstruction, infertility, pelvic pain, compartment syndromes, edema of the lower limb, and other functional disorders of the lower body. **(Paoletti, 2006)**

Re-epithelialization of surgically incised peritoneum is rapid (~5-8 days). The principal mediators of the healing process are leukocytes, mesothelial cells, and fibrin. Fibrinolytic enzymes normally modulate proliferation of the fibrin gel matrix associated with the healing peritoneum. **(DiZerega, 1997)** Persistent pelvic tissue inflammation prolongs elevation of epidermal growth factor and transforming growth factor-alpha and promotes the development of pelvic adhesions. **(Chegini, 1994)** A smaller amount of inflammation at the site of peritoneal healing produces less adhesion formation. **(Elkins, 1987; Van Der Wal, 2007)**



Uterine "Listening" test for organ motility

It has also been proposed that visceral adhesions result, in part, from over-production of reactive oxygen species (ROS) by phagocytes during healing, triggering over-expression of vascular endothelial growth factor. **(Roy, 2004)**

Intra-pelvic adhesions have been associated with a wide range of symptoms that include meteorism, irregular bowel movements, chronic abdominal pain, digestive disorders, infertility, and intestinal obstruction. It has been proposed that 40% of all intestinal obstructions are due to post-surgical adhesions. **(Bruggmann, 2010)**

Visceral Myofascial Therapy by Barral

Barral, a French osteopath, has developed and refined a system of visceral manipulation in the osteopathic tradition. **(Barral, 2005)** Consideration is given for the axes of motility inherent to the individual viscera resulting from embryological development. According to Barral's model, the organs have a motility cycle that is similar to, but independent of, the craniosacral respiratory cycle and, though each organ has unique movement patterns determined by its relationships to the peritoneum, including the mesentery and omenta, the cyclical movement is, ideally, synchronous. Here, motility is defined as the movement inherent to the organ itself, and mobility is the sliding movement between the organs.

This system distinguishes visceral restrictions as being functional and positional. Articular restrictions are functional visceral restrictions that cause loss of organ mobility. Articular restrictions are further classified as adhesions if the restriction results in diminished motility, but leaves mobility intact, and fixations if the restriction results in both diminished motility and mobility. These distinctions become more apparent, it is claimed, with the development of skills necessary to palpate them.

Muscular restrictions (viscerospasms) arise from spasticity or adhesions within the wall of hollow organs such as the small and large intestines, stomach, gall bladder, biliary and pancreatic ducts, and sphincters (e.g., sphincter of Oddi).

Ligamentous laxity, often resulting from lost elasticity due to the chronic restrictive strain of visceral adhesions, can result in ptosis of the organs, a positional restriction.

Barral describes three types of visceral manipulations – direct, indirect, and induction. The direct and indirect techniques are primarily concerned with restoring mobility, though they will indirectly address — at least to some extent – limitations in motility. Induction is primarily concerned with motility and requires highly developed palpatory skills, while indirect technique requires long lever methods utilizing body position to enhance mobility, tissue tension or traction.

Barral offers manipulations for ptosis as well, which will not be described in lieu of the description for the applied kinesiology specific method to follow.

There are two types of direct techniques for articular restrictions – rocking and recoil. In both methods, one should use the distal palmer surface of the hand and fingers and gently traction the tissues perpendicular to the direction of adhesion/restriction for mobility of the soft tissues as determined by palpation and/or a direct AK MMT challenge.

For the rocking method, one should apply short back and forth rocking movements at approximately 10 cycles

per minute. The practitioner will feel the underlying tissues become more pliable, elastic, and relaxed under the hands, and when complete, a direct challenge in the direction of previously positive challenge will be negative.

In the recoil method, traction to the tissues is released suddenly and repeated 3-5 times. Again, the tissues should feel softer and more pliable under the hand, and a repeat of the previously positive challenge should now be negative.

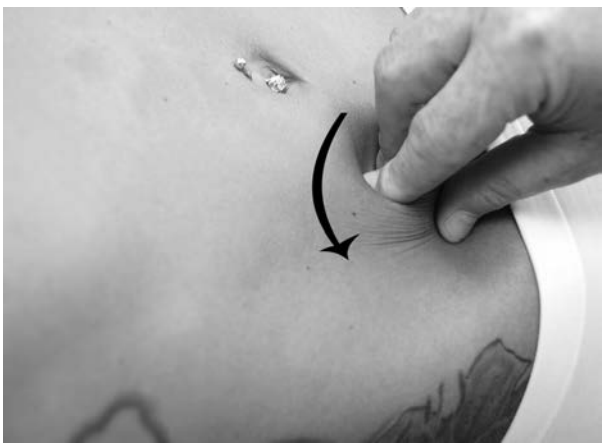
For viscerospasm, Barral recommends putting the soft tissues under traction and then mobilizing toward the direction of greatest mobility. Here, too, one can apply an AK MMT challenge in addition to palpation in order to determine when the muscular restriction or viscerospasm has been resolved.

It should be kept in mind that Fryer et al. **(2010, 2005)** have attempted to detect via palpation tissue irregularities in segmental tissues associated with viscerosomatic dysfunction. Their results failed to show a correlation between palpable changes and irregular motor activity of deep paraspinal muscles. However structural changes in deep and superficial paraspinal muscles and referred hyperalgesia are present in cases of visceral disease and dysfunction. **(Giamberardino et al., 2005; Vecchiet et al., 1990)** These structural alterations in muscles and functional alterations in strength, measurable with the MMT, may be considered a key factor in the more reliable diagnosis of viscerosomatic dysfunctions.

Visceral Myofascial Therapy by Walker

Walker **(2012)** describes a method of visceral manipulation utilizing a torquing contact that can be effective for both determining the presence of visceral adhesions and for resolving them.

While holding the hand with the interphalangeal joints in a slightly flexed position ("eagle claw") push lightly into the abdominal wall and turn, or torque, the hand in a counterclockwise direction. The pressure should be firm enough that the force transmits through the abdominal wall and into the underlying viscera, but not be painful. Challenge in this fashion over the suspected area of visceral adhesion or screen by challenging the abdominal quadrants.



Counter-clockwise sensorimotor challenge followed by MMT

Challenge with counterclockwise torque over myofascial adhesions will cause a previously strong indicator muscle to weaken, and inspiration or expiration will be found to negate the challenge. Treatment is done with a clockwise torque over the area of positive challenge during the phase of respiration that negates challenge, then easing this pressure with a slight recoil when the patient reaches full inspiration or expiration. Repeat 5-10 times, and re-challenge with the counterclockwise torque to evaluate for effectiveness of correction.

It is useful to include therapy to the segmentally associated area of the spine while treating the area of visceral adhesion to improve results of this therapy. Torque the area of positive visceral challenge on the indicated phase of respiration, and hold the torquing pressure on either full inspiration or full expiration as indicated. Simultaneously, use counter-torque pressure to the skin over the spine opposite the involved viscera. Then continue to hold the torquing pressure on both points while the patient resumes breathing until the clinician and/or the patient feels the visceral tension let go under the anterior (visceral) contact hand.

In chronic cases, it is recommended to include treatment of reflexes associated with the involved viscera/organ as indicated by AK. Finish the manipulation by maintaining pressure over the area of treatment throughout 3-5 complete respiratory cycles. Apply visceral therapy while the patient is in different positions (supine, prone, lateral recumbent, on all fours, and in suspect positions of stress associated with activities of daily living), and simultaneously to surrounding areas of visceral adhesion as indicated by AK MMT challenge.

Walker includes other protocols into his treatment of visceral adhesions, but they are specific to methods taught in association with Neuro-Emotional Technique, **(Walker, 2012)** which he developed, and are outside the scope of this chapter.

We have found it useful to follow up this protocol with caudal, cephalad, and left and right lateral AK MMT challenges with moderate pressure to the area of visceral adhesion with the distal palmer surface of the hand and fingers. Typically, restriction will be felt that correlates with the direction of positive challenge. Use light thrust in the vector of positive challenge with the same contact as for your challenge and repeat 4-10 times. Re-challenge for correction. Greater elasticity and pliability under the hand during challenge will usually be felt when therapy has been successful.

Visceral Myofascial Therapy by Chaitow (Specific Release Technique)

Chaitow describes a method of visceral therapy emphasizing rapid traction manipulation with a slight rotary component. **(Chaitow, 1988)** In this form of visceral manipulation, one hand serves as an anchor for the tissues immediately adjacent to the area of adhesion, and the other

hand is placed abutting the anchoring hand prior to rapidly pulling away from it.

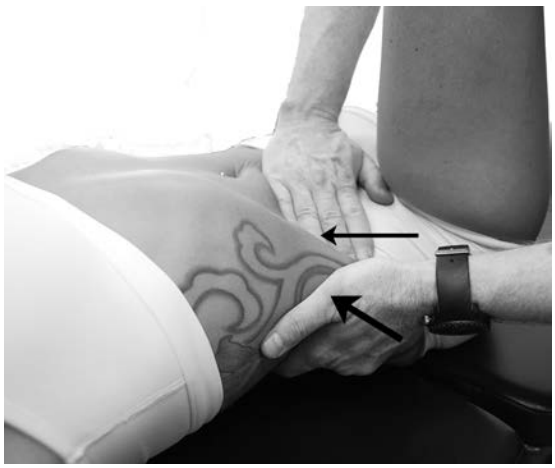
The active hand makes contact using the ventral pads of the distal aspects of the slightly flexed fingers. Chaitow suggests setting the ventral pad of the middle finger in a slightly forward position relative to the rest of the hand, making it the focus of palpatory diagnosis and manipulation. The lateral aspect of the thumb of the opposite anchoring hand, by pressing into the abdomen posteriorly, serves as a fulcrum point for manipulation by the active hand.

The patient is supine with the knees flexed and the head resting in a slightly flexed position in order to relax the abdominal wall. The doctor stands to the side of the patient opposite the area of adhesion (i.e. if the area of visceral adhesion is in the left lower quadrant of the abdomen, the doctor stands to the right side of the patient). With this example, the doctor will use the right hand as the active hand and the left hand as the anchoring hand. From this position, the doctor can palpate the location and quality of the adhesion by drawing the pads of the active hand across the involved area.

During manipulation, the lateral aspect of the thumb of the anchoring hand is set to the border of the adhesion, and the active hand pulls rapidly away from the anchoring hand. A slight clockwise or counterclockwise torque is added to the linear force.

As an example, if the right hand is the active, manipulating hand, moving the hand laterally and the elbow medially will create a clockwise torque, and moving the hand medially and the elbow laterally will create a counterclockwise torque. Speed will produce the requisite acceleration to develop adequate force necessary to mobilize the adhesion. Apply a very specific, high velocity and low amplitude torsional force to the focal area of the adhesion, analogous to chiropractic adjustment/manipulation to articular fixations/ adhesions. Palpation and manipulation is repeated over the area of adhesion until mobility and resiliency to the soft tissues are restored.

Chaitow recommends general neuromuscular treatment to the abdominal region prior to specific release technique and a general soft tissue procedure to lift the viscera into its normal anatomical position. Abdominal and postural exercises are recommended along with diaphragmatic breathing techniques to complement the more specific release technique.



Kidney lift technique
Mobilize the kidney anteriorly, medially, and superiorly

Visceral Repositioning (Ptosis)

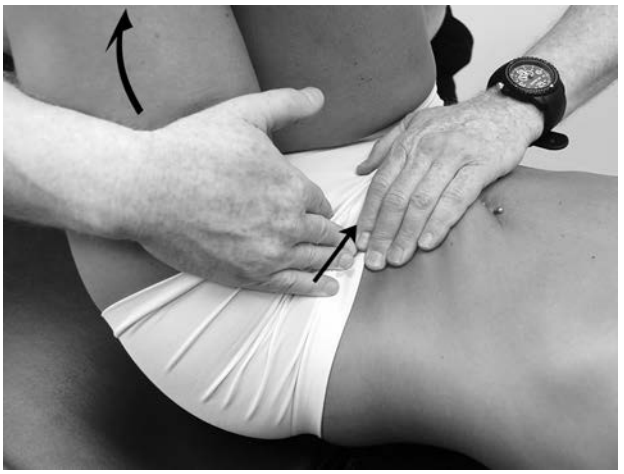
Portelli has elegantly combined the concepts of DeJarnette, Rees, Barral, Goodheart and others and described an AK sensorimotor visceral challenge using the MMT for organ position. In this methodology, when an organ (i.e. kidney, large intestine, liver, stomach, pancreas) displaced from its normal position is statically challenged in a vector that increases displacement, an indicator muscle in the organ-muscle relationship of AK will become inhibited. Testing muscles unrelated to the organ will not display a positive challenge, and traction of the organ will not result in a positive challenge unless displacement is present. **(Goodheart, 1992)** Portelli presented this technique for organs of the abdominal and pelvic region originally but later found the protocol applicable to the heart and esophagus as well. **(Portelli, 2001)**

Treatment is implemented with visceral manipulation to replace the organ to its normal anatomical position by tractioning the organ in the direction opposite to the positive challenge. The most common vector for reduction is cephalad since most organs descend due to gravity. **(Smith et al., 2007)** Traction is performed with the distal palmer surface of the hand and fingers increasing to a peak of pressure coincident with the patients' maximum inhalation followed by sudden and sharp coughs. This is repeated several times. Contact pressure is released while the patient exhales, and this maneuver is repeated a few times as needed to negate the positive challenge.

Using the cough to facilitate release of myofascial restriction appears to be very effective, and the increased mobility and elasticity of the tissues can be readily felt. Portelli recommends that the patient reproduce this procedure on oneself before retiring each evening for approximately three months. Testing and correcting for imbalances of the abdominal muscles (rectus abdominus — upper and lower divisions —oblique abdominus, transverse abdominus, and pyramidalis) is recommended.

This technique will commonly be useful for ptosis of the transverse colon and may be associated with recurring ileocecal valve syndrome, weakness of the abdominal wall muscles, premenstrual tension, and constant, nagging pain in the lower abdominal region. **(Portelli, 1987)**

"Soft-Tissue Orthopedics" was developed and researched by Dr. M. L. Rees in conjunction with DeJarnette, **(Rees, 1994)** and was influential in the approaches developed by Portelli. It is based on a unique set of indicators employing techniques involving soft tissue manipulation of the spine, cranium, extremities, organs, glands and other soft tissue, and the balancing of somatopsychic and psychosomatic components. Palpatory indicators were found that correlated spinal, cranial, soft-tissue reflex, acupuncture and other indicator areas that guided the clinician in diagnosing and treating organic disorders. "By non-cutting techniques, to prevent and correct soft tissue deformities, to preserve and improve the function of organs and organ systems and their nerve supply when such function is threatened or impaired by defects, lesions or diseases." **(Rees, 1994)**



Manipulation of sigmoid colon was shown to produce hypoalgesia in segmentally related spinal structures

An important new investigation by osteopathic researchers in the UK has shown that immediate effects can occur in the paraspinal musculature by improving pressure pain thresholds after manipulation of the sigmoid colon, an approach long used in AK. **(McSweeney et al., 2012)** This study showed that visceral manual therapy produced immediate hypoalgesia that was not systemic but only in somatic structures segmentally related to the organ being mobilized in asymptomatic subjects. The visceral manipulation employed in this study was like the one indicated in AK for the valves of Houston, wherein the supine subject's sigmoid colon was contacted laterally, in the left iliac fossa and drawing it superomedially, and then releasing it, for 1 minute duration.

The Pelvic Region Chronic Pelvic Pain (CPP)

Though inflammation, infection, and various pathologies must be considered in the differential diagnosis of pelvic pain syndromes, myofascial adhesions must also be considered. The literature now clearly supports the fact that the musculoskeletal system is both a cause and a consequence of CPP. **(Neville et al., 2012; Browning, 2009)** Neville et al. found that women with CPP had "core weakness" in their muscles (particularly the core abdominal and pelvic floor muscles); in these women there was also a higher Total Musculoskeletal Dysfunction (MKSD) Score. Neville et al. continue by reiterating a long-held contention in AK: "If core muscles are weak or the timing of activation and control of the muscles is altered by pain or other disruption, then compensatory mechanisms for muscle imbalances themselves may contribute to the patients' pain complaints." Further, they suggest that "Identifying musculoskeletal factors as a source of CPP would enable earlier and more precise diagnosis, and potentially earlier treatment, of musculoskeletal causes of pelvic pain."

Myofascial adhesions also appear to be a common factor for pelvic pain, with estimates for this etiology being as high as 25%. **(Stones & Mountfield, 2000)** Commonly observed presentations due to myofascial adhesions are deep pelvic pain during certain trunk or pelvic positions, deep respiration, defecation, or sexual intercourse in women (dyspareunia). CPP also appears commonly be a sign of pelvic floor myofascial dysfunction. In general, pelvic floor myofascial trigger points (MTrPs) refer pain to the vagina, penile base, perineum and rectum often with an urgency to urinate. MTrPs in the pelvic floor muscles can mimic coccydynia or levator ani syndromes. **(Simons et al., 1999)**

Extensive coverage of pelvic pain syndromes is presented in *Chronic Pelvic Pain and Dysfunction.* **(Chaitow, 2012)** CPP syndromes include anorectal pain syndrome, bladder pain syndrome, clitoral pain syndrome, endometriosis-associated pain syndrome, interstitial cystitis (IC), pelvic floor muscle pain, pelvic pain syndrome, penile pain syndrome, perineal pain syndrome, post-vasectomy pain syndrome, prostate pain syndrome, pudendal pain syndrome, scrotal pain syndrome, testicular pain syndrome, urethral pain syndrome, vaginal pain syndrome, vestibular pain syndrome, and vulvar pain syndrome. **(Fall, 2010)**

Chaitow cites Anderson **(2006)** concerning the failure of traditional medical therapy to adequately treat CPP "whether involving anti-biotics, anti-androgens, anti-inflammatories, beta-blockers, thermal or surgical therapies, and virtually all phytoceutical approaches."

Masarsky and Browning have developed a model for the pathophysiology of pelvic pain syndromes being potentially related to occult lumber nerve root involvement and resolved through lumbar decompressive therapy (Cox Flexion-Distraction treatment). **(Cuthbert & Rosner, 2012; Browning, 2009, 1988; Masarsky, 2001)** Dr. James Cox, **(personal letter)** founder of the flexion-distraction technique in the chiropractic profession, confirms that "the pudendal plexus is the chiropractor's map in our work to handle genitourinary disease."

Inguinal Hernia

Applied kinesiology treatment for inguinal hernia has been reported to be an effective therapy in both pediatric and elderly patients. Two patients in a case report by Kaufman **(1996)** had considered surgical repair. Treatment consisted of AK therapy to muscles of the pelvic region and groin (origin-insertion, Golgi tendon organ, muscle spindle cell) as well as neurolymphatic reflexes, neurovascular reflexes, ileocecal valve, a category II pelvis, and upper cervical correction. Clinical nutrition directed by AK MMT was included. Patients experienced resolution in 6-8 visits. Though a small case study, the rapid and cost effective resolution of inguinal hernia for patients considering surgical intervention merits consideration of an AK trial in patients with inguinal hernia.

Another case study reported resolution of a right inguinal hernia as well as cryptorchidism after two treatments, one week apart, for a nine month old boy indicating that this approach shows promise. The treatment was chiropractic care to the pelvis and spine directed by AK MMT. **(Maykel, 2003)**

Urinary Tract Dysfunction

Urinary Incontinence

Urinary incontinence (UI) occurs when there is leakage of urine involuntarily, most commonly in older patients. **(Amadhi et al., 2010)** Fantl et al **(1996)** state that incontinence affects 4 out of 10 women and 1 out of 10 men during their lifetime, and about 17% of children below the age of 15. A large post-partum study on the prevalence of UI found that 45% of women experienced UI at 7 years postpartum. Thirty-one percent who were initially continent in the postpartum period became incontinent in the future. **(Wilson et al., 2002)**

Continence depends primarily on the adequate function of two muscular systems – the urethral muscular support system and the sphincteric muscular network of the pelvic floor muscles (PFM). **(Ashton-Miller et al., 2001)** These systems include the levator ani muscle, detrusor muscle, and PFM (coccygeus, obturator externus, obturator internus, gemellus inferior, gemellus superior, and levator ani), as well as the pudendal nerve that emerges from the sacral plexus. The striated muscles of the pelvic floor play an integral role in the closure of the lumen of the urethra and the maintenance of continence. **(DeLancey, 1990)** In women with stress UI, ineffective contraction or control of the pelvic floor muscles permits descent of the bladder neck with inadequate closure of the urethra, resulting in the leakage of urine. **(Steensma et al., 2010)**

The co-morbidities of lumbo-pelvic pain, incontinence and breathing pattern disorders are slowly being elucidated. **(Smith et al., 2008)** Musculoskeletal impairments and specifically muscular imbalances between agonist and antagonist muscles in the pelvis create articular strain and soft tissue stresses which can lead to pain and UI. **(Thompson et al., 2006)**

Current observations suggest that patients with stress incontinence may have imbalances in several lumbo-pelvic muscles which inhibit the PFM and lead to incontinence. **(Smith et al., 2008)** Recent data also indicate that breathing difficulties and incontinence are associated with increased chances for the development of low back pain (LBP), **(Smith et al., 2006)** demonstrating that the interactions between the lumbar and pelvic muscles and joints may be an important consideration in cases of UI.

A recent study assessed strength changes in the PFM using a perineometer (a pressure electromyograph that registers contractions of the PFM) after the application of chiropractic manipulative therapy (CMT). This investigation showed that phasic perineal contraction and basal perineal tonus, force and pressure *increased after CMT*. **(de Almeida et al., 2010)** The duration of these force changes will have to be assessed in subsequent studies of this type.

It was Arnold Kegel who first advocated pelvic

Urinary incontinence:
Applied Kinesiology case series report

A retrospective chart review was described on the AK management of 21 patients (age 13 to 90), with a 4 month to 49 year history of daily stress and occasional total urinary incontinence (UI). Each of these patients had associated muscle dysfunction and low-back and/or pelvic pain, with 18 of these patients wearing an incontinence pad throughout the day and night because of unpredictable UI.

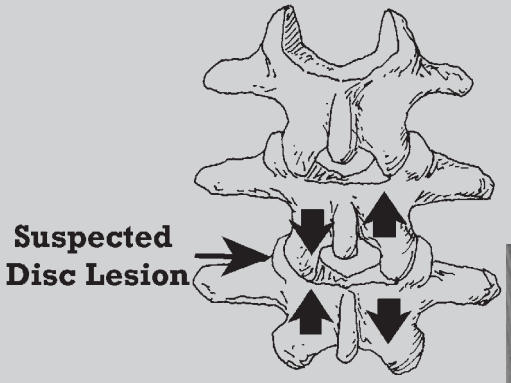
Applied kinesiology methods were utilized to diagnose and treat these patients for muscle impairments in the lumbar spine, pelvis and pelvic floor with concomitant resolution of their UI and low back and/or hip pain. Positive MMTs in the pelvis and lumbar spine muscles and particularly the pelvic floor muscles was the most common finding, appearing in every case. Lumbo-sacral nerve root dysfunction was confirmed in 13 of these cases with pain provocation tests (AK sensorimotor challenge); in 8 cases, this sensorimotor challenge was absent. Chiropractic manipulative treatment (CMT) to the soft tissue or articular disturbances related to these inhibitions normalized symptoms in 10 patients, considerably improved 7 cases, and slightly improved 4 cases from between 2 to 6 years.

The following MMT findings were present in this case series of 21 patients with UI:

Inhibition of left, right or bilateral gluteus maximus:	17
Inhibition of left, right or bilateral hamstring:	16
Inhibition of pelvic floor muscles:	14
Inhibition of diaphragm:	11
Inhibition of left, right or bilateral psoas:	11
Inhibition of left, right or bilateral rectus abdominus:	10
Inhibition of left, right or bilateral gluteus medius:	9
Inhibition of left, right or bilateral piriformis:	7
Inhibition of left, right or bilateral rectus femoris:	6

In 13 cases, the AK sensorimotor diagnostic challenge for lumbar disc involvement was positive. When the vertebra above the disc lesion was touched by therapy localization with one hand and the vertebra below with the other hand, therapy localization was positive in these cases. This means that it either strengthened the PFM or other involved muscles or weakened them if they tested initially strong. **(Goodheart, 1975)** This method of localizing disc involvement was added to the routine diagnosis of disc lesions and helped confirm the level of involvement.

An additional applied kinesiology diagnostic approach to disc involvement was the two-handed "AK challenge" of the vertebrae above and below the disc lesion. **(Goodheart, 1975)** The spinous or transverse processes were used as levers, and a separating or compressing force was applied between the two vertebrae. The muscles usually tested with this challenge were the hamstrings if they were initially strong in the particular case under examination. In all 21 patients, this particular sensorimotor challenge was performed; however, the challenge procedure – which guided the use of subsequent Cox flexion distraction decompression treatment – only influenced the function of the involved muscle inhibitions in 13 of the cases. The CMT utilized for the category III and lumbar disc challenges that were present was the flexion-distraction decompression adjustment method. After the Cox flexion distraction decompression treatment of these 13 cases, the positive indicator of a disc lesion using the AK method became negative and improved the UI in all cases.



The AK lumbar disc challenge. (Left) The vertebrae above and below the suspected IVD involvement are challenged by separating or compressing the spinous or transverse processes, then testing a strong indicator muscle for inhibition following the challenge.

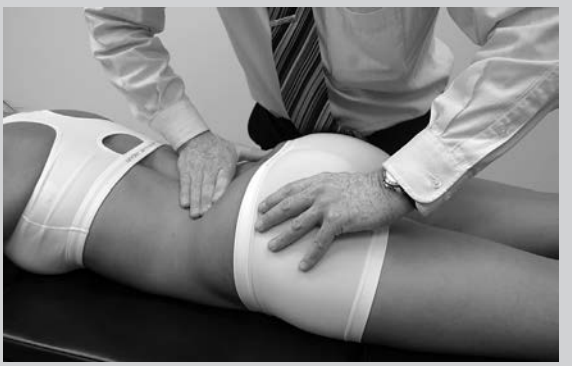
Flexion-distraction decompressive treatment corrected the AK sensorimotor challenge to the lumbo-sacral spine. (Right)

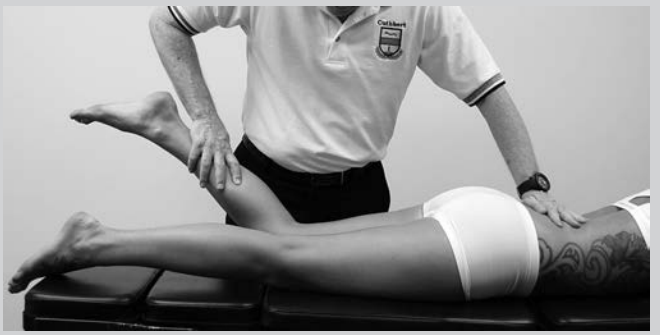
In 8 cases, there was no disc involvement indicated, and yet UI was present — suggesting that lower sacral nerve root dysfunction was not the only etiological cause of UI and PFM inhibitions. In these cases, the treatment for the muscle inhibitions present involved three approaches: (a) CMT for the pelvis, (b) remote treatment to articulations that innervate the tissues involved (the upper cervical spine and the gluteus maximus muscle for example), and (c) percussion to involved myofascial trigger points in the PFM.

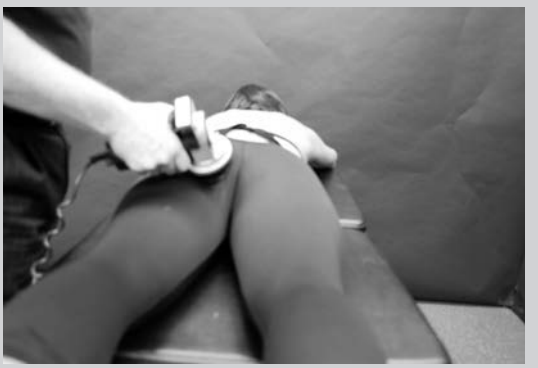
Breathing pattern disorders involving the diaphragm muscle were also involved in 10 cases.

In 13 cases, scars from pregnancy, caesarian sections, and one appendectomy were present. Diastasis rectus abdominus was present in 3 of the 13 cases with scars from pregnancy. Gentle pincer palpation of the particular scar, as well as stretching the underlying muscle produced weakening of the same muscle on MMT. AK mechanical treatment to the scar (using a Percussion device) **(IMPAC, 2012)** abolished the pincer palpation finding to the scars and the MMT weakness in the underlying muscle. **(Cuthbert, 2002)**

In 11 cases there was a positive finding of pincer palpation to the PFM. Treatment for the myofascial trigger points (MTrPs) found in the patient's PFM was made with a Percussor instrument. After 2 minutes of percussion upon the MTrPs found in the PFM, the AK "pincer palpation" test became negative, and pressure on the MTrPs that previously produced either referred pain or muscle inhibitions in previously strong indicator muscles no longer occurred. **(Walther, 2000)**







Pincer palpation of the PFM (a) produced weakening of the previously strong hamstrings. (b) Percussion on the PFM (c) corrected this finding and reduced palpation tenderness and referred pain from the MTrPs present in the PFM.

In six cases there had been previous spinal (1), abdominal (1), or pelvic organ (4) surgeries; in 1 of these cases (1), there was normalization of the UI condition; in 3 cases, considerable improvement; and in 2 of these cases, only slight improvement.

The first consultation, examination and treatment for each of these patients lasted for 1 hour. Follow up treatment sessions were required covering 1 to 13 treatment(s) for this cohort to reach maximum improvement for their UI, covering a period spanning 1 day to 6 weeks. The diagnoses of muscular inhibitions related to articular and soft tissue disorders of the pelvis and lumbar spine and their treatment using CMT were effective in attaining the resolution (n=10), considerable improvement (n=7), or slight improvement (n=4) of daily stress and occasional total UI in these cases. These burdens remained corrected as seen by yearly follow up examinations for the past 2 to 6 years.

It should be emphasized that improvement in patients with long-standing UI occurred in 10 cases, even in two patients with UI that had been present for 49 and 40 years, respectively. In these 21 cases, positive MMT of the pelvic and lumbar spinal muscles (particularly the PFM) was the most common manifestation in every case. The AK MMT identified the inhibited PFM or the inhibited muscles related to the PFM anatomically or functionally.

From these considerations and data, there may be reason to suspect that the patterns of muscle weakness associated with UI and disclosed by MMT methods may be of clinical utility in guiding treatment for this disturbing condition.

floor muscle strengthening and retraining for stress incontinence, **(Kegel, 1948)** indicating his recognition of the importance of muscle inhibitions in cases of UI. The usefulness of the AK MMT approach in this model would be the identification of the inhibited muscles involved, and the AK treatment approaches used to immediately address these inhibitions. A number of other reports have been made on the successful use of chiropractic treatment for elderly patients with UI. **(Kreitz & Aker, 1994)** Stude et al reported a case study of a 14-year-old female treated with CMT who recovered completely from traumatically induced UI. **(Stude et al., 1998)** The AK approach to a post-appendectomy induced case of UI has been recently described as well. **(Cuthbert & Rosner, 2011)** Duffy **(2004)** successfully resolved a complex case of UI as well, with treatments including CMT, soft tissue manipulation (uterine lift technique), and AK muscle-reflex therapies, including neurolymphatic reflexes and inactivation of myofascial trigger points for muscles of the pelvis, especially the levator ani. CMT has been shown to be effective in other reports on bladder control problems. **(Reed et al., 1994; Blomerth, 1994; LeBoeuf et al., 1991)** Allen, **(2000)** a Diplomate chiropractic neurologist, has noted a relationship between fixation of the cervico-dorsal spine and inhibition of these bladder related muscles.

Nocturia

Goodheart states that nocturia in children is commonly a result of impaired regulation of CO2 levels during sleep due to impaired responses of the respiratory centers in the lower brain stem. CMT and AK reflex techniques to the phrenic and intercostal nerves are the recommended therapy. **(Goodheart, 2003)**

Applied kinesiology was successful in treating a four year old male with nocturia. Treatment consisted of chiropractic and craniosacral therapy and supplemental phosphorylated B complex emphasizing thiamin. Correction of foot pronation was part of the therapy, which may potentially influence bladder meridian function. This case study included mention of testing for ADH for possible diabetes insipidus. This reflects good practice in applied kinesiology by including all the clinical tools a clinician has to draw from to arrive at an accurate differential diagnosis. **(Duffy, 2003)**

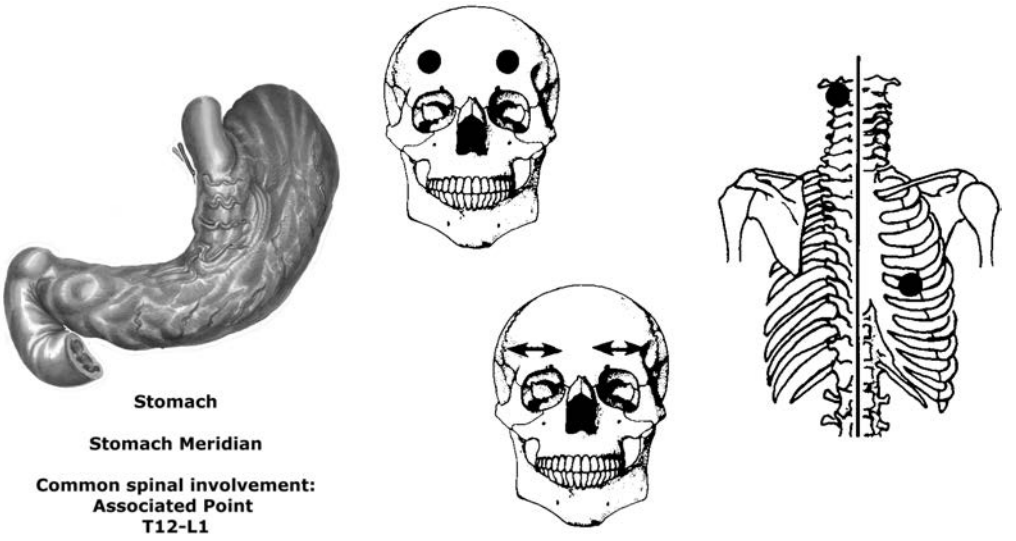
Additional causative factors for nocturia may be food allergy, **(Mungan, 2005)** sensitivity to artificial flavors, colors, sweeteners, **(Salzman, 1976)** and thiamine deficiency. **(Vanhulle, 1997)** Thiamin deficiency has been reported to be associated with increased nocturnal urination, a short breath holding time, (20 seconds or less) low pulse rate, low body temperature, and failure of the pulse rate to increase as predicted with exercise. **(Goodheart, 1989a)**

Cystitis and interstitial cystitis

Peters calls for evaluation of the pelvic floor when patients diagnosed with interstitial cystitis, urinary frequency and urgency, and pelvic pain have found therapies to the bladder epithelium unsuccessful. **(Peters, 2006)** The evidence for this comes from a study that found an 87% prevalence of pelvic floor dysfunction, as evidenced by levator ani hyperalgesia, in women with IC. **(Peters, 2007)**

Pelvic floor trigger point therapy appears to be effective for patients with chronic interstitial cystitis (IC) and urgency-frequency syndrome. (UFS) A study of 52 subjects (45 women, 7 men) with either interstitial cystitis or urgency-frequency syndrome who were treated at a frequency of 1-2 sessions per week for 8-12 weeks showed moderate to marked improvement or complete resolution of the UFS group (83%) and moderate to marked improvement in the IC group (70%). **(Weiss, 2001)**

Deficiency of vitamin A has been associated with cystitis. Rats fed a vitamin A deficient diet have been found to have a high prevalence of pyelonephritis (68%), cystitis (66%), nephrolithiasis (5%), and squamous metaplasia of the transitional epithelium (100%). **(Munday, 2009)** Another study in rats found that vitamin A deficiency resulted in squamous metaplasia of the urinary bladder and high incidences of cystitis, ureteritis, and pyelonephritis. **(Cohen, 1976)** Consider vitamin A deficiency in patients with chronic cystitis as directed by AK MMT and other clinical findings. Vitamin A deficiency is commonly associated with systemic lymphatic congestion as seen when patients need the AK retrograde lymphatic technique. **(Goodheart, 1979)** Sprieser **(2002)** reports on 50 patients with interstitial cystitis who improved using AK techniques involving treatment to the bladder meridian.



Reflexes for stomach

AK and Digestive Dysfunction

Here we are concerned with the viscera presenting signs and symptoms associated with the pelvic region - small intestine, ileocecal valve, and large intestine. This area has been covered in the AK *Synopsis* by Walther **(2000)** and our intent is to complement that text.

Stomach and small intestine

Hydrochloric Acid

"Too much acid in the stomach" is considered by many to be the characteristic situation when there is a problem with heartburn, or gastroesophageal reflux disease (GERD). **(Bredenoord, 2012)** Actual evaluation of the function of the body reveals that the opposite is true, as has been confirmed by AK analysis and treatment results for decades. Patients with reflux symptoms often do not have excessive esophageal acid exposure, and patients with severe gastroesophageal reflux often do not have reflux symptoms. Reflux characteristics other than acidity, such as the presence of bile, pepsin, liquid, or gas in reflux, and the proximal extent or volume of reflux, may also contribute to symptom perception. Factors contributing to greater esophageal sensitivity may include impaired mucosal barrier function, peripherally mediated esophageal sensitivity (enhanced esophageal receptor signaling), and centrally mediated esophageal sensitivity (physiological stressors, sensitization of spinal sensory neurons).

When there is a disturbance in hydrochloric acid balance, it is usually a *lack* of hydrochloric acid which is present rather than an excessive amount of it. It is well documented that as people age, less hydrochloric acid is produced. Unfortunately, the symptoms of hyperchlorhydria and hypochlorhydria somewhat parallel each other, with both causing a burning-type sensation and discomfort in the epigastrium. A rule of thumb (but by no means totally accurate) is that the person who has burning on an empty stomach has hyperchlorhydria and a person who develops pain after eating has hypochlorhydria. Gas immediately after eating, especially a protein meal, is indicative of deficient hydrochloric acid; gas that develops lower in the abdomen several hours after eating is indicative of a lack of proteolytic-pancreatic enzymes in the intestinal tract. These are strictly rules of thumb whereas a more accurate method is to test, therapy localize and challenge the mechanisms involved.

It is unfortunate that many people who have hypochlorhydria often obtain relief from symptoms by taking antacids. The mechanism for relief is an acid rebound as a result of insulting the body with an alkaline substance. Prolonged treatment of this nature, whether prescribed by a physician or self-administered patent medications, leads to many additional health problems, such as mineral deficiencies and protein imbalance leading to degenerative joint disease. **(Levin, 2004; Jensen & Anderson, 1990)**

Hiatal Hernia

The hiatal hernia can mimic the symptoms of hypo-or hyperchlorhydria. There is usually a substernal burning as the acid regurgitates into the esophagus; however, the burning pain can be in the epigastrium area, radiating to the chest and other places. The evaluation and treatment of hiatal hernia and diaphragm muscle weakness is indicated in all cases of apparent digestive disturbances. Burstein reviewed the case records of 92 patients in his AK-practice and found hiatal hernia dysfunction in 32 out of 92 new patients (34.7%), making it a common finding. **(Burstein, 1990)**

The use of chemical challenging by asking the patient to chew or suck on a tablet is invaluable, not only to determine what nutritional factor is needed, but also to find what is detrimental to body function. The patient in the case report above has no question that the exact answer

Case Report: AK and severe digestive burning

A 53-year old female presented herself for treatment of severe digestive burning. She had previously been a patient for temporomandibular joint disorders and bilateral knee pains. When she originally came to the office for treatment of those conditions, she was extremely skeptical of natural health care because of heavy indoctrination throughout her career as an emergency room nurse that medication was the best solution for any health problem. Our original treatment was successful for her. However, when the digestive disturbance occurred, she did not think of natural health care; she went back to her standard approach of allopathic medicine. Upper GI x-rays were taken with a barium sulfate swallow as well as a gall bladder study. Thorough physical evaluation could find no problem to cause the symptoms she was experiencing. The diagnosis of hyperchlorhydria was made, and the physician told the patient that she had a "nervous stomach," making it necessary for her to take antacid medication with every meal for the rest of her life. The patient did this for about three weeks, and the symptoms completely abated as long as she took the medication. If she skipped the medication with any meal, the symptoms returned – interestingly enough, more severe that before.

Fortunately, this patient retained enough of our previous broad-scope natural health care education to reject taking medication for the rest of her life; she decided to come in for evaluation and treatment. Upon examination, weak bilateral pectoralis major (clavicular division) muscles were found, which were strengthened by half an inspiration held while testing. This indicated a temporal bulge cranial fault (affecting the vagus nerve), which was confirmed by challenge. A parietal descent cranial fault was also found on the opposite side. Furthermore, a diaphragm deficiency was found, correlating with the substernal burning pain experienced by the patient. The key to the diaphragm weakness was found to be a reactive psoas. Because of the difficulty in obtaining the corrections, the patient was asked to walk for a short distance and then re-tested to make certain the bilateral pectoralis major (clavicular division) weakness and the diaphragm weakness did not return. After walking, the condition was present exactly as it had been prior to treatment. Another attempt was made to correct the temporal bulge causing the pectoralis weakness and to correct the reactive psoas-diaphragm complex. Again, correction was very difficult, and the condition returned after the patient walked. When a correction is difficult to obtain and a condition returns rapidly, some problem not yet located is recreating the involvement.

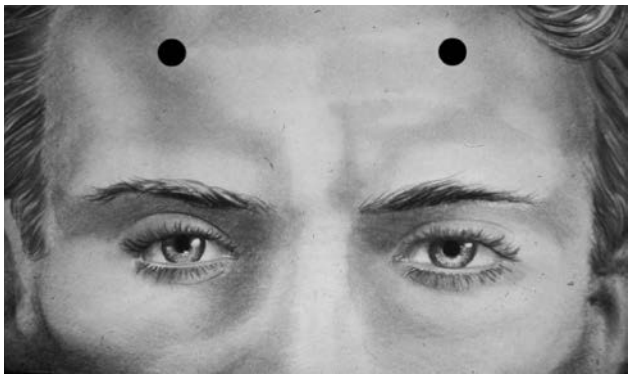
The patient was asked when she had last taken an antacid tablet; she answered approximately three hours earlier. With no further attempt to correct the temporal bulge and reactive psoas-diaphragm complex, she was given betaine hydrochloride to suck on. After she had sucked on the tablet long enough to get a good taste and to swallow some of the material, the pattern was re-tested. The pectoralis major (clavicular division) was now found bilaterally strong, and there was no evidence of temporal bulge or parietal descent cranial faults, and the reactive psoas-diaphragm complex was no longer present. The betaine hydrochloride made all these changes with no other treatment given. Since the patient had some of the antacid tablets she had been taking with her, she was then challenged with this formula. By simply sucking on the antacid tablet for no longer than 15 seconds, the temporal bulge/parietal descent complex returned, as well as the bilateral pectoralis major (clavicular division) weakness and the reactive psoas-diaphragm complex. Again, she sucked on a betaine hydrochloride tablet and the entire abnormal picture was alleviated, and walking no longer caused its return. The patient was given betaine hydrochloride tablets, one with each meal, for two weeks. She now frequently eats chili made with jalapeno peppers (a staple in Pueblo, Colorado) and any other spicy foods she desires. There has been no return of her symptoms of hyper-or-hypochlorhydria.

to her problem was found; she is also grateful that she does not have to take medication designed to override her system for the rest of her life. It is this type of correction and education that has proved itself to be invaluable in the word-of-mouth development of referrals for digestive disturbances in clinical practice.

In hydrochloric acid disturbances, there is typically a bilateral pectoralis major (clavicular division) weakness, as indicated previously. If this bilateral weakness is not present — though the symptomatic picture indicates a hydrochloric acid disturbance — test for a bilateral lower trapezius weakness. This can mask the bilateral pectoralis major (clavicular division) weakness. If there is a bilateral lower trapezius weakness, there will be a dorsal-lumbar fixation. After the fixation is corrected, the weakness of the pectoralis muscle will usually emerge.

There are many functions attributed to hydrochloric acid. One activity that is not widely discussed is the disinfecting action of hydrochloric acid upon bacteria which is swallowed. As the hydrochloric acid concentration in the stomach is lowered, the bactericide effect is diminished. Many germs can pass through the stomach without this protective effect. Hydrochloric acid is important in the first step of protein digestion. It splits the protein molecules and is necessary for the action of the proteolytic enzyme pepsin. Hydrochloric acid is responsible for splitting disaccharides and monosaccharides and for stimulating the flow of bile and pancreatic juice. Bacterial action continues in the stomach as long as the food bolus is alkaline. Basically there is a continuation of salivary digestion in the alkaline medium, where ptyalin (or salivary amylase) continues its work on carbohydrates. Micro-organisms can also continue working in an alkaline stomach to split carbohydrates into gases and organic acids. This contributes to the gassiness and burpiness so often observed in hypochlorhydria patients shortly after eating.

The stomach wall and duodenal area is adequately protected from hydrochloric acid by mucous secretion and an adequate blood supply. If the walls of the stomach and duodenal area are ischemic, there is a lowered resistance to



Stomach neurovascular reflex points

breakdown of tissue by hydrochloric acid and the action of pepsin. The neurovascular reflex on the frontal area is often found to be active in these cases. This reflex, of course, correlates with the blood vascular supply to the stomach; it also correlates with general stomach function, including hydrochloric acid production.

Protein Digestion

Many individuals have a protein deficiency, although high levels of protein are eaten in western countries. **(Gaby, 2006)** First, it is necessary to be certain that all the essential amino acids are present in the diet. Goodheart has an illustration of what takes place when one essential amino acid is missing. If you have a wooden barrel made up of staves, it is only functional in holding liquid up to the height of the lowest stave. The staves of the barrel can be likened to the essential amino acids; if one amino acid is deficient, the body will only utilize the amino acids present to the level of the one deficient. With many types of diets, especially vegetarian, it is very difficult to be certain all the essential amino acids are present in sufficient amounts. One method by which this can be evaluated is to have the patient fill out a frequency diet schedule, or keep an actual diary of what he or she eats for one week. The frequency diet schedule asks questions about how often certain types of foods are eaten throughout a month; the diary, of course, evaluates the actual diet of the individual. There are several companies that provide these forms and have computer tie-ins which evaluate the information provided by the patient, giving the amount of amino acids and other nutrients in the particular patient's diet.

Even if there is adequate protein in the diet, inappropriate digestion of protein often occurs, usually because of achlorhydria or hypochlorhydria. Attention should be given to the small intestine and pancreas for the production of the appropriate enzymes, and the liver should be thoroughly evaluated.

Indication of protein deficiency is most readily obtained by observing body language. In protein deficiency, there will be poor growth and quality of fingernails; the hair will be dry and brittle, with cracked ends, and it will tend to fall out; the skin will be dry, hard, and tend to wrinkle, with breaking and splitting. Blood laboratory findings may appear normal to those who are not aware of the body's attempt to maintain normalcy under adverse conditions. We must remember in evaluating blood laboratory that the blood stream is nothing more than a distribution route for nutrients within the body. For example, there is a concentration of 30 times more protein in the joint surfaces than in the blood stream. If the blood stream protein level is down, the body will draw from the joint surfaces to attempt to maintain normal blood protein levels. Rubel discusses the evaluation of the albumin-globulin ratio (AG ratio) as being more significant than evaluating the total protein levels. **(Rubel, 1959)** If protein is pulled from tissues, the AG ratio will change, with globulin rising. If the globulin is above 3 gm%, this indicates that the body is withdrawing protein from tissue to make up the deficient albumin levels.

Protein deficiency is significant in the total digestive process, because enzymes are manufactured from protein. When there is a relative hypoproteinemia, the body goes into a protein-sparing effect in which it reduces various physiologic processes, including enzyme production. Lowered enzyme production causes many phases of the digestive process to be lowered. We see here how one digestive fault – such as hypochlorhydria or achlorhydria – can cause a snowballing effect in the total digestive process. The correction of this problem can be as simple as correcting a temporal bulge cranial fault, or as complicated as searching each and every factor present in a total series of events.

Protein and arthritis

A protein deficiency is involved in many types of arthritis, not just in degenerative arthritis. Before discussing the actual effect on the joint, a brief discussion of protein evaluation and the types of imbalance is paramount.

The many utilizations of protein by every cell, organ, tissue, and function of the human body are well documented. Some specific areas of protein utilization and protein movement are important in the various forms of arthritis. Rubel **(1959)** reviews findings at the University of Illinois indicating that for every gram of protein in the blood stream, a healthy joint surface requires 30 times more. In the presence of a negative nitrogen balance, where there is less protein taken in than the needs of the body and excretion, there is a protein shift from one compartment of the body to another to supply adequate protein.

Frequently the negative nitrogen balance is not caused by a lack of dietary protein intake; it may be caused by the individual's inability to absorb and utilize the protein in the diet. Achlorhydria or hypochlorhydria, especially in aging people, contributes considerably to the inability to absorb protein. The acid content of the stomach has been determined – in the average normal person – to be ideal at the age of 25; at age 40, there is 15% less hydrochloric acid in the stomach; at age 65 there is only 15% of the amount produced at 25 years of age.

Indication of hypochlorhydria or achlorhydria is gaseous distension of the stomach shortly after eating. This pressure is frequently felt high in the abdominal area, perhaps even before the meal is finished, and is relieved by belching. Failure to eliminate the gas often causes

symptoms in the diaphragm, resulting in short-windedness and cardiac palpitation. Pruritis ani may be present due to the fact that the fecal material is too alkaline, never having been completely acidified in the stomach. As the food bolus proceeds down into the small intestine, there is action by secretions from the intestinal wall and from the pancreas. The pancreas secretions are stimulated by the food bolus in the duodenum and are influenced by the degree of acidity developed in the stomach. A high acid level will increase the secretion and concentration of the enzymes manufactured and secreted by the pancreas.

Enzymes developed by the pancreas are, of course, protein in nature; when there is a tendency for the body to conserve protein. A deficiency of pancreatic enzymes may be a result of hypoproteinemia.

When considering protein utilization, an important factor is that all the essential amino acids must be present for protein utilization. If there is a deficiency of one amino acid, the other amino acids will be utilized only to the level of the deficient amino acid.

In the presence of a negative nitrogen balance, the body will pull protein from the highly concentrated joint surfaces and synovial membranes. After this withdrawal has taken place to any degree, normal joint movement becomes trauma, with the consequent inflammatory reactions to trauma.

Protein deficiency is one of the mechanisms by which cortisone products and ACTH give relief from arthritis. These medications cause release of albumin from muscle tissue, hence making it available to the blood stream and joint surfaces and decreasing the irritability, inflammation, and arthritic manifestations of the joint. This is accomplished at the expense of muscle tissue; if it is continued for long, it leads to protein deficiency in other tissues, even though the arthritic process has been relieved.

The same mechanism can be applied to bursitis. The negative nitrogen balance causes withdrawal of protein from the synovial caps. Calcium in the body is carried united with albumin; when the albumin has been withdrawn, calcium is left as a precipitate, and consequently calcific bursitis develops.

There is a definite correlation between alkalosis and the protein deficiency mechanism of calcium precipitation and improper disposition. Poor absorption of protein correlates with a systemic alkalosis. The administration of betaine hydrochloride aids in the absorption of protein, and is also significant treatment toward the relative alkalosis.

A very common reason for negative nitrogen balance is excessive gluconeogenesis as the result of a sugar handling stress, such as hyperinsulinism or hypoadrenia. In this condition there is also usually a bowel problem resulting from excessive refined carbohydrates, which creates a malabsorption syndrome affecting protein utilization.

A deficiency in albumin (or other amino acids) will cause an increase of peripheral interstitial tissue fluid, and can frequently be seen in clinical evaluation as a pitting edema. Edema, ascites, thoracic cage fluid, semicircular canal fluid, or any peripheral tissue fluid should be evidence to examine for protein disturbances. **(See Edema section below)**

Laboratory evaluation of protein is helpful; however, more than just the total protein level must be observed. The albumin should be well within the normal range, and the albumin-globulin ratio (A-G ratio) should be normal. When the globulin is high in ratio to the albumin, this indicates that the body is withdrawing albumin from a body compartment, such as the joint surfaces or synovial membrane. If the total protein and the A-G ratio are normal, but the serum globulin is above 3 gm.%, a protein deficiency is indicated. The protein in the blood stream is kept normal at the expense of other tissues, which give up protein. When albumin is withdrawn from tissues to make up blood protein, globulin, which is a much larger molecule, is also pulled, causing a rise in the serum globulin level.

In the presence of protein deficiency, even though the total blood protein may appear normal, there will be many symptoms and conditions – such as anemia, fatigue, sensitivity to cold, myalgia, and possibly atrophy.

As the joint surfaces and synovial membranes are depleted of protein in a protein deficient condition, pathology is more readily developed. The intra-articular fluid balance is disturbed and barometric pressure changes cause the fluid within the joint to painfully expand the joint capsule. As protein is withdrawn, calcium – which has been held in combination with the protein – becomes separated and is precipitated, depositing in bone tissue as hypertrophic arthritis.

Treatment depends upon evaluating the patient's protein intake, including all of the essential amino acids, hydrochloric acid, and production of enzymes from the small intestine and the pancreas. General bowel action must be evaluated and treated if indicated. The usual approaches of applied kinesiology to these organs are indicated, with specific indication to evaluate for the temporal bulge cranial fault and spinal lesions for the improvement of hydrochloric acid production. The liver must be evaluated thoroughly for its role in the production of albumin, and its other roles in protein metabolism.

The patient probably will not gain relief from the joint problem immediately upon obtaining improved absorption of protein. The first protein increase only starts making up the protein deficiency within the tissues, caused by prolonged protein yielding by the tissues. Until sufficient time has been allowed for the tissues to rebuild, there will still be inadequate tissue capability.

When the small intestine is found to be involved, applied kinesiology examination of the pancreas, liver, and gall bladder is indicated. For effective function of the small intestine, it is necessary that an alkaline medium be maintained. This comes primarily from the secretion of sodium bicarbonate and potassium bicarbonate from the pancreas. When there is inappropriate response, either in weakness of response or failure to hold response by the usual nutritional factors for the pancreas, consider adding sodium bicarbonate or potassium bicarbonate one-half hour after meals. The amount should be approximately one-quarter teaspoon after each meal; be certain that the patient does not take it with the meal because it will counteract the effect of hydrochloric acid in the stomach. This alkaline medium in the small intestine is necessary for lipolytic and proteolytic action.

Acid-Alkaline Balance

Acid-alkaline balance within the body is very much stabilized by the buffer system. Actual measuring of the pH of the blood may show very little shift taking place. A shift toward acid or alkaline can take place systemically as the result of varied diets and other metabolic processes, but no major changes are made in the actual pH of the blood, which is held to its close tolerance by the buffer system. A relative alkalinity may cause calcium to be thrown out of solution and deposited into connective tissue, frequently those of the hard-working joints of the body. Deposited calcium and other mineralization are often seen on x-ray in the costal-cartilage of individuals with degenerative joint disease and with a tendency toward calcific bursitis. Previously, measurement of the salivary pH was used as an indicator of the blood pH. It was found that the saliva does not always tend toward an alkaline level when the blood pH varies toward alkaline. There has been, however, a correlation that when the salivary pH is off toward acid or alkaline there is a probability of an acid base balance shift in the blood stream – not necessarily paralleling the shift of the saliva. The normal saliva pH range is about 6.5 – 7.0. The salivary pH can be measured effectively using indicator paper available in rolls from several of the nutritional supply houses and from medical supply stores.

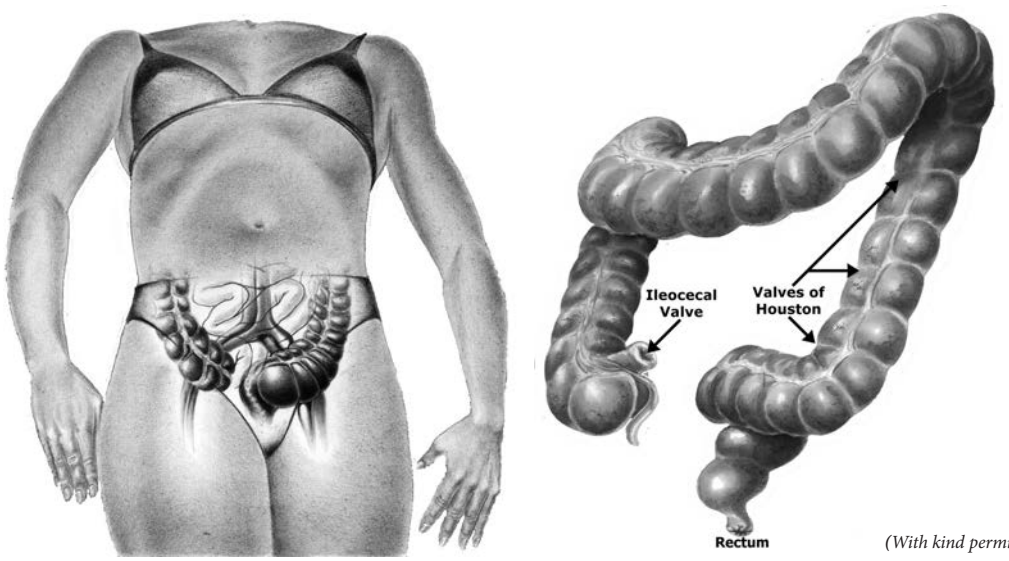
The folk remedy of apple cider vinegar for "arthritis" often improves a relative alkalosis, whether or not a true arthritis condition is present. **(Jarvis, 1985)** It tends to eliminate the joint pains, general achiness upon arising in the morning, etc.

Relative alkalosis commonly found in degenerative joint disease is also correlated with calcific bursitis. Before the diagnosis of calcific bursitis is made, the calcium deposit should be observed on x-ray. To observe this deposit, it is often necessary to do many x-rays of a joint, looking at the articulation from various angles. This is particularly true in the subdeltoid and supraspinatus calcific bursitis.

The shoulder is where more specific bursitis is found than any other area. The strain of muscles working in disharmony can be the possible cause of mechanical stress, complicated by metabolic problems like an acid-alkaline imbalance. The muscles of the shoulder should be evaluated for reactive muscles, weak muscles, hypertonic or shortened muscles, etc. As with many shoulder problems, the entire body's structural balance from the feet to the calvarium must be correlated.

An excellent method to shift an individual from a relative alkalosis to normal is nutritional supplementation in the form of acid calcium. Along with the acid calcium, vitamins A, C, and E are utilized. **(Schmitt, 1990)** When a low potency source of these nutritional products is used, it is given on an hourly basis for the first few days. An indication that the patient is shifting toward a relative acid

SYMPTOMS OF ALKALOSIS	SYMPTOMS OF ACIDOSIS
Heavy, slow pulse	**Suffocation symptoms**
	Frequent sighing
Stiffness of joints	Breathlessness
	Dislike for closed rooms
Symptoms after resting:	High altitude discomfort
Night Cramps	Irregular respiration
Cough	**Hyper-irritability symptoms**
Circulation disturbances	Voice affected during stress
Aching worse upon arising	Tachycardia
in the morning, eases with activity	Photophobia
Skin symptoms	Dysphagia
Dry	Restlessness
Thickened	Insomnia
Burning	Frequent contraction of erector
Itching	papillae
Formication	"Cold sweat" type perspiration
Rapid clotting time	**Dehydration symptoms:**
Feel worse after eating	Dryness of skin
	Dryness of mouth
	Dry, hard stool
	Diminished urination
	Diminished perspiration



(With kind permission, ICAK-USA)

The Large intestine and AK

balance is when they start yawning and shows other indications of shortness of breath. At this time, reduce the acid calcium dosage to one tablet. Continue to reduce it if these breathlessness symptoms do not abate.

If the symptoms are due entirely to calcium precipitation from a relative alkalosis, the patient should get relief within 24 to 48 hours. Within approximately one week, the calcium deposit in the bursa will be improved on x-ray. Calcium deposits observable on x-ray are not solid calcium but are, rather, of a toothpaste consistency *in vivo*.

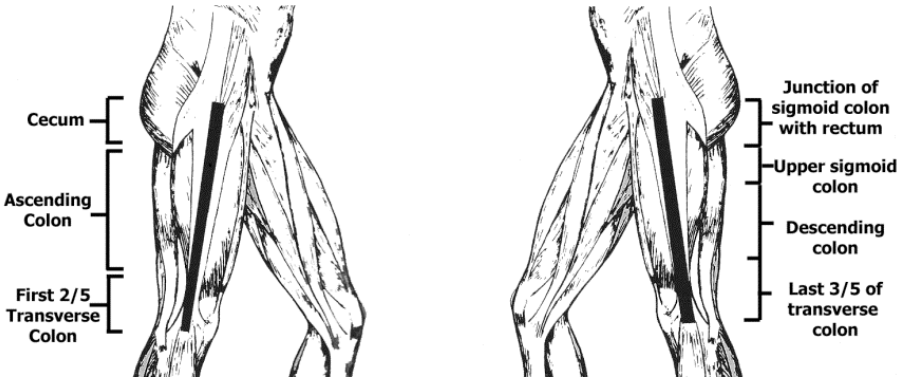
Applied Kinesiology and the Large Intestine

The large intestine is primarily thought of as a "garbage dump". Treatment is directed to the large intestine only when the garbage is dumped too fast (diarrhea) or too slow (constipation), or when it aches or gives symptoms of discomfort. A close look at large intestine function and dysfunction reveals the etiology for many health problems which are rapidly on the rise today. Every new patient entering natural health care should have his or her large intestine evaluated, at least on the consultation level, by the doctor.

It is interesting to note that the diseases associated with large intestine dysfunction are diseases which have developed in the last century. Their development has paralleled the dietary change, which is the primary cause of large intestine dysfunction. Two primary causes of the large intestine dysfunction are 1) an increased usage of refined carbohydrates, especially white sugar and white flour, and 2) the reduction of dietary roughage. Epidemiological studies of tribal communities in Africa and Oceania, who have retained their original diets, show a low incidence of these diseases. **(Price, 2003)** Both of these groups have diets which are high in fiber and low in refined carbohydrates. When these ethnic groups migrate to Europe or the United States and change their dietary habits, they become just as susceptible to the diseases as the Europeans and the Americans. As we study individual conditions, we find a common etiology. Accordingly we find the treatment to be similar and relatively simple, considering the major implications of the conditions involved.

Large Intestine and Rectal Cancer

The activity in the large intestine varies greatly between those on a high roughage diet and those on a low roughage diet. An individual on a high roughage diet has



The location of active neurolymphatic reflexes for the tensor fascia lata helps diagnose which area of the large intestine is involved. Bilateral TFL weakness indicates possible hypochromic anemia. Check adductors when TFL is weak.

voluminous bowel movements and the material moves rapidly through the large intestine, with 80% of the material removed from the bowel in less than a day and a half. Those on a refined, low roughage, American-type diet have a transit time through the body of about 3 to 4 days, with the material being in the large intestine most of that time.

There are two significant types of bacteria in the large intestine of the individual who consumes the typical low-roughage Standard American Diet, or SAD. These are known as *bacteroides* and *bifidobacteria*. **(Hill et al., 1971)** These cause the bile acid cholic acid to be converted to apcholic acid, and the bile acid deoxycholic acid to be turned into 3-methyl-cholanthrene. Both of these chemicals developed from the bile acids are carcinogens. The basic fact that these carcinogens are developed in the colon, added to the fact that there is stasis of the colon and that the consequent slow movement allows the chemicals to be in contact with the colon walls for long periods of time, leads to the ominous possibility of cancer developing.

The dominant bacteria in individuals with high fiber content diets are *streptococcus* and *lactobacillus*. These bacteria do not break down the bile acids, and these individuals have a much higher amount of bile acids in the feces and serum. **(Antonis & Bershon, 1962)**

As we ponder the tragic increase of large intestine malignancies, it stands to reason that the cause of the condition is contact with carcinogenic substances. It is significant that the incidence of cancer increases along the progression of the large intestine, the highest incidence being at the rectum. The only exception to this is the cecum, which also has a high occurrence of cancer.

The answer to the problem is prevention. Regaining normal bowel chemistry and bacteria, along with the rapid removal of waste products, is the preventative approach to cancers in the large intestine. **(Cheraskin et al., 1968)**

Ileocecal Valve Syndrome

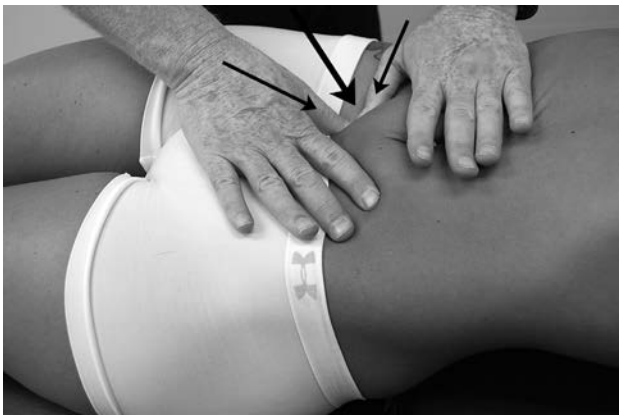
The ileocecal valve is at the very end of the small intestine (ileum) and connects it to the first part of the large intestine. If you imagine a line from the umbilicus to the right anterior superior iliac spine, this valve would be located just below the midpoint of that line. Dysfunction of the ileocecal valve has, in one report, been associated with low back pain (86.6%). **(Pollard, 2006)** A commonly observed sign in AK practice associated with an active ileocecal valve syndrome is acute onset of disabling pain, typically in the low back, but possibly anywhere in the body, with no apparent etiology. **(Shin, 2004)** Commonly, the low back pain will be mistaken for a lumbar disc syndrome. Just as frequently, symptomatology in the digestive system will be the primary symptom.

The valves of Houston, or transverse folds of the rectum (TFR), though different in structure and function from the ileocecal valve, do have a role in regulating transit through the intestinal tract. Most folds extend beyond the middle of the rectal lumen, are thicker at the base, and contain smooth muscle fibers. The TFR divide the rectum into compartments with an alternating side-to-side arrangement that creates a compartmentalization and shelving action, potentially responsible for retardation of stool movement in the rectum**. (Shafik, 2001)** The valves of Houston can produce similar symptoms.

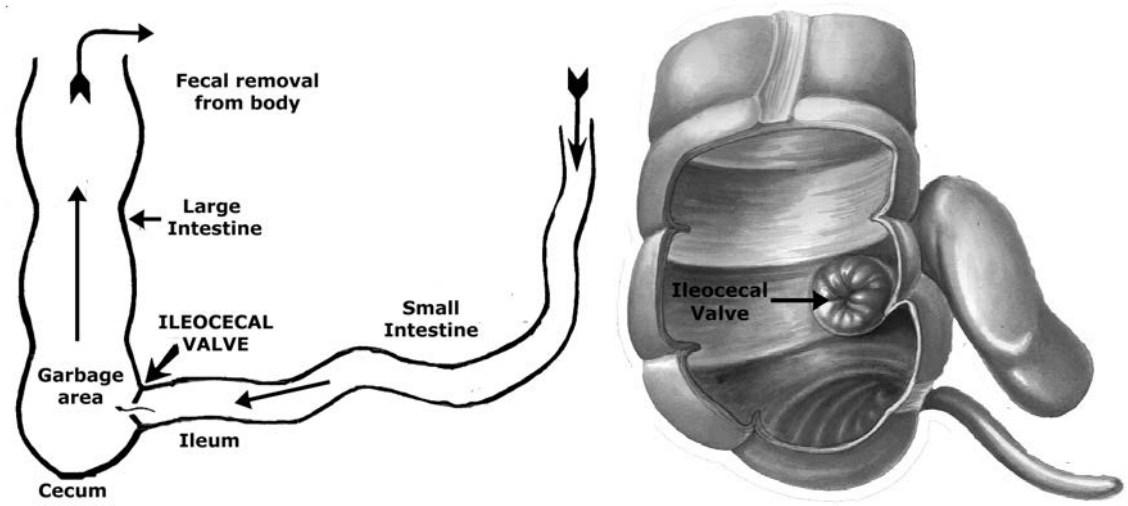
Symptoms that can occur with "open" or "closed" ileocecal valve syndromes include pain in the area of the valve (can be mistaken for right ovary or appendix pain), dizziness, low back pain, shoulder pain (mainly on the right side), nausea, faintness, paleness, sudden thirst, bad breath, black circles under the eyes, and ribbon like stools. Duffy reports on the association of open ileocecal valves in 12 cases of carpal tunnel syndrome. **(Duffy, 1994)** Ileocecal valve syndrome may also aggravate dysmenorrhea and endometriosis and should be evaluated in these conditions. Alis & Alis **(2004)** present a case of symptomatic endometriosis successfully treated with AK methods.

Open ileocecal valves often cause patients to suffer from loose bowels, emotional lability, **(Lebowitz, 1984)** and exhibit vitamin C deficiencies (their vitamin C is exhausted detoxifying the backflow of fecal material). Closed ileocecal valves tend to cause constipation. **(Maykel, 2004)** In many AK case and case series reports, diarrhea caused by the open ileocecal valve syndrome would improve the moment the ileocecal valve was treated.

Schmitt and Morantz **(1981)** observe that the ICV is related to the kidney acupuncture meridian. That is why the use of K-7 and B-58 for ICV problems has been standard treatments within AK for the ICV syndrome.



Therapy localization and challenge to the ileocecal valve followed by mobilization of the ICV



Ileocecal valve

> "It therefore often becomes necessary to take the entire acupuncture system into consideration in order to correct the recurrent ICV. For example, occasionally when you back up the acupuncture system using the law of 5 Elements, the problem turns out to be coming from a lung meridian involvement which is related to a T-3 subluxation. This makes no sense whatsoever, neurologically, but on an acupuncture basis, it explains how a T-3 subluxation could cause an ICV problem." **(Schmitt & Morantz, 1981)**

Schmitt **(2002)** also presents the concepts of Michael Gershon, MD (author of ***The Second Brain: A Groundbreaking New Understanding of Nervous Disorders of the Stomach and Intestine***) and shows the neurologic rationale behind the AK approach to the enteric nervous system. Fatty acids anywhere in the intestinal lumen stimulate the ENS to decrease peristalsis of the gut at the ileocecal area, what is called in AK the closed ileocecal valve (ICV) syndrome. This reflex exists to keep undigested fat from entering the large intestine, where it may stimulate the growth of unfriendly flora. Carbohydrate also stimulates the enteric nervous system and produces an open ileocecal valve syndrome. The gastrocolic reflex causes increased peristalsis in the large intestine following food intake that stretches the stomach. When the stomach is stimulated by the presence of food, the large intestine is stimulated to empty. These reflexes reflect 3 sensorimotor challenges developed in AK that evaluate whether or not they are functioning properly. It is proposed that these enteric nervous system concepts may be clinically applied by monitoring AK MMT outcomes following various specific sensorimotor challenges.

Normal large intestine function is seldom found in the general population. The most frequent involvement is large intestine stasis, which has many complications that may develop in later life. The significance of these complications ranges from mild general health interference to toxic reabsorption to the death-causing factors such as colon cancer. Every individual coming into the office should be evaluated for large intestine dysfunction, not just those with digestive disturbances. The large intestine, like other aspects of the digestive system, can produce symptoms in remote areas of the body.

There are other organs, glands, and structures which can interfere with normal digestive function. As we examine for any condition, we must remember the interplay which takes place within the body. Mentioned in this chapter are some of the more notable interactions. Quite often, the thyroid is involved with digestive function, especially that of the small intestine and urinary systems. The observant doctor will frequently notice a gurgling and heavy activity of the small intestine immediately after treatment of the thyroid by any one of the 5-factors of the IVF.

Alimentary Canal Flora

Dysbiosis of the intestinal flora has been implicated in the pathogenesis of atherosclerosis and dyslipidemias, hypertension, urolithiasis, pyelonephritis, gallstones, and hepatitis. **(Samsonova, 2010)** Dysbiosis, additionally, has been implicated in autoimmune disorders, allergy, and impaired resistance. **(Prakash, 2011)** Research indicates that dysbiosis may be a factor in the pathogenesis of obesity, inflammatory bowel disease, and gastroesophageal reflux disease (GERD). **(Henao-Mejia, 2012; Kim, 2010; Yang, 2009)** Changes in behavior and brain-derived neurotropic factor have been noted in dysbiotic mice. **(Bercik, 2011)**

Intestinal biota is adversely affected by antibiotics (prescription and as residue in foods), psychological and physical stress, sulfates and sulfites, and diets high in proteins, especially meat, and/or refined or simple carbohydrates. **(Hawrelak, 2004)** Bile appears to modulate the biota of the intestinal tract, as experimental occlusion of the common bile duct results in rapid changes in bowel flora and increased intestinal permeability. **(Fouts, 2012)** Proton pump inhibitors (PPIs) induce dysbiosis indicating

that adequate hydrochloric acid production and an acid food bolus entering the small intestine plays a role in normal gastrointestinal biota. **(Wallace, 2011)**

Inflammatory Bowel Disease (Ulcerative Colitis and Crohn's Disease)

Crohn's disease is a chronic, severe lymphatic congestion. There will usually be involvement of the quadriceps or abdominal muscles. Sometimes very prolonged neurolymphatic activity is necessary to bring this condition under control. There is a correlation between narcolepsy and small intestine involvement, especially if it has progressed to the state of Crohn's disease. The severe lymphatic congestion is due to the small intestine's very heavy workload. When there is improper small intestine activity, the area around the villi actually becomes clogged, causing further reduction in intestinal performance. The symptoms of narcolepsy develop as a result of an autointoxication from the activity of the small intestine. This also correlates with malabsorption syndrome and should be evaluated in hypoglycemia cases. The patient will often have an abnormal increase in the sensation of hunger, called bulimia. Sometimes they will literally stand in front of the refrigerator, knowing they want to eat but not knowing what.

Case studies indicate that ulcerative colitis and Crohn's disease are responsive to applied kinesiology therapies. Common findings are disturbances of the lumbar spine, an open ileocecal valve, and a need to eliminate nuts and seeds, grains, and milk from the diet as detected by AK MMT. **(Duffy, 1992)**

Patients with ulcerative colitis are frequently given Azulfidine and other sulfa drugs. A side-effect of the sulfa medication is that it rampantly destroys vitamin C. Patients who have colitis or irritable bowel syndrome or the more severe ulcerated colitis should be evaluated for the tissue levels of vitamin C, since it is one of the necessary natural agents for controlling inflammation. It might be observed that Beauchamp et al. **(2005)** suggest that 3.5 tablespoons of olive oil has the same effect as a 200-mg tablet of ibuprofen. While pharmacological treatment of inflammation remains an option for patients, there are now well-established strategies for modulating inflammation via dietary and nutritional manipulation. **(Gaby, 2006)**

Adrenal involvement in large intestine dysfunctions

Accompanying an increase in refined carbohydrates and a loss of fiber in the Standard American Diet is the problem of a functional hypoadrenia. As a result of functional hypoadrenia, the individual may have a lack of mineralocorticoids and glucocorticoids, the pro-inflammatory and anti-inflammatory hormones produced by the adrenal glands. Colitis often accompanies functional hypoadrenia and can ultimately end in diverticulitis and ulcerative colitis. As well as making dietary changes for normal colon action, it is important to support the adrenal if it is involved with inflammatory changes in the large intestine.

Variations are found in anti-oxidant enzymes in IBD subjects. Low plasma selenium and glutathione peroxidase has been found along with low intestinal mucosal concentrations of zinc and metallothionein, a zinc dependent enzyme. **(Sturniolo, 1998)** Metallothioneins, antioxidants and regulators of the transcription factor nuclear factor (NF)-kappaB, are found to be abnormally expressed in IBD subjects, indicating an inborn error of zinc homeostasis. **(Waeytens, 2009)** Intestinal mucosal copper/zinc isoform of superoxide dismutase (Cu/Zn-SOD) has also been found to be low in IBD subjects. **(Kruidenier, 2003)** N-acetylcysteine and l-carnitine have been found to restore intestinal mucosal glutathione peroxidase levels. **(Cetinkaya, 2006; Nosáľová, 2000)**

The pro-inflammatory metabolites of arachadonic acid, prostaglandins E2 and thromboxane B2, are elevated in the colonic mucosa in ulcerative colitis. **(Pavlenko, 2003)**

Melatonin appears to play a role in moderating the mucosal inflammation of IBD, indicating that abnormal circadian rhythm may be a factor in the pathogenesis. **(Necefli, 2006)**

Methylsulfonylmethane (MSM), an organosulfur compound that appears to enhance glutathione peroxidase levels, has been shown to decrease intestinal mucosa damage in IBD. **(Amirshahrokhi, 2011)** Studies have shown that vitamin E is anti-inflammatory and glutathione peroxidase sparing of IBD intestinal mucosa. **(Tahan, 2011; Beno, 1997)**

Dietary phenethylisothiocyanate, a natural constituent of foods from the brassica family (broccoli, spinach, cabbage, cauliflower, Brussels sprouts, kale, collard greens, bok choi, kohlrabi), improved body weight and stool consistency and decreased intestinal bleeding and mucosal inflammation in IBD mice. **(Dey, 2010)** It is interesting to note that cabbage juice is a traditional remedy for ulcerations of the stomach and intestines.

Always rule out underlying intestinal infection, especially *Entamoeba histolytica*, in patients presenting with IBD. **(Horiki, 1996; Chan, 1995; Patel, 1989)**

Ulcers of the intestinal tract is one of the three conditions of the Triad of Selye, three conditions always found in lab animals if stressed sufficiently to induce adrenal fatigue. The Triad of Selye is (1) hypertrophy of the adrenal glands, (2) atrophy of the lymphatic system, and (3) ulcers in the intestinal tract. **(Selye, 1978)**

Calcium decreases the inflammatory effect of bile acids on intestinal mucosa. **(Wargovich, 1983)** Vitamin D receptor polymorphisms **(Naderi, 2008)** and vitamin D and K deficiencies **(Nakajima, 2011; Kuwabara, 2009)** are common in IBD populations. Some studies have proposed that low vitamin D plays a role in the pathogenesis of IBD. **(Souza, 2012; Ananthakrishnan, 2011)**

Celiac disease is a very specific type of inflammatory bowel disease that is caused by gluten allergy and avoidance of all gluten is the primary therapy. Besides the gluten containing grains rye, wheat, and barley, the patient must be aware of gluten used as a food additive.

Interestingly, restless leg syndrome (RLS) occurs frequently in association with Crohn's disease and may be an extraintestinal manifestation of the disease. **(Gemignani, 2010)** Other diagnostic considerations for the syndrome are uremic or diabetic neuropathy, Parkinson disease, multiple sclerosis, caffeine, tricyclic antidepressants, barbiturates, benzodiazepines, and anemia.

Irritable Bowel Syndrome (Mucous Colitis)

Mucous colitis (IBS) is a functional inflammatory condition of the large intestine. Symptoms can be useful to discriminate between IBD and IBS, with straining upon defecation, diarrhea, and abdominal bloating.

Probiotic support appears to suppress inflammation and colonic barrier disruption induced by stress; the most beneficial strains appear to be the *Lactobacilli* group and *Saccharomyces boulardii* and the *benefits extend to IBD.* **(Agostini, 2012; Goldin, 2008)** Bile acid malabsorption may be a trigger for diarrhea predominant IBS and *Lactobacilli* and *Bifidobacteria* subspecies are able to deconjugate and absorb bile acids. **(Camilleri, 2006; Smith, 2000)**

Food allergy, as measured via IgE and IgG antibodies, is more common in IBS populations when compared to controls, particularly to milk, wheat, and soy. **(Carroccio, 2011)** A food allergy model for IBS is supported by the observation that both numbers of mast cells and their mediators are increased in the intestinal mucosa for patients with IBS. **(Walker, 2008; Kalliomäki, 2005)** Lactose intolerance should be considered in patients with IBS as well. **(Vernia, 2001)**

IBS populations have higher arachidonic acid (AA) levels than control populations, along with the pro-inflammatory metabolites of AA, prostaglandin E(2) (PGE(2)) and leukotriene B(4) (LTB(4)), suggesting that prostaglandin imbalances are a factor in the pathogenesis of IBS and dietary therapy should include decreasing AA rich foods in the diet and increasing omega 3 fatty acids to improve prostaglandin balance. **(Clarke, 2010)** Intake of hydrogenated oils (HOs) in the diet should be limited in IBS patients since HOs impede conversion of the anti-inflammatory prostaglandin groups PG1 and PG3 and aggravate PG2 dominance.

Activation of protease-activated receptors (PARs) in the intestinal tract modulates motility, inflammation, visceral nociception, and epithelial functions (immune, permeability and secretory) suggesting a regulatory role for pancreatic proteolytic enzymes. **(Vergnolle, 2004)**

Peppermint oil is a promising therapeutic agent for IBS, possibly due to anti-spasmodic and choleretic effects. **(Grigoleit, 2005)** The choleretic benefit suggests looking to biliary insufficiency and low ejection fraction as a possible underlying and aggravating conditions in patients with IBS.

A study was also conducted to evaluate the comparative effect of yoga and conventional treatment in diarrhea-predominant irritable bowel syndrome (IBS) in a randomized control design. The patients were 22 males, aged 20-50 years, with confirmed diagnosis of diarrhea-predominant IBS. The conventional group (n = 12, 1 dropout) was given symptomatic treatment with loperamide 2-6 mg/day for 2 months, and the yoga intervention group (n = 9) consisted of a set of 12 asanas along with Surya Nadi pranayama (right-nostril breathing) two times a day for 2 months. All participants were tested at three regular intervals, at the start of study — 0 month, 1 month, and 2 months of receiving the intervention — and were investigated for bowel symptoms, autonomic symptoms,

Warrior Two pose and Utkatasana (Chair Pose) are purported to help with gastric and digestion problems

autonomic reactivity (battery of five standard tests), surface electrogastrography, anxiety profile by Spielberger's Self Evaluation Questionnaire, which evaluated trait and state anxiety. Two months of both conventional and yogic intervention showed a significant decrease of bowel symptoms and state anxiety. This was accompanied by an increase in electrophysiologically recorded gastric activity in the conventional intervention group and enhanced parasympathetic reactivity, as measured by heart rate parameters, in the yogic intervention group. The study indicates a beneficial effect of yogic intervention over conventional treatment in diarrhea-predominant IBS. **(Taneja et al., 2004)**

Many yoga asanas focus on moving the stomach and intestines in all directions to aid the digestive process. **(Shankardevananda, 2003; Iyengar, 1995)** This stimulates the flow of blood to all parts of the intestines and generally helps foods pass through the body.

Constipation

Increased sympathetic activity decreases bowel tone by decreasing peristalsis and tightening the sphincters. Increased parasympathetic activity increases bowel tone by increasing bowel wall peristalsis and relaxing the sphincters. Always consider autonomic tone when treating the patient suffering constipation. **(Lee, 1998)**

When the diet is highly refined, there is a slower passage of the large intestine contents and increased contractions of the large intestine to move the hard, dry material. The effort of peristalsis along the narrow colon stretches and eventually causes the pouches of diverticulosis. It is a short step to diverticulitis as a result of a piece of hardened fecal matter or fecalith blocking the opening to diverticula. Inflammation, infections, and abscesses can occur.

Underlying causative factors to consider in constipation are dehydration (may be aggravated by electrolyte deficiency), serotonin deficiency or receptor resistance, biliary and pancreatic insufficiency, low fiber diet, dysbiosis, food allergies, and hypothyroidism. **(Syrigou, 2011; Jamshed, 2011; An, 2010; Gale, 2009)**

Dehydration is an almost universal nutritional deficiency in our culture. Satisfactory respiration, digestion, assimilation, metabolism, elimination, waste removal, and temperature regulation are bodily functions that can only be accomplished in the presence of water. Water is essential in dissolving and transporting nutrients such as oxygen and mineral salts, vitamins and nucleoproteins via the blood, lymph, and other bodily fluids: these are the constituents of connective tissue. Effective nutritional testing requires that the mouth and tongue be moist, so that the nutrition can mix with the lingual receptors of the tongue. Good lymphatic function also depends on an adequate daily intake of water. For tissue healing to take place, water must be plentiful. A healthy person should consume a minimum of 1/3 ounce of water for every pound of body weight, and double this amount in times of stress or illness. This usually means drinking 6-8 glasses of water every day. This should be purified water, not coffee, tea, fruit juice, sodas, milk and other liquids. The AK test for dehydration can help the patient and the clinician recognize when there is a problem.

Applied kinesiology treatment can correct chronic constipation. A case report of treating lifelong constipation in a thirteen year old boy included chiropractic care to the lumbar spine, ileocecal valve, avoidance of common

Dehydration may be evaluated using the AK hair-pull test.

Ask the patient to tug the hair on their scalp and test a previously strong indicator muscle.

If it weakens, ask the patient to drink some water. Retesting the muscle in a dehydrated patient will usually show strengthening.

allergens (milk, corn, soy, wheat), and inclusion of dietary water-soluble fiber. **(Maykel, 2003)** Treatment of neurolymphatic (Chapman's) reflexes has been reported to be successful therapy for chronic constipation and associated viscerosomatic reflex low back pain in an AK setting. **(Caso, 2001)**

Chiropractic manipulation of the spine, cranium and neck has been shown to beneficially change bowel behavior, even in cases of chronic constipation, sometimes after just one treatment. **(Browning, 2009; Masarsky & Masarsky, 2001; Wagner, 1995)**

Diarrhea

Underlying etiological factors of diarrhea may be hypochlorhydria, pancreatic and biliary insufficiency, open ileocecal valve, and intestinal flora. **(Mossner, 2010; Sarles, 1989)** Consider possible gastrointestinal infection - bacterial, viral, mycotic, or parasitic. **(Rajamanicka, 2009)**

Saccharomyces boulardii has been shown to be an effective probiotic for treating infectious diarrhea. **(Dinleyici, 2012)** Consider activated charcoal as an adsorbent for diarrhea due to infectious, toxic, or inflammatory agents. **(Sergio, 2008)** Research has shown that vitamin A and zinc deficiencies predispose to chronic diarrhea. **(Bhan, 1996; Sinha, 1995)**

Infantile Colic

Research indicates that coliform concentration is higher in infants with colic when compared to controls and *Lactobacillus* cultures are antagonistic to coliform populations. **(Savino, 2011)** Feeding infants symbiotic formulas of both prebiotic and probiotic cultures has been found to be safe and to decrease incidence of respiratory infections during the first two years of life. **(Kukkonen, 2008)**

Breast-fed infants have significantly higher levels of coliform-antagonistic bifidobacteria and salivary IgA when compared to bottle-fed populations. **(Wold, 2000; Yoshioka, 1983)** Insufficient or imbalanced intestinal microbial flora has also been associated with atopic eczema in infants. **(Abrahamson, 2011)**

A study of 316 infants with the cooperation of 38% of the chiropractors in Denmark was conducted by Klougart et al. **(1989)** Based on the diaries of parents, 94% of the patients resolved their colic in 2 weeks or less (an average of 3 visits), with 23% of the patients resolving their colic after a single treatment. The upper cervical area was the most frequently adjusted. The authors noted that spontaneous resolution of infantile colic usually takes 12 to 16 weeks.

Obesity

Although obesity is commonplace today, **(Centers for Disease Control and Prevention, 2012)** it has not always been that way. In fact, even today in some tribal areas of Africa, India, and Oceania, and other areas where an unrefined diet is used, obesity is almost unheard of. According to the CDC, more than one-third of U.S. adults (35.7%) are obese. Approximately 17% (or 12.5 million) children and adolescents aged 2—19 years are obese. In 2010 no state in the United States had a prevalence of obesity less than 20%. Thirty-six states had a prevalence of 25% or more; 12 of these states (Alabama, Arkansas, Kentucky, Louisiana, Michigan, Mississippi, Missouri, Oklahoma, South Carolina, Tennessee, Texas, and West Virginia) had a prevalence of 30% or more.

There are two primary causes of obesity today. First are the so-called "empty calories" which add to the body's calorie count, but provide no material for healthy tissue-building. These items are primarily white sugar, white flour, and alcohol. The second item is a lack of fiber in the diet. A diet comprised primarily of fiber dense foods, such as fruits, vegetables, beans, nuts and seeds, and unprocessed grains requires more chewing, slower intake, and promotes a sense of satiety at lower caloric intake. **(Isaksson, 2012; Beck, 2009)**

A major key to many of the health problems resulting from large intestine dysfunction lies in increasing the fiber content of the diet. Unfortunately with today's refined, ultra-processed foods, it becomes almost impossible to obtain fiber from foods purchased at the supermarket.

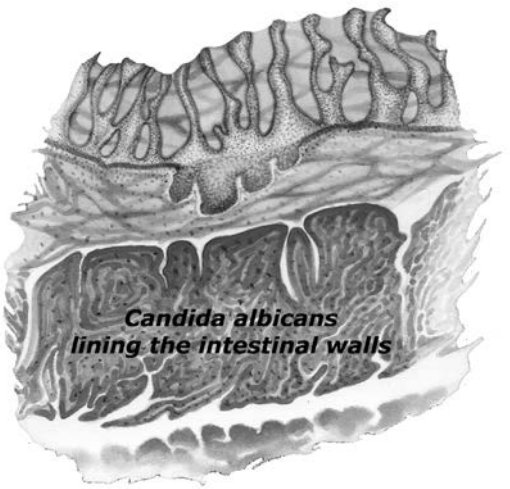
An easy answer is to include the so-called "waste products" from wheat in our diet. Bran can be purchased very economically from any health-food store and added easily to the diet. Make it a drink by mixing it with water or juice, or mix it with such foods as meat loaf, cereals, etc.

The amount of bran necessary for normal colon function varies with individuals. Start with one teaspoon three times per day, and increase until the desired results are obtained. Some individuals will get good results with three teaspoons per day; others will require several tablespoons per day. The basic criterion is that the individual have a well-formed, soft stool, preferably on a daily basis. The stool should be completely odor-free. The odor on the stool is the classic criterion for adequate bowel action. This gives indication of the bacteria content as well as whether putrefaction processes are taking place.

Flatulence may occur when first incorporating bran into the diet. The amount of bran ingested can be decreased until the flatulence diminishes and the digestive tract adapts to it, at which point the dosage can then be increased. If the problem persists consider the possibility of gluten allergy or hypochlorhydria.

Post-Antibiotic Effects and Candidiasis

Antibiotics are non-discriminating in their action and commonly result in intestinal dysbiosis. When antibiotics are given for an infection, they also affect the flora of the intestines. William G. Crook, MD, and author of *The Yeast Connection*, **(1986)** states that "Broad-spectrum antibiotics resemble machine gun-shooting terrorists in a crowded airport. While they're killing enemies, they also kill friendly and innocent bystanders. In a similar manner, antibiotics knock out friendly bacteria on the interior membranes of a person's body while they're eradicating enemies. When this happens, yeasts flourish and put out a toxin that affects various organs and systems in the body, including the



immune system." A major side effect of chronic candidiasis is persistent inflammation of the intestinal wall and increased intestinal permeability. This permits absorption of multiple antigenic and toxic substances into the bloodstream, resulting in allergies. **(Schmidt et al., 1994)**

Sometimes it is necessary, after antibiotic therapy has been used, to re-implant bacteria or provide a better culture medium for their growth. Support with probiotic and prebiotic formulas may be needed to restore intestinal biosis. Using viable colonizing strains of lactobacillus acidophilus (small intestine) and bifidobacteria (large intestine) will check and inhibit growth of innate intestinal opportunistic yeasts. **(Chaitow & Trenev, 1989)** The dosage is usually ½ teaspoons in a glass of warm water three times per day for children and two to three capsules (one or two teaspoons powder) three times daily for adults. Temporary use of anti-fungal compounds may also be required to control yeast overgrowth in the intestinal tract. Prescription anti-fungals are rarely necessary; the anti-fungal action of caprylic acid, garlic, cinnamon, aloe vera juice, Spore-X (Nutri-West®), pau de arco, and other anti-fungal herbs is usually effective, if the intestinal flora is restored adequately.

Anyone with a great deal of yeast in the system may experience numerous "die-off" symptoms during the remission of the candida infestation. The yeast organisms carry poison within them. As they die, their membranes eventually rupture releasing the poison or toxins into the system. The actual "die-off" can cause some "I-just-don't-feel-good" types of symptoms. This signifies a healing crisis, and is evidence that recovery from candida infestation is progressing. Although patients sometimes get worse before getting better, once the poisons are flushed through, they usually continue to get well.

Limiting soluble carbohydrate intake during the phases controlling yeast overgrowth, restoring intestinal flora, and healing the inflamed and hyper-permeable intestinal wall will improve outcomes. AKMMT, elimination diet, or blood or skin allergy testing can also be used to identify allergenic foods that need to be eliminated from the diet to down-regulate intestinal wall inflammation and promote restoration of optimal permeability. It has been this authors' experience that the broad spectrum of mild and moderate food reactions found upon AK MMT and laboratory food allergy testing will resolve once digestion, intestinal flora, and gut wall permeability are restored.

While mushrooms, yeasts, and fermented foods (i.e. vinegar, soy sauce, miso) do not promote growth of intestinal yeasts, it is common for patients with intestinal yeast overgrowth to become immunologically sensitive to these foods and avoidance until the issue is resolved will promote recovery."

Incorporating healthy lifestyle changes make a big difference in fighting candida. First, the yeast must be killed, then the intestinal garden replanted, the immune system and digestive system strengthened, and diet and lifestyle modified. The most important factor involves withholding the type of food on which the yeast thrives. Using the AK MMT, the elimination diet, or blood or skin allergy tests that identify allergenic foods that need to be eliminated from the diet can down-regulate intestinal wall inflammation and promote restoration of optimal permeability. It is wise to leave out sugar, refined carbohydrates such as white bread and pasta, alcohol, and even honey. This strict diet regime is required only for a period of time – not forever. If successful in controlling candida symptoms within a few months, the patient can slowly reintroduce these allergenic foods into the diet.

Vinegar, soy sauce, and miso are several examples of fermented items that should be avoided by most candida patients. Restrict from the diet all yeast-containing foods for a minimum of three months. While candida is different from baker's yeast or brewer's yeast, patients are advised to adhere to the philosophy that if yeast is growing out of control in the body, any yeast may further upset the digestive system.

Expect to experience more allergic reactions to yogurt, cheese, or kefir containing some type of yeast. Yogurt has been over emphasized in many books. Actually, yogurt doesn't contain the curative properties touted mainly because its amounts of friendly bacteria, *acidophilus*, are miniscule compared to what is truly needed. Additionally, most yogurt products are loaded with synthetic sugars that are one of the basic foods of the candida organism in the first place.

It's smart to avoid any fungus-containing food such as peanuts and mushrooms. It's much easier to overcome candidiasis if the patient simply leaves out fermented and fungal foods – the yeasts, sugars, and alcohol – altogether. Antifungal foods such as garlic and olive oil (oleic acid) are good choices.

Fecal matter matters

We can now see how small and large intestinal dysfunction is endemic to modern life simply by the proliferation of over-the-counter preparations for large intestine or bowel control. There are heavy metal preparations to bring more fluid into the bowel, stimulants, bulks, items for starting the bowel, means for stopping the bowel, gums to chew, and enema bags. There are even high-colonic irrigating machines for home use.

Physicians do not always help these cases, because they reassure patients that it is normal for some people to have a bowel movement every 3rd or 4th day or even once a week; as long as the stool moved is soft, there is no need for concern. For too many years the accepted treatment for

conditions such as colitis, diverticulitis, and diverticulosis has been a bland diet – which is the wrong thing for the patient to do, even though the diet is a so-called soothing diet to the bowel.

Because there has been so much misinformation among the lay public, and from doctors as well, a major project of patient education lies before us. We must overcome these improper treatments and lack of concern, and in fact reverse certain types of treatments which, in the past, were thought to be correct, but are in reality making the patient worse.

Quite often obtaining normal large intestine function is extremely important in obtaining correction of the acute problem which brings the patient to the physician. Obviously in these cases we are concerned with getting the condition under control as rapidly as possible. The patient who is perhaps of greatest concern is the individual who has significant large intestine stasis and/or abnormal flora, but has no symptomatic complaint upon initial consultation. This is the person who has problems in the developmental stage. These potential problems are of major significance and early correction may possibly be life-saving. These are the conditions which must be ferreted out and correction obtained while correction is still possible.

Female Reproductive Systems

Infertility and Amenorrhea

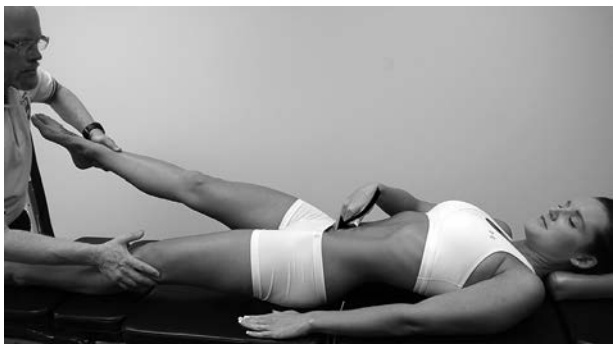
In a study by Wurn, 28 infertile women diagnosed with complete fallopian tube occlusion underwent a twenty session trial of manual therapies to address pain and restricted soft tissue mobility due to adhesions in the pelvic region. It resulted in unilateral or bilateral patency (as measured by hystero-salpingography) in 61% of the subjects. Subsequent to treatment, 53% of the subjects in the fallopian tube patient group reported natural intrauterine pregnancy. **(Wurn, 2008)** These findings conformed closely to a previous study by the same researcher. **(Wurn, 2004)**

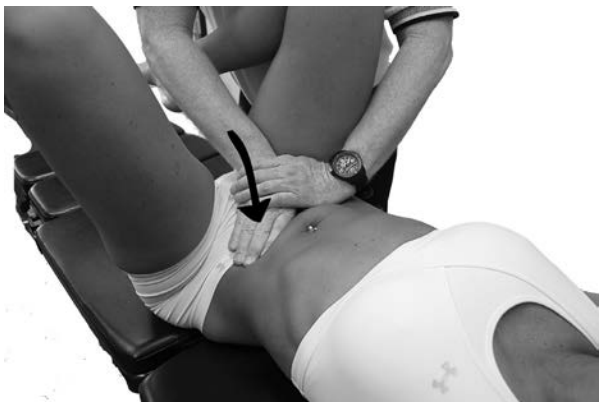
A number of case reports suggest that AK treatment is effective for resolving infertility. The non-invasive nature, safety, and cost-effectiveness of this conservative approach to infertility when compared to standard fertility medicine should be borne in mind. The AK therapies include chiropractic, craniosacral, meridian (acupuncture), and clinical nutrition. Based upon these reports, consideration should be given to pituitary function (pituitary drive technique), thyroid function (including Grave's disease), adrenal function, iron deficiency, and essential fatty acid deficiency, including wheat germ oil. **(Kaufman, 1996; Duffy, 1993; Heidrich, 1991)** AK combined with clinical nutrition has been successful in resolving amenorrhea. **(Kharrazian, 2007)**

Infertility has been linked to aberrant modulation of the hypothalamic-pituitary-gonadal axis. **(Scott, 1989)** AK methods for normalizing adrenal function, identifying food allergies, and decreasing mechanical stress to the uterus (uterine lift technique) show promise for resolving dysmenorrhea and infertility in some patients. **(Hickey, 2007)**

There appear to be four primary types of amenorrhea - hyperprolactinaemic, hypogonadotrophic, hypergonadotrophic, and normogonadotrophic. Hyperprolactinaemic amenorrhoea is often due to pituitary adenoma, hypergonadotrophic amenorrhoea is due to ovarian failure, and normogonadotrophic amenorrhoea is due to aberrant GnRH secretory patterns and is often secondary to PCOS. Hypogonadotrophic amenorrhoea is usually associated with stress and nutritional deficiency, often as a result of restricted dietary patterns and insufficient body fat. **(Crosignani & Vegetti, 1996)** Stress of various types, such as dieting, heavy training, or intense emotional events, appears to be a pathogenic factor for both normogonadotrophic and hypogonadotrophic patterns of amenorrhea. These forms of amenorrhea are mediated via the hypothalamus and may manifest with or without weight loss. **(Genazzani, 2010)**

Under-eating and overexercising are common stresses leading to amenorrhea. One study showed that twenty percent of ballet dancers studied were amenorrheic. **(Stokic, 2005)** It has been common to associate low body fat with amenorrhea, though some research challenges this common view and has indicated that low overall body weight has a closer correlation. **(Estok, 1991; Sanborn, 1987)** Always consider eating disorders in the low weight amenorrheic patient. **(Gentile, 2011)** Goodheart suggests that to restart the menstrual cycle, a course of increased calcium levels 1,500 mg. per day (the same as postmenopausal females), with 300 mg of zinc and 150 mg vitamin B6 daily may be useful. **(Goodheart, 1992)**

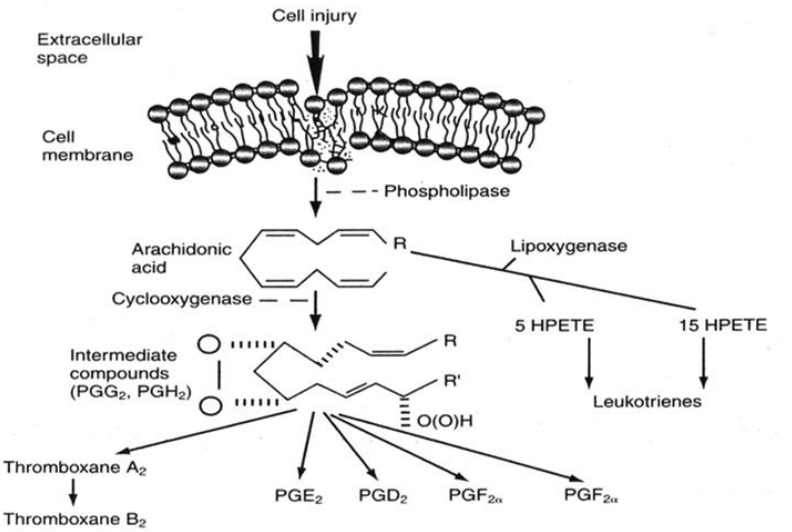




Uterine Lift technique: challenge and visceral manipulation

Dysmenorrhea

The pain of primary dysmenorrhea can be very intense. Pain threshold measurements to electrical stimulation of the skin, subcutaneous tissue and muscle have been seen to be lower than normal in women with dysmenorrhea, particularly in the rectus abdominus muscle. **(Giamberardino et al., 1997)** As reviewed in the text ***Applied Kinesiology Essentials***, muscle pain usually creates muscle weakness on the MMT, indicating the relationship between positive MMT findings and women with this condition. These conditions of the female reproductive system are characterized by a generalized hypersensitivity to painful stimuli, suggesting that these patients experience a wide spectrum of functional disorders. It is reasonable to suggest that even if only the muscular impairments (in addition to the other AK approaches) are corrected, significant improvements can be expected in these patients. It is also probable that addressing the physical impairments found in these patients will impact psychosocial disturbances as well, further advancing the goals of treating the patient's total problem.



Pain, prostaglandins and dysmenorrhea

Women suffering dysmenorrhea have been shown to have higher 17 beta-estradiol (E2) level, when compared to asymptomatic controls, in the latter half of the menstrual cycle and low progesterone levels, thereby creating a high estrogen/progesterone ratio. **(Zahradnik, 1984; Ylikorkala, 1979)**

Prostaglandin metabolism is an important consideration for management of dysmenorrhea. Dysmenorrhea and dysfunctional uterine bleeding are also associated with up-regulation of arachadonic acid-derived PG2 series prostaglandins, **(Coll Capdevila, 1997)** and management with NSAIDs to down-regulate production of PG2 has proven effective **(Dawood, 2007)**

Nitric oxide (NO) appears to play a role in modulating myometria and may play a role in managing dysmenorrhea. **(Wetzka, 2001)**

The prostaglandin fractions PGE2 and PGF2α have been implicated as the agents producing uterine smooth muscle contraction and vasospasm of the uterine arterioles, leading to ischemia and the cramping sensation of dysmenorrhea. **(Dawood, 1990)** Medications most commonly used to treat dysmenorrhea are the nonsteroidal anti-inflammatory drugs (NSAIDs) ibuprofen, naproxen, and mefenamic acid. These act by inhibiting cyclooxygenase in the inflammatory pathway reflecting arachidonic acid metabolism to the prostaglandins, as shown in the figure. **(Dawood, 1988)** However, the side effects of NSAIDs (gastrointestinal disturbances, nausea, vomiting, constipation, headache, vertigo, fatigue, and allergic reactions) have been documented **(Mehlisch, 1988; Shapiro, 1988; Calesnick, 1987)** and need to be taken into consideration.

A series of studies over the years have suggested that spinal manipulation is capable of relieving the symptoms of dysmenorrhea. **(Snyder, 1996; Boesler et al., 1993; Smith and Rogers, 1992; Kokjohn et al., 1992; Liebl and Butler, 1990; Arnold-Frochot, 1981; Thomason et al., 1979; Hitchcock, 1976)** The most compelling of these has provided a sound biochemical rationale for the analgesic effect for the intervention, demonstrating that spinal manipulation in the lumbar region also reduces the concentration of the metabolite of the PGF2α fraction (15-keto-13,14-dihydroprostaglandin F2α) in plasma fractions taken from patients 1 hour after treatment. **(Kokjohn, 1992)** A larger randomized controlled trial **(Hondras, 1999)** delivered inconclusive results; however, that investigation was compromised by the facts that (1) effleurage was applied to both control and experimental groups of patients and (2) pain upon entry and washouts of exercise and the taking of NSAID medications were eliminated, all reducing or eliminating the possible effects of spinal manipulation.

Higher levels of the prostaglandin, 6-Keto-PGF1 alpha, correlate with increased severity of dysmenorrhea in women with endometriosis. **(Koike, 1992)** Fish oil and olive oil have been shown to down-regulate production of 6-Keto-PGF1 alpha. **(Correa, 2009; Faust, 1989)**

Research indicates that functional bowel disorders may underlie dysmenorrhea. **(Crowell, 1994)**

According to Schmitt, **(1990, 1981)** graphing basal temperature for a complete menstrual cycle can give insight into the relative balance and ratios between progesterone and estrogen. Schmitt also states that scanty menses can indicate high thyroid, low estrogen, and/or high progesterone levels, whereas heavy menses can indicate a low thyroid, high estrogen, and/or low progesterone. Additionally, short menstrual cycles (<26 days) typically have a high estrogen/progesterone ratio, whereas long menstrual cycles (>29 days) typically indicate low estrogen/progesterone ratio. Goodheart suggests that a menstrual cycle of more than 28 days indicates a hypothyroid condition, and one of less than 28 days indicates a hyperthyroid one. These observations have been congruent with our clinical experience and can be a useful complement to physical exam and laboratory findings.

Vitamins B6, B12, and/or folic acid deficiency should be considered in women using hormone replacement or contraceptives. **(Veninga, 1984; Anderson, 1976)**

Carbamide has been researched as a possible useful agent to promote uterine smooth muscle relaxation with a therapeutic effect for dysmenorrhea. **(Novakovik, 2007; Cheuk, 1993)**

Massage therapy to the pelvic region has been shown to be viable therapy for relieving dysmenorrhea in women with chronic endometriosis diagnosed with laparoscopy. **(Valiani, 2010)**

Premenstrual Syndromes

Premenstrual syndrome (PMS) is related to dysmenorrhea, with as many as 150 symptoms. **(Sveinsdottir & Reame, 1991)**

Estrogen imbalances

A high estrogen-progesterone ratio is associated with premenstrual syndromes and is characterized by mood disorders. The PMS group shows water and salt retention, abdominal bloating, mastalgia and weight gain that is associated with high aldosterone levels. The group is also characterized by blood sugar handling stress (premenstrual craving for sweets — especially chocolate) increased appetite, palpitation, fatigue, headache, and even syncope. A rarer type of PMS is associated with polycystic ovary syndrome (PCOS), low estrogen, high progesterone, and high adrenal androgens. **(Abraham, 1983)**

PMS is usually associated with persistence of high estrogen after the normal mid-cycle ovulatory spike. This is usually due to insufficient clearance of circulating estrogen by the Phase II detoxification pathway in the liver. An increasing body of scientific data supports the hypothesis that conjugation (sulfation) and deconjugation (desulfation) of estrogens is important in the regulation of biologically active steroid hormones in target tissues as well. Polymorphisms appear to dramatically influence the rate of these processes. Methylation is the most quantitatively active pathway in the sulfation and desulfation of estrogens. Glutathione (GSH) is a major enzyme in this system and has major antioxidant/anti-inflammatory effects systemically, as well. **(Raftogianis, 2000)** Xenoestrogens can also alter endogenous estrogen signaling and disrupt normal signaling pathways. **(Watson, 2011)** Xenobiotic compounds may accentuate estrogen activity and be an underlying and aggravating factor in PMS.

Xenoestrogens tend to accumulate and persist in adipose tissue for decades and may cause long-lasting, adverse endocrine effects. Xenoestrogen compounds include dichlorodiphnyltrichloroethane (DDT) and its metabolites, bisphenols, alkylphenols, dichlorophenols, methoxychlor, chlordecone, polychlorinated benzol derivatives (PCBs), and dioxins. Besides being receptor competitors and having higher biological activity than endogenous estrogens, some xenoestrogens may interfere with the production and metabolism of ovarian estrogens. **(Lorand, 2010)**

Phytoestrogens and bioflavonoids can be used to down-regulate estrogen activity. Phytoestrogens have a lower level of estrogen activity at receptor sites and can be used to attenuate the estrogenic effects of xenobiotics. **(Zava, 1997)** Procyanidin compounds from red wine and grape seeds have shown suppression of estrogen synthesis through aromatase inhibition. **(Eng, 2003)**

Altered serotonin activity may also be a factor in the causation of PMS. **(Halbreich, 1993)**

The depression commonly associated with PMS may be due to the increased demand on vitamin B6 reserves as a cofactor in conjugation during the luteal phase of the menstrual cycle and the need for B6 as a cofactor in the production of 5-hydroxtryptamine (serotonin). As high as 31% resolution of premenstrual depression with vitamin B6 supplementation has been reported in a group of women taking oral contraceptives. Possible symptoms of vitamin B6 deficiency include hyperirritability, loss of appetite, loss of weight, general weakness, lassitude, confusion, and a hypochromic, microcytic anemia with a high serum iron level. **(Prothro, 1981)** The usefulness of B6 and, to some degree magnesium, has been confirmed by other studies. **(De Souza, 2000; Doll, 1989; Bermond, 1982)** The patient may be advised to eat whole grains, nuts, seeds, beans, lentils, liver, organ meats, wheat germ, and Brewer's yeast. In a literature review, Douglas **(2002)** also supports calcium supplementation in managing PMS.

Prostaglandin imbalance seems to play a role in many cases of premenstrual syndrome and dysmenorrhea with a deficiency of dihomo-γ-linolenic acid (DGLA) being the most common pattern. Support DGLA levels by providing gamma-linolenic acid (GLA) with evening primrose, black currant, borage oils, and/or spirulina. The cofactors magnesium, zinc, vitamin C, B3, and B6 may be needed for conversion of GLA to DGLA.

A crossover randomized controlled trial, **(Walsh et al., 1999)** a case series, **(Wittler, 1992)** and numerous case reports have shown that spinal manipulative therapy may alleviate this condition. **(Masarsky & Masarsky, 2001)**

Breast tenderness is a frequent symptom of PMS. To determine whether AK technique was of benefit to women with breast pain, an open pilot study was conducted at the Hedley Atkins Breast Unit, Guy's Hospital, London, UK. **(Gregory et al., 2001)** Eighty-eight newly presenting women with self-rated moderate or severe mastalgia were recruited for the study. The AK treatment involved rubbing the neurolymphatic reflexes of the TFL muscle while monitoring painful areas of the breasts. The women were predominantly pre-menopausal, and patients with both cyclical and non-cyclical pain were included in the study. Patients' self-rated pain scores, both before and immediately after applied kinesiology, were compared, together with a further score 2 months later. Immediately after treatment there was considerable reduction in breast pain in 60% of patients with complete resolution in 18%. At the visit after 2 months, there was a reduction in severity, duration and frequency of pain of 50% or more in about 60% of cases ($P<0.0001$). This preliminary study suggests that applied kinesiology may be an effective treatment for mastalgia, without side-effects and merits testing against standard drug therapies.

Menstrual headaches were successfully treated with AK protocols that included craniosacral and chiropractic manipulative therapies, adrenal support, clinical nutrition and the avoidance of aspartame along with good food combining principles. **(Calhoon, 2004)**

Endometriosis

As mentioned previously, endometriosis has been associated with dysmenorrhea and high levels of 6-Keto-PGF1 alpha (a metabolite of PG2), and fish oils have been shown to lower the levels of this prostaglandin. Other approaches like this would be to decrease arachadonic acid intake (less animal fats), avoid trans-fats, and increase the intake of omega-3 and omega-6 fatty acids to improve the ratios between prostaglandin families (Pg1, PG2, and PG3) and to lower levels of pro-inflammatory PG2 and higher levels of the anti-inflammatory PG1 and PG3 families.

AK care has been successful in the treatment of endometriosis in a 25 year old patient with chronic, severe abdominal pain associated with her menstrual cycle and dyspareunia despite laparoscopic surgery for endometriosis six months prior to treatment. Care included chiropractic, dietary modification (primarily avoidance of refined food, use of digestive enzymes, and progesterone cream to address estrogen dominance.) **(Alis & Alis, 2004)**

Polycystic Ovary Disease

Estrogen activity may by high due to insulin resistance and resulting polycystic ovary disease (PCOS). The solution here is improved diet with carbohydrate restriction and intense interval exercise to improve insulin sensitivity. Chromium, B fraction (primarily thiamin), and optimizing mitochondrial function (mostly citric acid/Kreb's cycle and electron transport systems) is often helpful for improving blood sugar regulation. An estimated 90% of the American population does not consume the minimum recommended intake of 50 micrograms of chromium daily. **(Shils et al., 1999)** Researchers at the United States Department of Agriculture have estimated that up to 25% of heart disease in the United States could be prevented merely by consuming adequate quantities of chromium. Human subjects who take 200 micrograms daily of chromium supplements lose more fat and gain more lean tissue. **(Gaby, 2006)** Another study found that chromium provided significant drops in fasting blood glucose in diabetics. **(Evans et al., 1993)** Several studies have shown that chromium supplements lower fasting blood glucose levels in normal healthy individuals. **(Anderson et al., 1983)**

Vaginitis/Vaginosis

Women who have higher than normal estrogen levels, diabetes, and vaginal dryness possess predisposing factors for vaginal candidiasis and vaginosis. **(Dennerstein, 1998)** Soy isoflavones have been reported to be effective management for menopausal vaginal dryness. **(Li, 2010)**

Supplementation of vitamin A and beta-carotene has been reported to decrease the risk for bacterial vaginosis. **(Christian, 2011)** Vaginal application of vitamin C has also shown promise for management of vaginosis. **(Petersen, 2011)**

Oral and vaginal *lactobacilli* probiotic therapy has been shown useful for management and decreasing risk for vaginosis. **(Delia, 2006; Reid, 2001)** The approach appears to be more effective than metronidazole vaginal gel. **(Anukam, 2006)**

Broda Barnes **(1976)** has linked hypothyroidism (and hypoiodinism) with uterine fibroids and vaginitis. In women with thick mucus secretions in the vaginal area, iodine should be considered. **(Goodheart, 1998; Schmitt, 1990)** Goodheart also suggests vitamin B and wheat germ oil and have been successful for many women with vaginitis.

Benign Uterine Fibroid/ Leiomyoma

Risk factors for the development of uterine fibroids include obesity, **(Shikora, 1991)** low vitamin A status, **(Martin, 2011)** and zinc deficiency. **(Sahin, 2009)** Cadmium toxicity appears to be a risk factor for both uterine fibroids and endometriosis, **(Jackson, 2008)** and lycopene supplementation, like iodine, may decrease the risk for developing fibroids. **(Sahin, 2004)**

Serum estrogen and progesterone levels do not differ between normal myometria and uterine leiomyometria groups, but uterine leiomyoma tissues are characterized by significantly increased estrogen and progesterone levels and a high estrogen-progesterone ratio. **(Potgieterm, 1995)**

Male Reproductive Systems

Prostate Disease

The prostate gland is part of a man's urinary and sex organs. It is about the size of a walnut, doughnut-shaped, and it surrounds the urethra which exits the bladder. The urethra has two functions in men. The first is to carry urine from the bladder, the second to carry semen during sexual climax.

Over 50% of men will develop an enlarged prostate (or benign prostatic hypertrophy) in their lifetime. A man over 50 having problems urinating is usually suffering from an enlarged prostate. As men get older, there is a tendency for the prostate to grow. As it grows, it squeezes the urethra. Since urine travels from the bladder through the urethra, the pressure from the enlarged prostate may affect bladder control. The symptoms of BPH are:

- A frequent and urgent need to urinate that occurs first at night.
- Trouble starting a urine stream. Straining required to get the urine flowing.
- A weak stream of urine. Takes longer to urinate than when younger.
- Only a small amount of urine flows.
- Feeling that more urine remains, even when finished.
- Leaking or dribbling.

Aside from the symptoms associated with BPH, the condition is a risk factor for developing prostate cancer and merits the clinicians' focused management. The healthcare burden of prostate disorders is evidenced by the 2 million or so physician office visits per year in the US. **(Chaitow & Jones, 2012)** It is often recommended that men over the age of 40 have yearly prostate exams. The exam involves a

doctor inserting a gloved finger into the rectum and feeling the lower part of the prostate for any abnormality. However, in the case of BPH, often the prostate has not enlarged to a point that can be recognized by physical exam. Ultrasound measurements are another common diagnostic method, and then a blood test may be used to differentiate BPH from a more serious prostate cancer. Surgery for BPH may have only temporary, but sometimes permanent effects on sexual function. **(Garnick, 2012)** Most men recover sexual function within a year after surgery. The exact length of time depends on how long the symptoms had been present before the surgery was done and on the type of surgery. Side effects include erectile dysfunction and loss of bladder control as well as semen that no longer goes out of the penis during orgasm. Instead it goes backwards into the bladder.

Risk for developing benign prostatic hypertrophy (BPH) has been associated with high testosterone levels, but research indicates that testosterone (TT) has an anti-inflammatory effect on prostate tissues and development of BPH has closer association with dihydrotestosterone (DHT) levels. **(Vignozzi, 2012)** Specifically, there appears to be a direct association between larger prostate volume and higher DHT and DHT/TT levels. **(Liao, 2012)**

The enzyme 5-alpha reductase converts testosterone to dihydrotestosterone and inhibition of 5-alpha reductase has been shown to "block the undesirable effects of T on the prostate, without blocking the desirable anabolic effects of T on muscle, bone, and fat." **(Borst, 2005)** Zinc has been shown to be an important 5-alpha reductase inhibitor that decreases prostate weight in rats without affecting testicular function. **(Fahim, 1993)**

Saw palmetto (Serenoa repens, sometimes referred to as *sabal* in Europe) grows naturally in the southeastern United States, including Georgia, Mississippi, and particularly Florida. Saw palmetto has been shown to inhibit 5 alpha-reductase in some studies, though there is some controversy as to its therapeutic value clinically. **(Habib, 2005)** One study in rats found the combination of serenoa repens, lycopene, and selenium is more effective for preventing hormone dependent prostatic growth than serenoa repens alone. **(Altavilla, 2011)** Other studies have discovered a correlation between BPH and selenium deficiency. **(López Fontana, 2010; Muecke, 2009; Thomas, 1999)** One study concluded that both vitamin E and selenium are potentially protective for BPH. **(Klein, 2003)**

Though testosterones are the focus in the pathogenesis of BPH, estradiol stimulates proliferation of prostate stromal cells suggesting that excessive aromatization of testosterone may be a factor. **(Zhang, 2008)** Prostate size has been found to be associated with estradiol/bioavailable testosterone ratio. **(Roberts, 2004)** Exposure of rats to the xenobiotic estrogenic endocrine disruptor, bisphenol A (BPA), has been shown to induce BPH, suggesting a possible generalized role for xenobiotic compounds in BPH pathogenesis. **(Wu, 2011)**

Dietary flavonoids may have beneficial effects that decrease BPH risk through their modulating effect on the phase I detoxifying enzymes (cytochrome P450 pathway) and activation of UDP-glucuronyl transferase, glutathione S-transferase, and quinone reductase of the phase II pathway. Dietary flavonoids, also, inhibit aromatase activity, thereby decreasing the pathogenic effects of xenobiotic compounds. **(Moon, 2006; Hiipakka, 2002)**

Flavonoids are widely distributed in plants with common dietary sources being citrus, berries, rutin from buckwheat, onion, legumes, tea, red wine, and cocoa. Supplemental sources include quercitin, the isoflavones (genistein, daidzein, glycitein), proanthocyanidins, and anthocyanidins.

Lycopene has support as prevention and therapy for BPH. This carotenoid antioxidant is more biologically active in singlet oxygen quenching than vitamin E (~100x) and glutathione (~125x). Though it is a red carotenoid found in a number of foods, the most significant dietary source is tomato, with the concentration and bioavailability being the most prominent in tomato paste. Research indicates that lycopene is therapeutic for BPH. **(Wertz, 2009; Schwarz, 2008)**

Fats seem to play a role in BPH pathogenesis. Vitamin D appears to be anti-proliferative and promote cellular maturation of prostate cells. **(Feldman, 1995)** The role of vitamin D may be through the down-regulation of COX-2 expression and PG2 production and by the arrest of NF-kappaB. **(Penna, 2009)** Research finds that the essential fatty acid profile in BPH is deficient primarily in the omega3 fatty acids and, secondarily, in the omega6 fatty acids. **(Yang, 1999)**

A literature review showed an inverse relationship between exercise levels in general and the incidence of BPH. **(Sea, 2009)**

One of the most impressive aspects in studies on natural approaches to prostate health has been the improvement in quality-of-life scores. **(Murray & Pizzorno, 1997)** Many men who suffer from an enlarged prostate also suffer from sleep deprivation. By improving the bothersome symptoms such as nocturia and the sleep deprivation it produces, a man's mental outlook is dramatically improved. Usually symptoms resulting from mild-to-moderate prostate enlargement respond more readily to these treatments than symptoms due to severe enlargement. No significant side-effects have been reported in the medical literature from these natural treatment methods.

Aching Pain, Lactic Acidosis, and Mitochondrial Dysfunction

Mitochondrial diseases are a group of potentially life threatening disorders, primarily effecting the nervous system, that represent severe impairment of mitochondrial function due to genetic or mutagenic mitochondrial DNA aberrations. Here we are discussing mitochondrial function relative to sub-optimal tricarboxylic/citric acid (TCA) cycle and electron transport system (ETS) functions.

It is common in some quarters of orthopedics and biomedicine to consider dysfunction of body systems to be an either/or phenomenon where a given system either functions adequately or has pathology. Fundamental to AK is the concept that many illnesses seen in clinical practice are due to dysfunction and dys-ease of systems that results in functional, rather than pathological illnesses. In this model, health is a spectrum and the early stages of many disease processes experience a stage of dysfunction before developing into tissue breakdown and pathology. An example here would be the slow and incremental

development of prostate cancer, which may go through a phase of benign prostatic hypertrophy for a long period before the inflamed and dysfunctional prostate tissues cross the threshold into cancerous pathology. In this case too there is a range of mitochondrial dysfunction from the fatigue and tissue soreness from suboptimal cellular energy production to life threatening pathology. Here we will cover functional issues concerning mitochondrial energy production.

Functional systemic lactic acidosis is a state of suboptimal function of the TCA cycle resulting in increased reliance upon anaerobic glycolysis and increased production of lactic acid. Additionally, reduced function of the TCA cycle will result in diminished CO_2 production and put stress on the systemic buffering role of the bicarbonate system. The combination of increased lactic acid production and bicarbonate deficiency disturbs the systemic pH.

Patients with suboptimal mitochondrial function will most commonly complain of fatigue and generalized aching and stiffness in their muscles and sometimes in their joints. An inability to concentrate is common, and patients will often say they feel "foggy headed." This condition is often misdiagnosed as fibromyositis or fibromyalgia and, sometimes, as chronic fatigue syndrome. The serum CO_2 levels will be either in the low range of normal (common) or just below normal range (less common). It has been proposed that though the norm for serum CO_2 is 23-32 mmoI/L, an optimal range is 26-31 mmoI/L, and below this range the patient has a relative systemic lactic acidosis. Low CO_2 can be due to a number of causes, including ketoacidosis, renal failure, and intoxication of organic acids or sulfates, but the most common in a functional medicine practice is lactic acidosis. **(Eidenier, 2007)**

If the lactic acidosis becomes more severe, symptoms may include anxiety, shortness of breath, arrhythmia, tachypnea, tachycardia, nausea, and generalized muscle weakness. **(See Acid-Alkaline section above)** The anxiety associated with mitochondrial dysfunction may be partly due to impaired GABA, since alpha-ketoglutaric acid, produced by TCA cycle function, is a necessary precursor for glutamic acid and GABA production.

Schmitt observes that when patients have functional lactic acidosis muscle weaknesses found with AK MMT will be negated temporarily by having the patient rebreathe their own air in-and-out of a paper bag for a few cycles. This test is suggested to be reliable indicator of low CO_2 reserve and suboptimal TCA cycle function and sometimes an indicator for the need of Vitamin B6. **(Schmitt, 1990)** This approach has been examined by Ozello. **(Ozello, 2006)**

When the rebreathing negates muscle inhibition as determined by AK MMT, one or more of the nutrients essential to the TCA cycle may be sufficient for restoration of adequate TCA cycle function. Thiamine (B1) deficiency is the most common need, but also riboflavin (B2), niacin (B3), pantothenic acid (B5), manganese, magnesium, biotin, iron, sulfur, or phosphorus.

Lipoic acid is also critical for the TCA cycle function and mercury is known to deplete sulfur-containing antioxidants such as L-acetyl-L-cysteine, L-glutathione, and alpha-lipoic acid. **(Houston, 2011)** If lipoic acid tests deficient according to AK MMT, differentially diagnose the patient for possible mercury toxicity. Less commonly, arsenic toxicity will be a factor in the lipoic acid depletion.

Coenzyme Q_{10} (ubiquinone) has been identified as a critical component of the electron transport chain, the next step in the respiratory cycle following the TCA cycle. **(Brandt & Trumpower, 1994; Rich & Wikstrom, 1986)** Its role in preserving mitochondrial integrity cannot be overemphasized.

Once a patient is supported with the nutrients indicated from AK MMT, the symptoms associated with lactic acidosis will diminish within a few days, though a loading dose for one to two weeks may be needed initially. If thiamin is indicated, based upon AK MMT, the phosphorylated form — thiamin pyrophosphate — may be required for optimal clinical response. According to Schmitt, if the patient has a good clinical response initially and then regresses, support of the electron transport system is indicated. **(Schmitt, 1990)** The nutrients essential for electron transport are CoQ_{10}, iron, copper, and phosphorus. CoQ_{10} deficiency as determined by AK MMT is the most common finding. Examine for a CoQ_{10} deficiency in patients taking statin drugs as they inhibit production of mevalonic acid, the precursor in the synthesis of CoQ_{10}. **(Deichmann, 2010)**

When TCA cycle dysfunction is present, anaerobic glycolysis will be up-regulated, putting stress on blood sugar regulating systems, since anaerobic glycolysis produces only two molecules of ATP per each molecule of glucose compared to 38 molecules of ATP per each molecule of glucose. This may result in chronically high cortisol levels in an effort of the body to up-regulate gluconeogenesis and place an excessive demand on glucagon production, gluconeogenesis, and chromium reserves.

The catabolic shift required by the up-regulation of gluconeogenesis is likely to be an underlying factor for low DHEA reserves, impaired protein and collagen metabolism, impaired immune-competency, osteoporosis, slow wound healing, and muscle wasting seen in this group of patients.

Urea-Guanidine Cycle and Deep Aching Pain

Excessive guanidine production has been proposed as a possible causative agent for pain. Guanidine, a metabolite of the urea-guanidine cycle, is a nitric oxide synthase (NOS) inhibitor. **(Ruetten, 1996)** Low tissue NO has been associated with vaso- and broncho-constriction, increased arterial wall inflammation, increased oxidative stress, up-regulation of renin release, impaired macrophage functions, impaired gastric motility, and diminished intestinal mucin secretion. Excessive guanidine is also known to cause muscle spasticity, **(Martensson, 1946)** and Duffy has associated high guanidine tissue levels with the deep, aching pain seen in patients with fibromyositis/fibromyalgia. **(Duffy, 2008)**

With impaired urea cycle function, there will be an adaptive up-regulation of the guanidine cycle to preserve production of the essential metabolite creatine with an associated increase in the production of guanidine. **(Natelson, 1979)** Guanidine inhibits the TCA cycle just discussed, lowering the production of two TCA metabolites, CO_2 and aspartate, which are cofactors for the urea cycle, further down-regulating the TCA cycle and up-regulating anaerobic glycolysis and lactic acid levels. **(Rao, 1992)**

Schmitt has developed a protocol for determining urea cycle functions through AK MMT. An ammonia sniff challenge is used to determine deficient, sufficient, or excessive ammonia tissue levels. With optimal balance of the urea cycle, sniffing ammonia will neither weaken nor strengthen an indicator muscle. **(Schmitt, 1990)**

When an ammonia sniff challenge strengthens a weak indictor muscle, there may be protein deficiency, but the most common reason is deficient pyridoxal-5′-phosphate (P5P), the activated form of vitamin B6. This may be caused by deficiency of the cofactors necessary for conversion of vitamin B6 to the coenzyme P5P. These cofactors are magnesium, zinc, riboflavin, and/or phosphorus. Since transamination reactions depend on P5P, a common finding is low or low-normal alanine transaminase (ALT) and/or aspartate transaminase (AST). **(Lacour, 1982)** A number of polymorphisms can inhibit both production of P5P and activity of P5P on dependent enzyme systems. **(Clayton, 2006)**

When the ammonia sniff challenge is positive, tissue ammonia burden is most likely too high due to impaired synthesis of urea or creatine from ammonia. If the ammonia sniff test is positive with AK MMT, test for the following nutrients: B6 (possibly, P5P), iron, magnesium, manganese, molybdenum, biotin, thiamin, riboflavin, niacin, pantothenic acid, lipoic acid, and a source of the enzyme arginase. Many of these nutrients are cofactors in the TCA cycle and indicate the interdependence between the TCA and urea cycles. In our opinion, Schmitt has developed an elegant and clinically effective method for testing and resolving many functional metabolic faults and his work is both recommended and beyond the scope of this discussion. **(Schmitt, 2005)**

Inactivity Aggravated Pain and Calcium Metabolism

High calcium-phosphorus ratio is associated with a particular pattern of deep aching pain and stiffness that is accentuated by inactivity and attenuated by movement. The relief from a few minutes of movement will often be complete and the soreness and stiffness will return when immobile for more than ~30 minutes.

Typically, the patient will have a serum calcium-phosphorus ratio greater than 2.4 (ideal value is approximately 2.4) and will find almost miraculous relief once the calcium-phosphorus ratio is improved. Stress to calcium-phosphorus metabolism will often be due to over-utilizing calcium supplementation or from a need for more dietary phosphorus. A trial of supplementation with liquid ortho-phosphoric acid will typically bring about a dramatic change in one to two days and confirm the diagnostic suspicion. Sources of phosphorus are milk, egg yolk, meat, grains, nuts, legumes, and lecithin.

Burning pain, NSAIDs, and the prostaglandin system

Chronic burning pain is usually due to imbalanced prostaglandin (PG) metabolism with relative up-regulation of the pro-inflammatory prostaglandin2 (PG2) family. When non-steroidal anti-inflammatory drugs (NSAIDs) provide temporary relief of burning pain, underlying prostaglandin imbalance is strongly indicated. The underlying PG imbalance can be due to excess prostaglandin2 (PG2) levels; deficient anti-inflammatory prostaglandin1 (PG1) levels; or prostaglandin3 (PG3) levels, or both. The patient usually needs less animal fats in the diet (animal fats are the most common source of arachidonic acid, the precursor of PG2) and more vegetable, nuts, seeds, grains, or fish oils.

Clinical disorders due to insufficient PG3 levels appear to be common and account for a wide range of illnesses. **(Simopoulos, 2006; Rudin, 1982)** Prostaglandins may sensitize nerve endings to the pain-producing effects of other compounds, such as bradykinins. Omega3 fatty acids are precursors for PG3 and dietary sources are nuts, seeds, beans, grains, flax, fish, meat, eggs, and milk. Animal fats from grass-fed sources are naturally higher in omega3 fatty acids than grain fed sources. **(Daley, 2010)**

Occasionally, deficiency of PG1 is primary. This is usually seen in patients with disturbances of female reproductive system. Most vegetable oils are naturally high in omega6 fatty acids with black currant, borage, and evening primrose oils being particularly concentrated sources and are unaffected by delta-6 desaturase inhibition. **(Horrobin, 1983)**

Trans-fats (hydrogenated oils) inhibit delta-6 desaturase (D6D) and limit production of the anti-inflammatory PG1 and PG3 families. This process underlies the prevalence of high pro-inflammatory PG2 levels relative to PG1-PG3 levels and the prevalence of chronic inflammatory diseases and pain in cultures consuming refined foods, especially when animal fats are over-indulged. D6D activity is attenuated by hydrogenated fats, alcohol, arachidonic acid, glucose, fructose, smoking, heavy metal burdens, catecholamines, glucocorticoids, and thyroxine and is accentuated by B6, magnesium, zinc, protein, insulin, and ATP.

Eliminate Trans fats from the diet

When trans fat inhibition of D6D is present, patients can be helped with PG1-PG3 precursors that are D6D dependent, using evening primrose, borage, and black currant seed oils for the PG1 family and fish oils for the PG3 family.

In December 2006, New York became the first U.S. city to ban artificial trans fats at restaurants — from the corner pizzeria to high-end bakeries. **(New York Times, 2006)** The city's prohibition on trans fats was a victory for Mayor Michael Bloomberg, an outspoken health advocate, and his activist health commissioner, Dr. Thomas R. Frieden.

When PG2 levels are too high in relation to the PG1-PG3 families, inflammation may be up-regulated and the patient may experience temporary relief when using NSAIDs. **(See discussion in Dysmenorrhea section above)** There will also be facilitation of inhibited muscles upon insalivation (AK sensorimotor challenge) of a powdered mixture of aspirin, acetaminophen, and ibuprofen. Conversely a facilitated indicator muscle may weaken when a patient is challenged with a concentrated source of arachidonic acid (lard is very effective for this purpose). Essential fatty acids and cofactors can be screened for negating the positive arachidonic acid challenge and the patient can be supported appropriately.

When PG imbalances are present, having the patient smell chlorine will cause a strong indicator muscle to weaken upon AK MMT, and testing with insalivation of an indicated essential fatty acid or cofactor nutrient at the same time will negate the positive chlorine challenge. This test was originally described by Schmitt. **(Schmitt, 2005)**

Itching, edematous pain and bradykinin

This type of pain is often associated with edema (bradykinin is a potent vasodilator), itching, and sensitivity to pressure. **(Ständer, 2006; Graven-Nielsen, 2001)** Interestingly, bradykinin excess caused by ACE inhibitors may be associated with a persistent slight, dry cough. **(Dicpinigaitis, 2006)** Kinins appear to be an important mediator of the pain, swelling, and cellular damage associated with inflammatory joint diseases. **(Bhoola, 1992)**

Bradykinin release is triggered by injury, but abnormal modulation may play a role in chronic pain. **(Raja, 1988)** Natural bradykinin inhibitors are bromelain, aloe, and polyphenols (bioflavonoids), as found in citrus, red wine, coffee, tea, and chocolate. **(Bautista-Pérez, 2004; Bouskela, 1997; Lotz-Winter, 1990)**

Systemic inflammatory pain

Oxidation

Reactive oxygen species (ROS) are essential metabolites of oxidative phosphorylation in the mitochondria, and their role has been documented in producing DNA damage, lipid peroxidation, oxidation of amino acids, inactivation of enzymes, degenerative diseases and aging. Antioxidant enzymes, primarily superoxide dismutase, glutathione peroxidase, methionine reductase, and catalase, normally control ROS levels and mitigate their damage.

Poorly modulated ROS metabolism results in up-regulation of cellular and systemic inflammation. High or high normal C-reactive protein (CRP), fibrinogen, and erythrocyte sedimentation rate (ESR) can be used as indirect monitors of ROS-triggered inflammation when informed by the overall clinical picture. Urinary malondialdehyde (MDA) may be a clinically useful monitor of ROS stress.

AK MMT can be used to challenge for the presence of ROS stress by having the patient smell chlorine. Chlorine is a source of hypochlorite free radicals and will cause a strong indicator muscle to weaken when ROS stress is present. Any of the antioxidant promoting nutrients can be tested for negation of this positive chlorine olfactory sensorimotor challenge; support accordingly.

Methylation

Hyperhomocysteinemia due to impaired methylation metabolism has been associated with atherosclerotic cardiovascular diseases, stroke, peripheral arterial occlusive disease, venous thrombosis, autoimmune and neurological disorders, including autism. **(Houston, 2012; James, 2004; Richardson, 2003; Herrmann, 2001)**

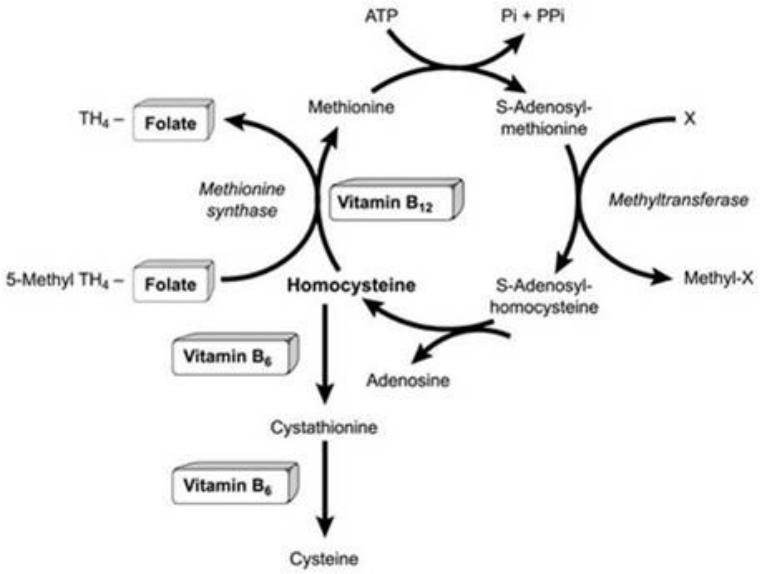
AK MMT has shown an association with high homocysteine levels, a common cause of systemic inflammation, and bilateral psoas muscle weakness, a common cause of low back and pelvic pain. **(Rogowsky, 2005)** High homocysteine levels are usually moderated through sufficient methylcobalamine (B-12), 5-methyltetrahydrofolate (MTHF), folic acid, pyridoxyl-5-phosphate (P5P), serine, betaine, and/or arginine. **(Gaby, 2006; Figure below)**

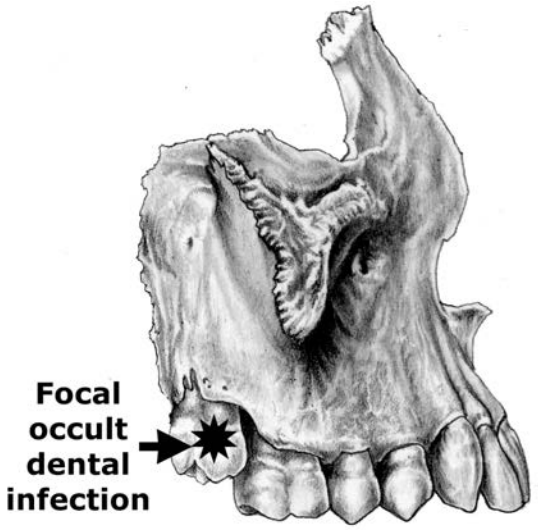
When smelling chlorine causes an indicator muscle to weaken with AK MMT and the positive challenge is negated by taurine, methylation metabolism is impaired. Test methylation cofactors (methionoine, magnesium, methylcobalamine (B-12), folic acid, 5-methyltetrahydrofolate (MTHF), a methyl donor (betaine, choline), and molybdenum, and support as indicated using the nutrient(s) that negated the positive chlorine smell challenge.

Leukotrienes

When the production of PG2 is inhibited by NSAIDs, the production of leukotrienes from arachidonic acid is up-regulated. Leukotrienes are powerful pro-inflammatory compounds that have been associated with the pathophysiology of inflammatory and allergic conditions affecting skin, joints, and respiratory and gastrointestinal systems. **(Haeggström, 2002)** Leukotriene up-regulation from NSAID use accounts for the side effects from this family of drugs. **(Stevenson, 2003; Rainsford, 1993)**

When leukotrienes are improperly modulated in a patient, there will be inhibition of previously strong muscles with insalivation of a powdered mixture of aspirin, acetaminophen, and ibuprofen. Check the patient using AK





Homocysteine Pathway

MMT for essential fatty acid metabolism precursors and cofactors to improve the ratio between pro-inflammatory PG2 and the anti-inflammatory PG1-PG3s, and guide the patient on how to decrease their arachadonic acid intake through less animal fats in their diet and to avoid NSAIDs. Also consider testing for leukotriene-inhibiting nutrients. These are vitamin E (>200 IU/day), eicosapentaenoic acid (EPA), quercitin, glutathione, selenium, and aloe vera.

Glycation

Impaired glucose metabolism (either diabetes or insulin resistance) results in increased glycation of proteins or fats with sugars to produce advanced glycation endproducts (AGEs) which contribute to ROS stress and inflammation. This is a major mediator of the high incidence of neuropathy, cataracts, and cardiovascular disease seen in diabetics. **(Veves et al., 2002; Kanauchi, 2001)** One researcher has proposed that glycation is pathogenic for a wide array of chronic, degenerative illnesses and may be associated with senescence itself. **(Cárdenas-León, 2009)**

Glucose is a key component of this process, but the efficiency of fructose glycation is approximately ten times greater than that of glucose, leading some researchers to conclude that fructose-dependent diets lead to higher AGEs production and accentuated development of AGEs-mediated diseases. **(Gul, 2009; Gul, 2009a; Mikulíková, 2008; Tokita, 2005; Schalkwijk, 2004)**

Fundamental to control of glycation is improving blood sugar metabolism and optimizing insulin sensitivity through diet and exercise. The dietary issues do not need to be belabored here, but research does indicate that short, intense, interval exercise is more effective for improving insulin sensitivity than longer, non-interval exercise sessions. **(Metcalfe, 2011; Babraj, 2009)**

As for clinical nutrition, lipoic acid has been shown to decrease glycation, and taurine has been shown to alleviate glycation-mediated changes to collagen in fructose-fed rats. **(Thirunavukkarasu, 2005; Nandhini, 2005)** Rutin, a dietary flavonol, has been shown to inhibit glycation and would, at least, suggest the same role for other favonols. **(Muthenna, 2011)** A wide range of polyphenols widely found in fruits and vegetables show inhibition of glycation. **(Saraswat, 2009; Biesalski, 2007)** Citric acid from citrus fruits and ginger has also shown promise. **(Saraswat 2010; Nagai, 2010)**

Focal occult infection

The theory that focal occult infections can mediate systemic inflammatory and degenerative effects was first proposed in the 1920s. **(Lee, 1925; 1923)** Though discredited for many decades, the focal infection theory of systemic disease is becoming once more better established. **(Goymerac, 2004)** The most recognizable example here is periodontal disease being a risk factor for systemic inflammation and related degenerative diseases, such as cardiovascular disease. **(Glickman, 2009)**

One study of periodontal disease indicated a causative role for systemic inflammatory markers through the lowering of CRP, interleukin-6 (IL-6), and LDL cholesterol levels from baseline after two months of periodontal therapy. **(Somma, 2010)** It is possible to trace bacteria recovered from peripheral blood to occult focal infection in tooth apices after a root canal, and it has been suggested that the resultant bacteraemia and circulating endotoxins may have systemic effects. **(Murray, 2000)**

The Medical Journal of Applied Kinesiology **(2012)** has been publishing reports from dentists and other clinicians in the German-speaking world on this subject and has produced an impressive compendium of the AK diagnostic findings in cases of focal occult infections and their treatment.

Sinusitis is another common focal occult infection, with the presenting complaint often being fatigue, in many cases accompanied by the complaint of "foggy-headedness" or some similar symptom. **(Chester, 1996)** A study from the Mayo Clinic of 210 subjects with chronic sinusitis concluded that 96% were also positive for fungal infection. **(Ponikau, 1999)**

Protozoan infection is known to be a causitive agent for irritable bowel syndrome, and *Blastocystis hominis* infection has been reported to produce reactive arthritis, indicating systemic inflammatory and immunoactive effects. **(Stark, 2007; Lakhanpal, 1991)** Emulsified oregano oil is an effective therapy for *Blastocystis hominis* and other protozoan infections. **(Force et al., 2000)** Intestinal bacteria and viruses and food allergens can dysmodulate immune and inflammatory responses, primarily through promotion of IFN-gamma, TNF-alpha and other pro-inflammatory cytokines. **(Peña, 1998)**

Joint pain

Osteoarthritis

Risk factors for osteoarthritis (OA) include arthrogenic muscle inhibition, muscle weakness, joint injury, age, obesity, genetics, and aberrant biomechanics. **(Hurley, 2002)** Patients will usually complain of stiffness, limited range of motion, and feel worse after physical activity. Interestingly, imaging studies for joint pathology often do not correspond to symptoms indicating the critical role of biomechanics. This concurs with the conclusion of Rosomoff, who stated, "It is our opinion that in many cases attributed to disc pathology, arthritis or spinal canal stenosis the symptoms may, in fact, be caused by soft tissue dysfunction. What appears as pathology on an X-ray may be the end result of abnormal biomechanics resulting from this dysfunction." **(Rosomoff, 1989)**

Applied kinesiology plays an important role in the amelioration of osteoarthritis by optimizing muscle strength, coordination and timing to optimize postural and movement patterns, including the gait-cycle. By optimizing muscular and articular biomechanics, joint injury with use is minimized and the mechanical pathogenesis of osteoarthritis minimized.

The degree of synovitis found with osteoarthritis correlates with synovial concentration of both leukotrienes and kinins, indicating a critical role for prostaglandin and bradykinin metabolic faults in OA. **(Nishimura, 2002; Bhoola, 1992)**

Glycation promotion, ROS stress, and low-level chronic inflammation have also been implicated in the pathogenesis of OA. **(Shane-Anderson, 2010; Ziskoven, 2010; Regan, 2008; Hadjigogos, 2003; Verzijl, 2002)**

Joint immobilization promotes ROS through diminished superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px) in the joint capsule. **(Erdem, 2009)** Yoga and Tai Chi/Qi Gong may be useful for management of OA through decreasing arthritic symptoms and improving joint function and balance sense. **(Ebnezar, 2012; Lee, 2009; Kolasinski, 2005; Song, 2003)**

Rheumatoid Arthritis

Patients with RA show increased ROS stress. **(Seven, 2008; Cai, 2005)** Free-radical oxidation products in serum were significantly elevated in patients with RA compared to normal controls. **(Lunec, 1981)** SOD values were significantly lower in RA patients compared to controls. **(Disilvestro, 1992)** Dysmodulation of DNA methylation has been proposed to have epigenetic effects that may be a factor in the pathogenesis of autoimmune diseases, including RA. **(Strickland, 2008)**

Zinc, copper, and selenium metabolism may be vectors in the pathogenesis of RA as well. In RA patients the erythrocyte sedimentation rate (ESR), acute-phase proteins, interleukin-1 beta (IL1 beta), and tumor necrosis factor alpha (TNF alpha) imbalances correlated negatively with serum zinc and positively with serum copper. **(Zoli, 1998)**

Mean serum copper concentration in RA subjects has been positively correlated with erythrocyte sedimentation rate (ESR) and Ritchie articular index. **(Strecker, 2005)** Low zinc and low Zn/Cu ratio are found in plasma of RA patients. **(Ala, 2009)** Patients with RA may also tend toward dietary patterns that are deficient in pyridoxine, zinc and magnesium. **(Kremer, 1996)**

Bradykinin up-regulation may be a factor in RA as synovial fluids show excessive release of bradykinin. **(Sharma, 1994)**

As with any autoimmune disorder, the triggering mechanism may be an occult locus of infection as previously discussed. For this reason, closely examine the sinuses, teeth and gums, intestinal and urinary tract. Intestinal inflammation has been associated with arthritis. **(Mielants, 1987)** Occult focal apical infections of teeth after root canal have been implicated in the pathogenesis of RA, **(Murray, 2000)** and periodontal treatment was reported to result in RA remission. **(Iida, 1985)**

Total fasting of 7-10 days induces marked reduction of RA pain, inflammation, and edema within a few days and the remission subsides over time after discontinuing the fasting. **(Palmblad, 1991; Sundqvist, 1982)** Intestinal permeability improves with fasting, suggesting a role for food allergy and/or digestive dysfunction in RA. **(Sköldstam, 1991; Panush, 1991)** Direct involvement of food allergy in the pathogenesis of RA was challenged in a study suggesting that the contribution of food allergy to RA pathogenesis may be mediated through impaired intestinal permeability rather than through direct influence on humoral immune response. **(Kjeldsen-Kragh, 1995)**

Fasting followed by a lactovegetarian diet appears to be good management for RA and one study showed significant decreases in platelets, leukocytes, total IgG, IgM rheumatoid factor, and complement components C3 and C4 after one month. **(Müller, 2001; Kjeldsen-Kragh, 1995)** The benefit, at least in part, is likely due to the decreased arachadonic acid intake associated with vegetarian diets and the shift to a lower PG2/PG1-PG3 ratio compounded by an increased consumption of dietary omega3 fatty acids. **(Simopoulos, 2002)**

Electric blankets may be a causative or aggravating factor in arthritis and the adverse effect of electromagnetic radiation can be observed with AK MMT. **(Maykel, 2007)**

Circulatory system

Differential diagnosis is essential for circulatory problems of the lower extremity. Bilateral edema of the lower extremities should always lead to minimally considering

heart, kidney, or liver pathologies or dysfunction, while unilateral edema should lead to consideration of deep vein thrombosis, thrombophlebitis, pelvic tumor, and cellulitis. Differential diagnosis of these possible pathologies is outside the scope of this chapter.

Varicose veins

Hemorrhoids and varicose veins are one of the most common afflictions of the Western world, affecting people of all ages and both genders. Worse yet, the problem typically worsens over time. Known causes include constipation, pregnancy and poor toilet habits such as straining while reading a favorite book. Symptoms include itching, bleeding and pain. Genetics play a large role in the predisposition to varicose veins. Don't overlook, however, the role that collagen formation plays in optimal venous structural strength and elasticity and the influence portal congestion on venous pressure in the pelvis and lower extremities have in predisposing to varicose veins and hemorrhoids. Also, note the influence of catecholamines and thiamine for increasing vascular tone.

Bioflavonoids have been shown to decrease capillary fragility and have positive effects on capillary permeability and blood flow. They show promise for the treatment of bruising, varicose veins, edema, and hemorrhoids. **(Martin, 1955)** Additionally, bioflavonoids show antioxidant, antihistamine, and anti-inflammatory effects. Numerous studies have shown bioflavonoids improve outcomes in treatment of hemorrhoids. **(Di Pierro, 2011; Misra, 2000; Ho, 2000)**

The authors have also found that the dietary supplement stone root, also called collinsonia root (Standard Process®), can be helpful. This product has been successfully used for decades for patients with hemorrhoids and/or varicose veins. Two capsules with a glass of water morning and evening on an empty stomach can help even the most stubborn cases. Patients usually take these for a month or two, and then discontinue when symptoms are gone. Patients keep some of this on hand for the first sign of any recurrence, which may indicate the need to assess the diet once more.

Edema

Generalized edema must be considered on the basis of the cellular metabolism of the cell wall which, in effect, is the osmotic transfer control that governs the movement and permeability of all body fluids. If not due to heart failure, consider liver, kidney, and adrenal dysfunction or pathology in the differential diagnosis. In women experiencing edema that occurs during the middle and late stages of the menstrual cycle, excessive estrogen may be suspected. This may be due to overproduction or inadequate deconjugation by the phase II detoxification pathway of the liver.

Edema due to nutrient deficiencies

It is worth noting that three classic nutrient deficiencies have been associated with edema in the lower extremities: kwashiorkor (protein), beriberi (thiamin), and pellegra (niacin). Though the symptoms by standard criteria are limited to severe deficiencies, it is important to consider clinically that nutrient deficiencies and their related symptoms exist in a spectrum of severity, and that sub-clinical deficiencies exist, particularly in the elderly. **(Gaby, 2006; Shils et al., 1999)**

Biochemical individuality and subclinical nutrient deficiency

Cheraskin, Ringsdorf, and Williams have each proposed models of biochemical individuality wherein each individual has an utterly unique biochemistry based on their genetic constitution, epigenetic history, and environment — often resulting in inborn errors of metabolism. These inborn errors can result in resistance to the influence of nutrients on enzyme systems and predispose patients to a need for higher levels of nutritional supplementation than the average population. **(Bucher, 2011; Cheraskin, 1977; Cheraskin, 1976; Williams, 1956)** AK adds to the physician's nutritional knowledge an ability to determine, to a certain extent, the effects of various nutritional products on the biochemical individuality of the patient being evaluated.

Zinc, magnesium, and vitamin D are good examples of this as they are commonly deficient upon laboratory exam and can underlie a wide array of symptoms in patients that would appear to be sufficiently nourished based upon an analysis of their diet. **(Hambidge, 2007; Holick, 2006; Whang, 1987)**

Sub-clinical B12 deficiency is now recognized as a valid clinical entity with subtle manifestations of the classic deficiency pattern and being responsive to B12 supplementation, especially in the elderly. **(Herrmann, 2003)** Based upon this principle, consider sub-clinical protein, thiamin, or niacin deficiency in patients with otherwise idiopathic edema of the lower extremity. **(Padhila, 2011; Igata, 2010; Singleton, 2001)**

Morabia has argued that laboratory testing is inadequate to diagnose these syndromes and that clinicians must still rely on observation of patients' patterns of signs and symptoms for adequate differential diagnosis. **(Morabia, 2011)**

McCarty has proposed a model for subtle nutrient deficiency leading to aberrant physiology and functional illness when he states "many nutritional agents involved in bioenergetics (regulating mitochondrial and antioxidant functions) are often functionally sub-saturated." **(McCarty, 1981)**

Rudin **(1982)** has proposed a model for pellagraform and beriberiform diseases that present as syndromes mimicking true pellagra and beriberi, but lack the developed pathologies associated with these classic nutrient deficiency diseases. Rudin calls these disorders substrate pellagra and substrate beriberi. Rudin proposed that the interdependency of B vitamins and their cofactor substrate essential fatty acids (primarily omega3 fatty acids) for production of structural and regulatory proteins and lipids could be compromised by malnutritional synergy — the combined subclinical deficiencies of proteins, fats, vitamins, and minerals.

Nutrient Deficiencies Associated With Edema

Kwashiorkor	BeriBeri	Pellagra
Pedal edema, ascites, enlarged and fatty liver, thinning hair, loss of teeth, skin depigmentation, dermatitis, irritability, anorexia.	Lower extremity edema, weight loss, emotional disturbances, impaired sensory perception (Wernicke's encephalopathy), parasthesias, arrhythmia, mental confusion/speech difficulties, vasodilation, awakening at night, short of breath, tachycardia, shortness of breath with activity.	Dermatitis (red skin lesions desquamation, erythema, scaling, keratosis of sun-exposed areas), photophobia, aggression, anxiety, mental confusion, insomnia, dementia, sensitivity to odors, neuralgia, neuritis, ataxia, weakness, alopecia, edema, glossitis (smooth, beefy red), diarrhea, dilated cardiomyopathy.
Protein deficiency	**Thiamin deficiency**	**Niacin deficiency**
Adequate hydrochloric acid and pancreatic enzymes are required for absorption; cooking all proteins may result in deficiency of heat-labile amino acids, small intestine absorption must be sufficient, and the urea cycle must be functional to have adequate protein metabolism.	Provide B1 (thiamine); a phosphorylated form may be needed as many patients ineffectively phosphorylate synthetic forms (i.e. thiamin hydrochloride). Refining removes naturally occurring thiamin from foods and sulfites degrade it. Refined carbohydrates increase tissue demand for thiamin.	Provide B3 (niacin); a phosphorylated form may be needed. Avoid corn, corn-derived sugars, and excessive sun exposure. Niacin is synthesized in the liver from tryptophan; tryptophan is heat-labile and a lack of uncooked protein sources may predispose to tryptophan deficiency.

Table 1

Table 1 may help the clinician understand the etiology for some presentations of edema in the lower extremities. It is important to note here that we not claiming that true kwashiorkor, beriberi, and pellagra are common diseases; they are, indeed, very rare, especially in industrialized societies. What we are attempting to make the clear is that functional (rather than pathological) nutrient deficiencies are more prevalent than supposed.

According to Rudin,

> "...since substrate essential fatty acids are processed by many B vitamin catalysts, an EFA deficiency will mimic a panhypovitaminosis B, i.e., a mixture of substrate beriberi and substrate pellagra resembling vitamin beriberi and pellagra but exhibiting as even more diverse endemic disease. This would constitute a second stage of the modern malnutrition and explain why some workers now hold the dominant diseases of modernized societies to be new, nutritionally based, pellagraform yet lipid-related and to range, once again, from heart disease to psychosis."

Impaired Microcirculation Sympathetic Vasoconstriction

In patients with chronic cold hands and feet, especially when concurrent with clamminess, look to sympathetic dominance of the autonomic nervous system since sympathetic tone is peripherally vasoconstrictive and diaphoretic. Other considerations for cold extremities are anemia and conditions that lower metabolic rate and body temperature, in general. These include hypothyroidism, hypoglycemia, mitochondrial dysfunction (citric acid cycle and electron transport), and essential fatty acid and/or protein deficiency.

Raynaud's Syndrome

In patients with Raynaud's syndrome secondary to sclerodermal disease, look to high blood viscosity due to increased fibrinogen levels. **(Sergio, 1983)**

Other causes of Raynaud's are increased sympathetic tone, hyperviscosity, and hyperactivation of platelets and erythrocytes. Increased prostaglandins PGE1 improved peripheral blood flow in Raynauds patients. **(Belluci, 1988)** Goodheart noted that patients with essential fatty deficiency commonly complain of being cold despite a normal body temperature. **(Goodheart, 1985)**

Blood Hyperviscosity Syndromes

Increased viscosity increases peripheral resistance of the microcirculation. Blood viscosity is known to increase with increased IgG, IgA, IgM, fibrinogen, and triclyceride

levels. These can be found in Waldenström's disease and hypertriglyceridemias. **(Crepaldi, 1983)** There is a strong correlation between metabolic syndrome and increased viscosity and the correlative physiological changes likely to be causative factors for arterial hypertension. **(Zhang, 2006; Hrnciarová, 1995)**

Increased blood viscosity decreases oxygen perfusion, results in decreased total oxygen delivery, and increases risk of veno-occlusive syndromes. **(DeFilippis, 2007; Maeda, 2006)** Interestingly, Qi Gong exercise has shown positive effects on blood viscosity. **(Lee, 2009)** The cardiovascular protective effects of omega3 fatty acids may be in part due to lowering blood viscosity. **(Reiner, 2007)**

Rouleaux Formation

Rouleaux formations are aggregates (clumping) of RBCs. Conditions associated with rouleaux formation are diabetes mellitus, infection, autoimmune and inflammatory disorders, anemia, and cancer. Rouleaux formation results in higher ESR results. Rouleaux formation increases with rising pH and decreases relative to pH lowering influence of CO_2. **(Cicha, 2003)** High plasma triglycerides and platelets increase rouleaux formation. **(Cicha, 2001; Baumler, 1987)** RBC cell membrane charge appears to modulate rouleaux formation. **(Antonova, 2006; De Lorenzo, 2004)**

Blood Viscosity And Rouleaux Interactions

Hyperviscosity syndromes are associated with rouleaux formation of the RBCs. **(Ballas, 1975)**

High blood viscosity, a determinant of total arterial resistance (TAR), and rouleaux formation has been proposed as a causation of arterial hypertension due to increases peripheral resistance of the microcirculation. **(Cicco, 1999; Zannad, 1985)**

Applied Kinesiology's Future in Stress-Related Illness

Most illnesses in industrialized societies are due to functional rather than pathological processes; most pathological illnesses are preceded by a chronic period of functional illness. Health is not an accident; it is the outcome of the interaction of an individual's genetic constitution and environment. Many people "get by" throughout their lives without optimal organic or biomechanical function and yet remain asymptomatic. This may depend on the goddess Fortune as well as the world-view and impulses of the person in addition to their inherited characteristics, nutritional status, psychosocial factors, life history and more – in other words, the entire context within which the applied kinesiology triad of health is experienced and embraced. If one of the objectives of work in this field is to prevent illness and ameliorate the burdens of the living patient and to help them realize their full potential, then what has been discussed in this chapter will become a part of the health care approach physicians and knowledgeable patients around the world embrace.

The Canadian endocrinologist, Hans Selye, (1976) developed the model for functional illness when he published his extensive research concerning the processes underlying this type of disease. He clearly shows that disease results when the homeostasis of a living organism is overcome due the cumulative effect of stressors to which it must adapt, be they physical, chemical, thermal, or mental.

From the beginnings of applied kinesiology, practitioners have observed an association between muscle-joint function and visceral-autonomic dysfunction. It is exciting to see accumulating research and developing models from a wide range of academicians and clinicians converging toward concurrence with the field of applied kinesiology. This development will, ideally, lead to more coordination with physicians from other fields and backgrounds to work synergistically with clinicians utilizing applied kinesiology methods in the treatment of patients with functional illnesses.

Evidence-based medicine, basic science and clinical outcomes data now exists to support the assessment and treatment (frequently co-treatment with other specialist physicians) for patients with disorders of the nervous, autonomic, neurohumoral, immune, respiratory, circulatory, and lymphatic systems using applied kinesiology methods. The objective of this work is to prevent illness, ameliorate suffering, and to help patients reach their full potential. Hopefully, the methods here will be embraced and used toward those ends.

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