The Common Compensatory Pattern: Its Origin and Relationship to the Postural Model

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Introduction

J. Gordon Zink, DO[1] was the originator of the term Common Compensatory Pattern (CCP). He used the term to describe commonly found patterns of dysfunction in the body (neuromyofascial-skeletal unit[2]) as a whole. Several other physicians[3-6] before and since, have also described recurring patterns of dysfunction found in their patient populations. Dr. Zink, however, is considered to be "… the first to provide a written, understandable, and clinically useful explanation for treatment, with a method of diagnosing and manipulative methods of treating the fascial patterns of the body."[7] Zink himself considered these concepts to be the basis of a respiratory and circulatory care model.[2]

As osteopathic clinicians we frequently find recurrent patterns of fascial bias, postural asymmetry, somatic dysfunction, and functional disturbances. We frequently see a clinically short right leg, a cephalad pubes dysfunction on the left, a posterior ilium on the left and an anterior ilium on the right. Patients regularly display a left-on-left sacral torsion with L-5, side bent left and rotated right as well. These are just a few of many commonly found somatic dysfunctions; the list is long. Radiographically, with our patients' postural studies, we can find recurring patterns of postural asymmetry that includes the anatomic short right leg and a sacral base declination to the right with compensatory rotoscoliosis. Beyond these findings we have recurrent patterns of functional disturbance such as muscle imbalance and visceral dysfunction, coupled with common systemic complaints.

Why do we see these same patterns over and over again? Is there a linkage between all of these commonly found clinical phenomena? Further, what is the clinical significance of these patterns? There appears to be an inherent fascial bias found in most people. There also appears to be a causal linkage between fascial bias and subsequent growth of the individual. Could these governing factors explain recurrent patterns of postural asymmetry that we find in the postural model? The probable key to these questions and their answers reside in the fascia.

The Fascia

" The fascia is the place to look for the cause of disease and the place to consult and begin the action of remedies in all diseases" — A.T. Still.

The fascia is found in sheets or bands of fibroelastic connective tissue throughout the body. The term is Latin for 'band' or 'fillet'. Every bone, muscle, nerve and organ develops within and is covered with some form of fascia. "If all other organs and tissues were removed from the body, with the fascia kept intact, one would still have a replica of the human body".[8]

Fascia is classified as deep, subserous, and superficial.[9] The deep layer serves to compartmentalize organs and muscles and nerves. Examples of these deep and thick fascias include the fibrous pericardium, parietal pleura, perineurium, and perimysium. The subserous fascias are fibroelastic connective tissues that cover and protect organs. Examples of these are the pleura, pericardium, peritoneum, and other organ capsules. The superficial fascia lies beneath and is continuous with the reticular dermis. There are numerous small fibrils that act to anchor the superficial to the deeper fascias of the body.

From the study of anatomy we know that the majority of fascia is arranged longitudinally. Consequently, we would expect that forces directed through palpation parallel to fasciae would allow an examiner to appreciate a greater sense of freedom in this direction than in the side to side direction. But clinically we can find that the fasciae move with greatest ease obliquely in a direction of side bending and rotation[10], thus displaying a combination of longitudinal and lateral movements.

Areas of muscular imbalance or somatic dysfunction can impose functional restrictions that will inhibit fascial motion. Frequently, the regions

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of most restriction can be found in what is known as transitional zones (Table 1).

Anatomically, these areas are also known as junctions, where the function of the spinal column changes. Zink[11] considered these the anatomical weak points. Additionally, each of these zones is associated with an actual or functional transverse diaphragm. There is extensive mobility at the OA or the craniocervical junction. At this junction the heavy head balances on the supple cervical spine. This is the site of the tonic neck reflexes, which influences postural muscular tone throughout the trunk.[13] If function is disturbed here, it frequently creates hypertonus of the postural muscles, disturbances of equilibrium and locomotor deficits. Rotational movement is most affected at this junction because only the atlantoaxial joint is ideally suited for rotation. There is a direct connection between the dura at the rectus capitis posterior minor at this junction, and cranial nerves IX, X, and XI also traverse this junction.

TABLE 1. TRANSITIONAL ZONES

ZONES	JUNCTIONS	TRANSVERSE DIAPHRAGMS
Occipital-Atlantal (OA)	Craniocervical Junction	Tentorium Cerebelli
Cervico-Thoracic (CT)	Cervicothoracic Junction	Thoracic Inlets/Outlets
Thoraco-Lumbar (TL)	Thoracolumbar Junction	Respiratory Diaphragm
Lumbo-Sacral (LS)	Lumbosacral Junction	Pelvic Diaphragm

The cervicothoracic junction is the region where the most mobile part of the spinal column is joined to the relatively rigid thoracic spine. It is also where the powerful muscles of the upper extremities and shoulder girdle insert. It is associated with the thoracic outlets/inlets through which traverse the lymphatic ducts, the right and left brachial plexus, and the phrenic and vagus nerves.

At the thoracolumbar junction spinal function changes abruptly as is seen in the differences in the upper (thoracic) and lower (lumbar) apophyseal joints of T-12. Somatic dysfunction in this area can be associated with hypertonus of the iliopsoas, quadratus lumborum, thoracolumbar erector spinae and inhibition of the rectus abdominus muscles. The abdominal diaphragm, which is physiologically the most important diaphragm, is found in this transitional zone. Through it passes the esophagus, the thoracic duct, the aorta, vena cava, and the azygous veins as well as the vagus and phrenic nerves. Contraction and relaxation of this diaphragm provides the impetus for breathing and it also produces alternating intrathoracic and intra-abdominal pressure gradients which provide the pumping mechanism for the venous and lymphatic circulation.

The lumbosacral junction forms the base of the spinal column and is therefore a major determinant of body statics. Movement from the legs is transmitted through this junction to the superincumbent spine. By muscular and fascial continuity the pelvic diaphragm is associated with this junction. It supports the pelvic viscera and invests the sacral plexus. It transmits lymphatics, splanchnic and pudendal nerves, the anal canal, the urethra, and the vagina. Its normal function is to remain relaxed and work in synchrony with the abdominal diaphragm and thus allow efficient return of lymph back into the venous circulation.

Restrictions in these transitional zones can cause major alterations in the function of surrounding structures, and thus directly or indirectly influence the health of the body. Zink studied people who considered themselves healthy and recorded "normal" fascial motions in each of these four zones.[12] He also studied the fascial patterns of hospitalized patients and outpatients who were considered to have low levels of wellness. With this information he identified three classifications of fascial patterning and labeled these *(1) ideal, (2) compensated, and (3) uncompensated*. He then associated these patterns with perceived patient wellness.

The ideal pattern is demonstrated by equal fascial glide in the side to side and longitudinal directions. Thus, there would be no apparent preference for fascial rotation or sidebending to either the right or the left, in any transitional zone. This ideal pattern is seldom if ever seen in the clinical setting. Alternating patterns of fascial ease and restriction are common. Usually a rotational bias in one transition zone is accompanied by an opposite fascial rotation in the next zone throughout the body. This alternating pattern, found in healthy subjects, was considered compensated (Figure 1). Zink reasoned that counterbalanced rotations were more adaptive and that was why these individuals responded more favorably to stress or illness. Those people with uncompensated fascial patterns, where the rotational pattern did not alternate, were thought to be less healthy.[12] They were more likely to have suffered trauma and demonstrated slower recovery from illness.

During these studies, Zink found that approximately 80% of healthy people had body patterns of L/R/L/R, while the other 20% displayed the opposite R/L/R/L pattern. He named this first pattern the Common Com-

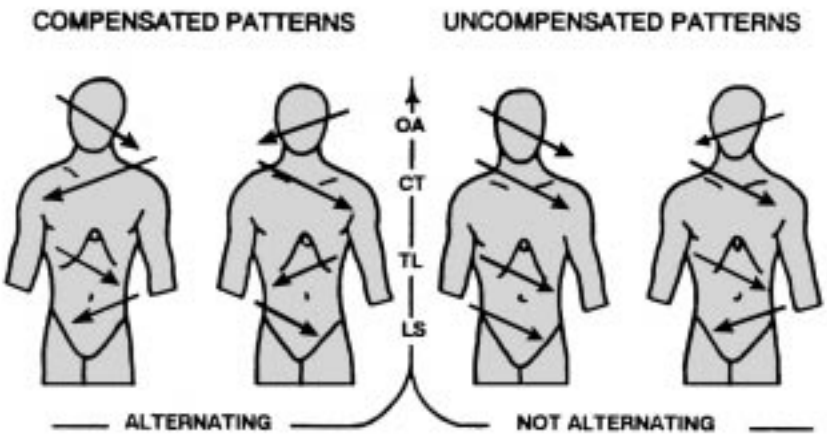

Figure 1. Compensated and Uncompensated Patterns. [Reprinted with Permission. Adapted from *Osteopathic Principles in Practice* by William A. Kuchera and Michael L. Kuchera, Copyright 1994.]

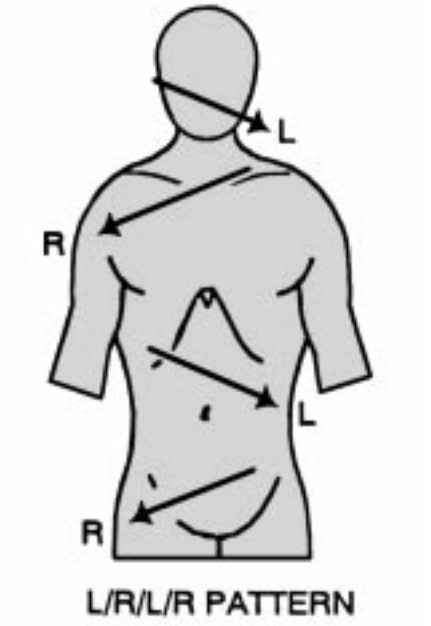

Figure 2. The Common Compensatory Pattern [Reprinted with Permission. Adapted from *Osteopathic Principles in Practice* by William A. Kuchera and Michael L. Kuchera. Copyright 1994.]

pensatory Pattern or CCP (Figure 2). The CCP can be seen as a bias of the fascias of the body along its length, occurring from the ground up. Such that, with respect to the feet the pelvic girdle is found to be rotated to the right, the lower thoracic outlet to the left, the upper thoracic outlet to the right, and the craniocervical junction to the left.

"The Tie that Binds"

The Common Compensatory Pattern can also serve as the common denominator between several of the therapeutic models used in osteopathic medicine. There are a number of recurrent patterns of dysfunction found in the muscle energy model that have already been mentioned and will be addressed further in the section entitled, Postural Asymmetries and the Postural Model. Janda[6] and Greenman[14] have described commonly found muscular adaptations where the postural muscles tend towards hypertonus and contracture while the dynamic muscles tend towards overstretch and hypotonus. These imbalances usually occur between the paired antagonist muscle groups in such a manner that the tight postural muscles, unopposed by the inhibited dynamic muscles mirror the sidebending and rotation of the body found in the common compensatory pattern. There are also many commonly found craniosacral patterns that are associated with the CCP. The relationships between the craniosacral model and the CCP are highlighted in a subsequent subsection entitled the "bent twig". Finally there are also numerous correlations between the postural model and the CCP which we will explore in some depth in later sections.

Of course as students and clinicians we all have an intuitive sense that all of these models should be interconnected, but what is their connection? This is a question that the osteopathic profession has been working with for a long time and it goes to the heart of one of the primary tenets of osteopathic philosophy, that "Structure and Function of the human body are interrelated at all levels."[15]

Thus far we have looked at the universal anatomical nature of the fascia and the universal clinical nature of the common compensatory pattern. To have a better understanding of how they are related and in turn how they relate to many different osteopathic models, let's look at these universal factors from a developmental standpoint. To begin with, how does the common compensatory pattern originate?

3. The Origin of the Common Compensatory Pattern

Figure 3 shows a brief overview of the development of erect posture.[16] We know that as the embryo is enfolded in the womb its back describes a C-curve. It is not one continuous curve but rather a series of bent segments that intersect at what will become the transitional junctions. The child attains upright posture first through the development an anterior cervical convexity and then an anterior lumbar convexity.

Zink[1] believed that the lumbar spine of the growing child was especially vulnerable to repeated minor traumas that result in twisting of the torso. He also felt that the ideal physiologic pattern was best suited for lo-

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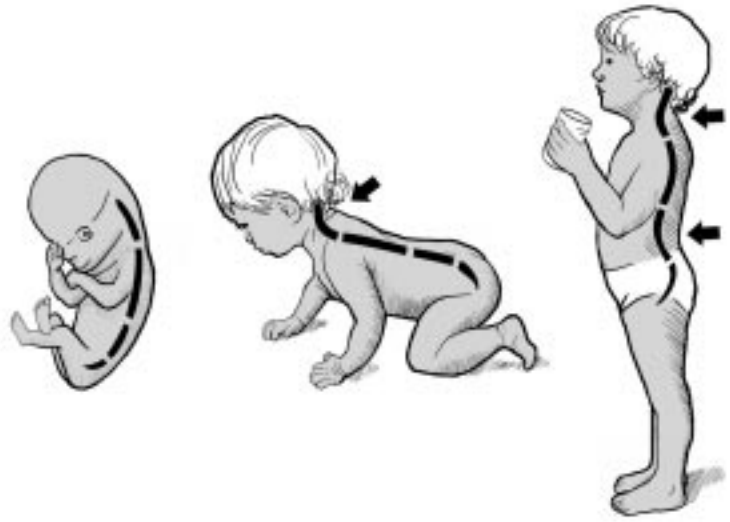

Figure 3. Developmental Stages. [Reprinted with Permission. Illustrated by Laura Maaske – Medimagery LLC, Copyright 2003. All rights reserved.]

comotion, and that while the CCP was not as efficient a pattern, it was very adaptive.

Implicit in these statements is the reasoning that during childhood development, as the infant attains the ability to crawl and then eventually to stand and walk, that they will adopt the more adaptive rotational pattern of the CCP. In other words, as a consequence of repeated minor traumas the lumbar spine develops a twist or bias of rotation. Then through the reciprocating rotational motions of walking this torsional bias is transmitted to the other junctional regions of the spine.

There have been several other reasons offered to explain the common compensatory pattern. It is generally known that there is a predisposition toward early left hemispheric dominance or cerebral lateralization in the human brain. This same cerebral lateralization has been found in primates and implies a genetic origin.[17] Gerchwind's theory[18,19] of cerebral lateralization acknowledges a genetic basis for predominance of left hemispheric dominance, hence right hand and foot dominance. He related variance in dominance to prenatal testosterone levels that account for a myriad of neurobiologic observations in children and adults. These findings include: (1) the excess of left-handedness in males, (2) male predominance in stuttering, autism and dyslexia, (3) superior verbal ability in females, (4) superior spatial ability in males, (5) left-handedness being more common in developmental disorders and learning disabilities, and (6) immune disorders being more common in non right-handers. Cerebral lateralization causes right hand and foot motor dominance, which through repetitive use is thought to cause the common compensatory pattern. Previc[20] postulated that right hand and foot dominance could also be in part due to left vestibular dominance. Interestingly enough he traced this vestibular lateralization to asymmetric positioning of the fetus *in utero* during the final trimester. We will discuss this concept in more depth in the section on postural control.

Some have even suggested a genetic basis by comparison with helical formations found in nature.[21] Structural asymmetries have also been implicated. Osteopathic clinicians have long thought that there is a positive correlation between the postural asymmetries (anatomic short leg, a small hemipelvis, and asymmetric position of the liver, etc.) and the CCP.[21] Hence, many have attributed the origin of the CCP to these asymmetries. Finally, still others have "…wondered if the fact that most children are delivered in a vertex presentation with the left occiput anterior might be a factor in the development of the functional asymmetry of the musculoskeletal system".[5]

As we have seen, Zink's explanation for the origin of the CCP has a developmental basis. There is further evidence, which will be discussed that supports the conclusion that the CCP and postural asymmetry may be developmentally related. It appears then; that there are several different factors related to the origin of the Common Compensatory Pattern.

1) Genetic Potential
2) Development Influences
3) Structural Asymmetries

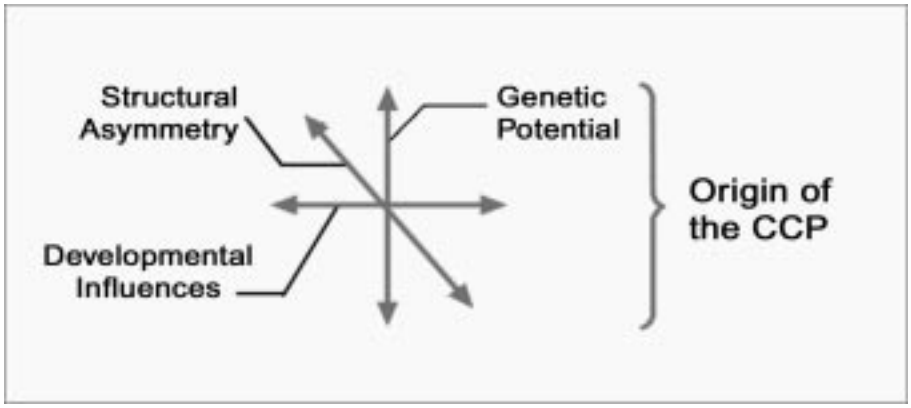

Figure 4. Origin of the Common Compensatory Pattern

This can be abstractly represented in the familiar xyz-axes of the Cartesian coordinate system and are shown in Figure 4.

For purposes of discussion we can divide developmental influences into the events that occur before, during and after birth. Gestation is the time period between conception and birth and lasts approximately 40 weeks. Birth itself is a period of marked environmental transition and is divided into the stages of labor and delivery. Then after birth, growth and development includes not only changes in the size of an individual but also continuing adaptations of the individual to their environment. Even once we achieve adult proportion development does not end. Bone can be remodeled throughout life as the relative stresses on it change. New collagen realigns in the connective tissue in response to vectors of stress. Finally, muscles continue to respond to stress through patterns of disuse and overuse and can adaptively change their physiologic type, i.e. Type I into Type II muscle fibers and vice versa.[22]

In the following sections we will examine several of these developmental influences that can have an impact human on structure and function. The first of these factors to be considered is fetal growth.

4. Fetal Growth

Fetal growth has been divided into three phases. The first phase, from conception to the early second trimester, involves cellular hyperplasia, an increase in the number of cells of all organs. This phase is followed by a period of continued hyperplasia and hypertrophy, involving both cell multiplication and organ growth. In the third phase, beyond 32 weeks, cellular hypertrophy is the dominant feature of growth. Cell sizes increase rapidly and fat deposition begins. Fetal weight may increase by as much as 200 grams per week.

In these later weeks of pregnancy, the fetus assumes a characteristic posture sometimes called its *attitude* or *habitus*. This characteristic posture results partly from the natural growth of the fetus and partly from the natural process of accommodation to the uterine cavity. The *lie* of the fetus is the relation of its long axis to that of the mother and is either longitudinal or transverse. The longitudinal lies are present in approximately 99% of labors at birth.[24] The presenting part determines the *presentation*, which in longitudinal lies results in either a cephalic or a breech presentation. Table 2 displays the presentations found at various gestational ages. [25]

We note that as pregnancy progresses the fetus is increasingly found in the longitudinal lie.

The reason for this is thought to be relatively straightforward. [23] Until about the 32nd week, the amniotic cavity is large compared to the fetal mass and there is no crowding of the fetus by the uterine walls. Beyond the 32nd week, on a relative basis, the amniotic fluid decreases and the fetal mass increases. Therefore as a result, the uterine walls are apposed more closely to the fetal parts. Data in the table also points out that an overwhelming majority of fetuses are found in the cephalic presentation as shown in Figure 5. Conventional wisdom explains why the fetus presents cephalically by pointing towards the piriform shape of the uterus. "Although the fetal head at term is slightly larger than the breech, the entire podalic pole of the fetus–that is the breech and its flexed extremities–is bulkier and more movable than the cephalic pole. Thus the bulkier podalic pole makes use of the roomier fundus." [23]

The *position* of the fetus refers to the relation of the fetal presenting part to the right or left side of the birth canal. Accordingly, with each presen-

TABLE 2. FETAL PRESENTATION AT VARIOUS GESTATIONAL AGES DETERMINED SONOGRAPHICALLY

Gestation (weeks)	Total Number	Percent		
		Cephalic	Breech	Other
21-24	264	54.6	33.3	12.1
25-28	367	61.9	27.8	10.4
29-32	443	78.1	14.0	7.9
33-36	638	88.7	8.8	2.5
37-40	463	91.5	6.7	1.7

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Figure 5. Left Occiput Anterior. [Reprinted with Permission. Illustrated by Laura Maaske – Medimagery LLC, Copyright 2003. All rights reserved.]

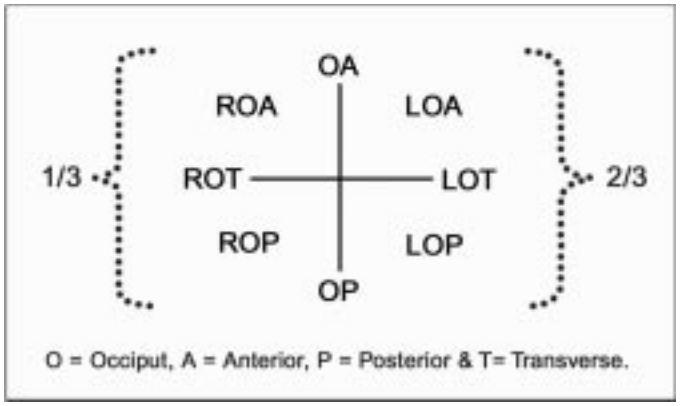

Figure 6. Fetal Presentation
LOA = Left Occiput Anterior, LOT = Left Occiput Transverse and LOP = Left Occiput Posterior. Of the three, LOA is the most frequent presentation and combined these three presentations comprise two-thirds of all births.

tation there can be two positions, either right or left. Finally, for still more accurate orientation, the relation of the presenting part to the anterior, transverse or posterior portion of the mothers' pelvis is considered *variety*. In a cephalic presentation, the presentation, position, and variety may be abbreviated and represented as shown in Figure 6. About two thirds of all vertex presentations are in the left occiput position, and about one third in the right.

As this data indicates, the primary fetal lie through pregnancy and through labor and delivery is with the head rotated to the left with the arms and legs otherwise curled in accommodation to the restrictions of the uterine cavity. The most compact profile for the fetus is for the arms and legs to curl in opposing directions with a resultant rotation along the longitudinal axis of the fetus. Some authors including Ida Rolf, PhD (the founder of Rolfing) have pointed out that this rotation could be an important factor in the final shape of the fetus.[26] It appears that as it grows, the fetus, the infant and ultimately the adult expands in size but retains this early pattern of rotation (Figure 7). There is a great deal of information, which supports this premise.

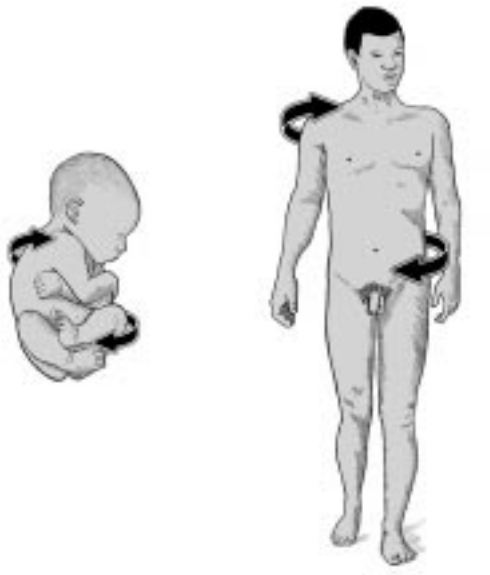

Figure 7. Fascial Bias in the Fetus and the Adult. [Reprinted with Permission. Illustrated by Laura Maaske – Medimagery LLC, Copright 2003. All rights reserved.]

First consider the connective tissue. We know it makes up a high proportion of body mass, connecting, supporting and organizing the body as a whole. It is known that during fetal development the majority of connective tissue growth occurs during the final trimester, during the time of greatest fetal restriction. Further, research demonstrates that pressure or tension in one area of the embryo results in increased secretion of connective tissue fibers in that area, and that these fibers tend to organize themselves along lines of tension.[26] Keeping in mind that all adults show adaptive rotational patterns, the most common being L/R/L/R. By comparison one can see the similarity between the fascial bias of the fetus and the common compensatory pattern in the adult. In both patterns the AO fascia rotates to the left and the LS fascia rotates to the right.

In the following section, we find another developmental factor–labor and delivery–which is also thought to have a significant impact on human structure.

5. Labor and Delivery

"Just as the Twig is bent, the Tree's inclined"

—Alexander Pope

The "bent twig" is an analogy used to describe the shape of the cranial bones and how they are often permanently modified by birth trauma before full ossification takes place. The perinatal period has been called "the

valley of the shadow of birth".[27] This somewhat melodramatic statement underscores the extreme nature of this "normal" process. A process traditionally recognized by the osteopathic profession, as one that can have potentially significant effect throughout the life of the individual.

The majority of the cranial bones of the fetus are relatively flat plates consisting of one layer of primary cancellous bone with no serrations. The vault is relatively large in comparison to the face and the rest of the body and is characterized by somewhat prominent frontal and parietal eminences. There are six fontanelles, one at each parietal angle, one at each mastoid, one at lambda in the occiput and one at bregma in the frontals. The base of the fetal skull is comprised of the occiput, made up of four flat cartilages and the temporal bones, each containing six separate cartilages. There is a great deal of prenatal molding of the fetal skull. "The vault lies against the pelvic inlet for the last two months or more—an inlet in which the sacrum sags forward while the ilia are pulled back by the gluteals in the effort to resist the anteriority of the pelvis".[27] Uterine contractions normally exert a pressure on the amniotic cavity, and subsequently on the fetus itself, varying from 4.5-26.5 pounds per square inch.

The intraosseous membranes serve as the only really effective protection for the immature brain during the last month prior to delivery when molding is taking place, as well as during the stress of actual delivery. The compressive forces of the uterus are carried by way of the spine to the base of the skull. Since the occiput is the presenting part it receives the most pressure, therefore ossification begins in the condylar parts before the other cranial bones.[27] "The skull of the infant is highly vulnerable to forces of labor. The physiological lack of development, the pliability necessary for the birth process…the disproportion between the passage and the passenger–all these militate against the proper growth and development essential to normal structure and function…"[27]

The mechanism of labor refers to the changes of the fetus as it passes through the birth canal. With the occipital presentation, the head must undergo several movements to accommodate to the maternal bony pelvis. This process has been divided into seven cardinal movements (1) engagement, (2) flexion, (3) descent, (4) rotation, (5) extension, (6) restitution, and (7) expulsion.[29] The following drawings, Figures 8 through 12 depict the mechanism of labor with respect to the most common LOA presentation. Each of the cardinal movements will be discussed separately.

Engagement is defined as descent of the biparietal diameter of the head below the pelvic inlet. Clinically, the head can be palpated below the level of the ischial spines. The fetal head enters the transverse diameter of the pelvic inlet, with the occiput to the left and with the saggital suture parallel to the long axis of the inlet.

Flexion of the neck will increase because of the drag of the forehead against the pelvic inlet. It allows for smaller diameters of the fetal head to present to the maternal pelvis.

Descent is in the oblique diameter because of resistance of the pelvis, which turns the occiput 45° to the left anterior position. As the head descends the left parietal bone will stem beneath the promontory of the sacrum.[27] The medial border of the left parietal will underride the edge of the more rapidly advancing right parietal bone. Meanwhile the cerebrospinal fluid and blood have partially transuded out of the cranium to lessen its volume. The occiput and frontals telescope beneath the parietals to further decrease the size of the head.

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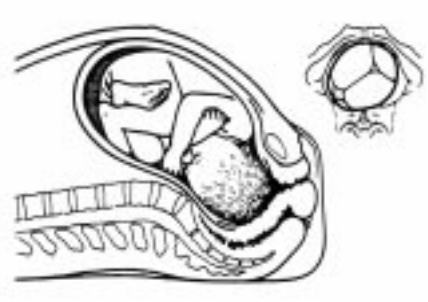

Figure 8. Engagement with Flexion. [Reprinted with Permission. Adapted from *Basic Gynecology and Obstetrics* by N. Gant and F. Cunningham. Copyright Appleton & Lange 1993, the McGraw-Hill Companies, New York, NY.]

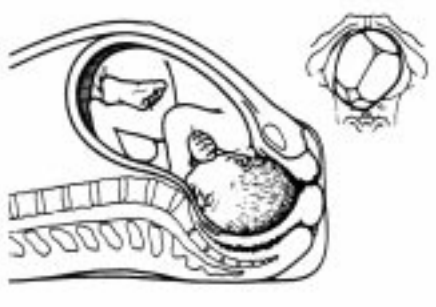

Figure 9. Descent and Beginning Rotation. [Reprinted with Permission. Adapted from *Basic Gynecology and Obstetrics* by N. Gant and F. Cunningham. Copyright Appleton & Lange 1993, the McGraw-Hill Companies, New York, NY.]

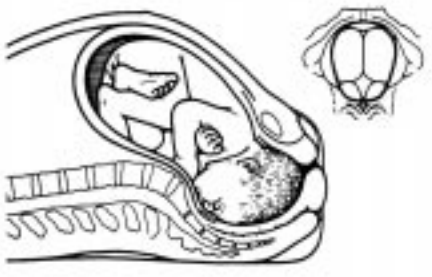

Figure 10. Complete Rotation and Beginning Extension. [Reprinted with Permission. Adapted from *Basic Gynecology and Obstetrics* by N. Gant and F. Cunningham. Copyright Appleton & Lange 1993, the McGraw-Hill Companies, New York, NY.]

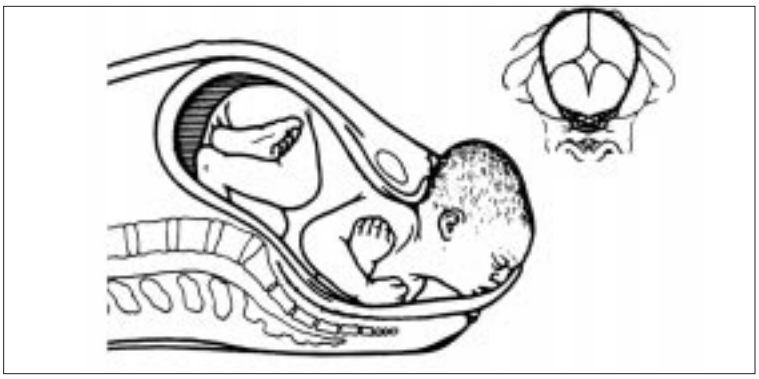

Figure 11. Complete Extension.
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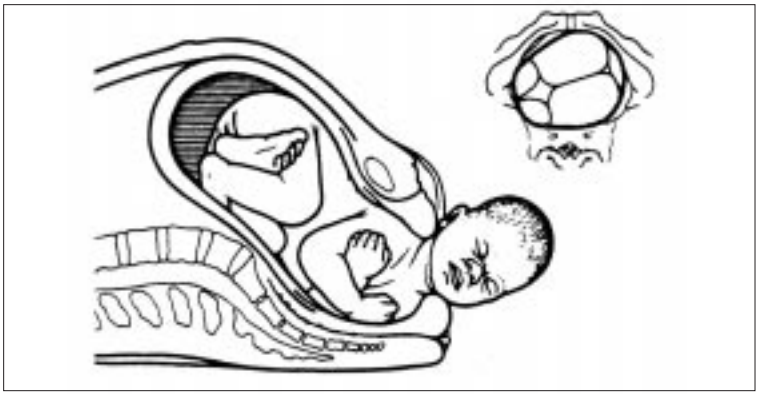

Figure 12. Restitution.
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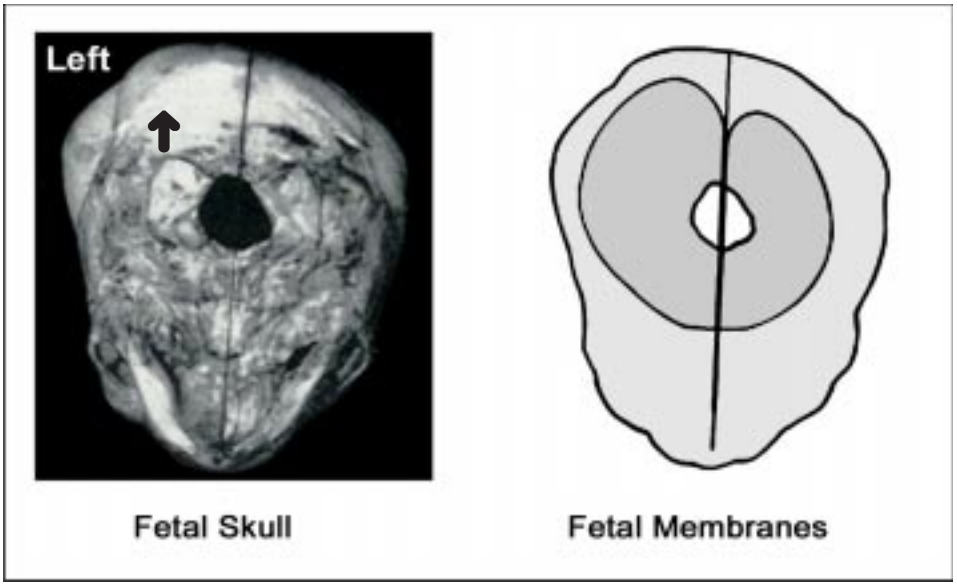

Figure 13. Cranial Asymmetry. [In the Public Domain. *Osteopathy in the Cranial Field*, 1st Edition, edited by Harold I. Magoun, Sr., published by the Sutherland Cranial Teaching Foundation. Fort Worth, TX.]

Rotation is then completed, which brings the saggital suture into an anteroposterior position. During internal rotation the occiput is subjected to significant forces of rotation and lateral resistance. After internal rotation the sharply flexed head reaches the vulva, it undergoes **extension**, which brings the base of the occiput into direct contact with the inferior margin of the symphysis. The head is delivered by further extension as the occiput, bregma, forehead, nose, mouth, and finally the chin pass successfully over the anterior margin of the perineum (Figure 11).

Restitution occurs when the delivered head externally rotates back to a 45° oblique position. The occiput, which was originally directed to the left, now lies towards the left ischial tuberosity (Figure 12).

Expulsion is the final delivery of the fetus from the birth canal and includes delivery of the right shoulder and then the left shoulder.

The "bent twig": During the internal rotation movement of labor the head moves from the oblique to the anteroposterior position. At this time the fetal skull must move against the resistance of the maternal symphysis. It is thought that this resistance is sufficient to keep the squamous portion of the occiput from achieving complete restitution. In a study of 1250 infant heads, Frymann[30] found less than 12% to be symmetrical with 69% displaying disturbances of the condylar parts. An example of this is asymmetry is shown in the skull of a newborn in Figure 13.

It shows that the squama of the occiput is bulging to the left and flattened on the right with mediolateral compression on the left and posteroanterior compression on the right. The lambdoidal suture overrides on the left and is separated on the right. The diagram to the right displays concurrent membranous tension and warping

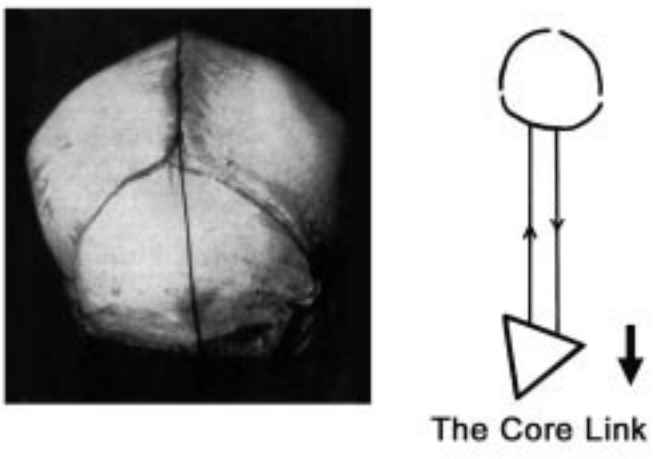

Figure 14. Craniosacral Tilt. [Reprinted with Permission. Adapted from the *American Academy of Osteopathy Yearbook* (1983) by Harold I. Magoun, Sr.: "Idiopathic adolescent scoliosis: A reasonabale etiology (1975)"]

of the tentorium cerebelli.[27]

Magoun[31] also describes a relationship between distortions of the infant head and the sacral base (Figure14), with the tilt of the occiput being similar to that of the sacrum. He commented that the sacrum necessarily assumes the same tilt because the meninges of the spinal cord attach firmly to the foramen magnum, the 2nd and 3rd cervicals and the 2nd sacral segment. This idea of a functional continuity between the cranium and the sacrum through the dura is an important osteopathic concept and that has been termed the "Core Link".[32]

It is believed that after delivery that most of the distortion of the fetal skull is corrected by the infant through crying which balloons the skull, and by sucking, which flexes the sphenobasilar junction thus normalizing the pull of the intracranial membranes.[27] Although in the majority of adults, residuum of the distortion persist. Given that in vertical posture the eyes are level in the horizontal and coronal planes, then these distortions would produce a vector of rotation to the left side (shown as an arrow in Figure 13) that could affect the incumbent neck and trunk. Also through the core link there could be a vector of sidebending of the sacrum and pelvis to the right (shown as an arrow in Figure 14). With the ubiquitous nature of this distortion it is likely that it is in part responsible for the CCP. These distortions could either cause or enhance the rotational bias of the fascia at the craniocervical junction to the left and may also increase the side bending bias of the pelvis to the right, both of which are found in the common compensatory pattern.

There could also be functional consequences to distortion of the cranial base. Clinical evidence that indicates that disturbance at craniocervical junction can have significant and primary affect upon balance and postural control. "By far the most important proprioceptive information needed for the maintenance of equilibrium is that derived from the *joint receptors of the neck".*[33] Lewit demonstrated that articular dysfunction at the craniocervical junction can cause an unequal distribution of weight between the lower extremities.[13] When weight distribution was measured by instructing a patient to put equal weight on both feet while standing on a pair of matching scales. Patients with movement restriction at the craniocervical junction, showed that one limb consistently registered at least 5kg (2.3lbs.) more than the other limb.

We have just seen how the developmental factors, prenatal habitus and perinatal labor and delivery, could have an impact on anatomic structure. We also have begun to see how these factors could affect function. One of the most important of all human functions is postural control.

6. Postural Control

The antigravity function of posture enables us to maintain an upright position and orientation. Postural control involves multisensory pathways, including visual, vestibular, and somatosensory data from proprioceptor and cutaneous receptors.[34] The central nervous system uses this sensory information to create an internal frame of reference that regulates the center of gravity. As conceptualized in Figure 15, feedback from somatosensory monitors includes neck and lower limb proprioceptors and pressor

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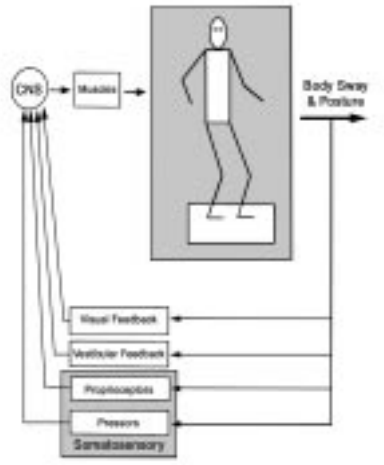

Figure 15. Postural Control. [Reprinted with Permission. Adapted from *Functional Movement in Orthopaedic and Sports Physical Therapy* by Bruce Brownstein and Shaw Bronner, Elsevier Science, Oxford, UK. Copyright 1998. Elsevier Inc.]

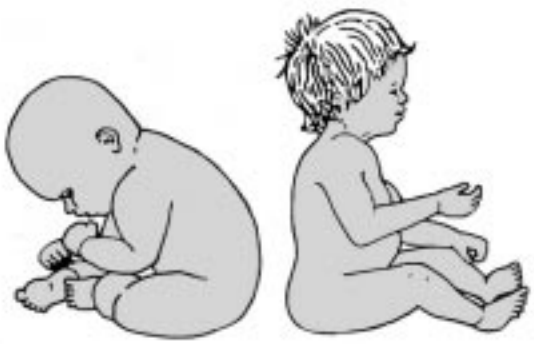

Figure 16. The Labyrinthine Reflex. [Reprinted with Permission. Adapted from *Muscles, Nerves and Movement* by Barbara Tyldesley and June Grieve, Blackwell Publishing, Oxford, UK.]

receptors from the feet. Feedback from these receptors is used to initiate postural compensation resulting in the activation of muscle groups to maintain or restore equilibrium through body sway. The central nervous system can also prepare against or anticipate disturbance in the center of gravity or the center of mass through feed forward control from visual and vestibular input.[34] The vestibular system is responsible for stabilizing the position of the body, head and eyes in space.

The earliest indication of vestibular control[35] is seen in the newborn with the labyrinthine reflex (Figure 16). This postural reflex which depends upon stimuli from both vestibular organs functions to automatically extend the head and hold it in an orthostatic posture.

Underlining the importance of this reflex we find that studies of posture in the adult show that the most stable segment of the body is the head and that displacement of the head is less than that of the trunk during balancing activities. We also know that when the head is in a near vertical position an adult can determine as little as a one-half degree of vertical tilt.[33] It is apparent that extreme sensitivity in the upright position is of major importance for maintenance of precise vertical equilibrium.

We know that each vestibular apparatus exerts control over the extensor muscle groups on both sides of the body, but its predominant effect is on the ipsilateral extensor or antigravity muscle groups. In other words the left vestibular apparatus primarily affects the left antigravity muscles while the right vestibular apparatus similarly affects the right side. This physiology becomes especially meaningful when we realize that there is a congenital or genetic bias towards one-sided vestibular dominance. This human trait is identified as vestibular lateralization.

Vestibular Lateralization: Several researchers have confirmed that left vestibular dominance occurs in roughly two-thirds of the human population.[36-40] Previc[20] describes a possible prenatal mechanism (figure 17) for the origin of left vestibular dominance. "Because the right side of the body faces outward in the left fetal position, the acceleratory component to the maternal walk would, from the standpoint of the fetus, be registered rightward. The more salient inertial force would consequently be leftward, providing for a more effective stimulation of the left utricle"; thereby promoting early growth and development of left vestibular neural and cortical control.

Overall, antigravity extension of the body is maintained by (1) Monosynaptic stretch reflexes operating at

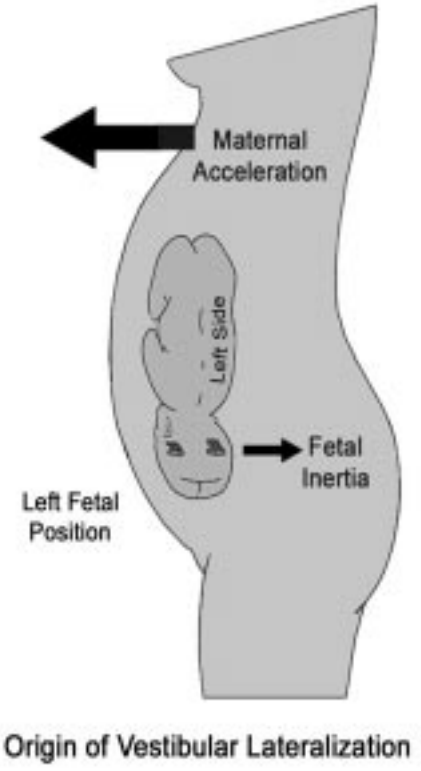

Figure 17. Origin of Vestibular Lateralization. In the Public Domain. Adapted from *Psychological Review*, 98(3): 299-334, by F. Previc: "A General Theory Concerning the Prenatal Origins of Cerebral Lateralization in Humans"

the level of the spinal cord, (2) Excitatory ipsilateral input from the vestibular organs and (3) Inhibitory input from the neck proprioceptors and the frontal cortex. Antigravity flexion activity of the body is under the control of the motor cortex.[20]

Therefore with general activities of daily living, one leg is primarily used for postural support (vestibular dominance) and the other for most voluntary activities (motor dominance). Kicking a ball (Figure 18) is a typical example; most people kick with the motor dominant right leg while simultaneously supporting themselves with vestibular dominant left leg.[41]

In support for this premise we find that in the majority of the adult population that the left leg has greater size and muscle mass.[42] Furthermore, this

Figure 18. Vestibular and Motor Dominance. [Reprinted with Permission. Adapted from *Anatomy of Movement* by Blandine Calais-Germain, Eastland Press, Seattle, WA. Copyright 1993. all rights reserved.]

physical asymmetry is not found at birth, but is a response to later growth and development.[43,44] This clearly shows how function can affect structure and further demonstrates the reciprocal nature of the two.

In the previous two sections we have discussed two mechanism that could cause asymmetric pressure upon the legs. The first is distortion of the cranial base induced by the birth process, which could result in persistent pressure differences between the lower extremities. The second is a functional control mechanism; we find that people primarily use only one leg for postural support. Could these factors coupled with later development be the explanation for why we commonly find growth differences between the lower extremities in children?

7. Leg Length Growth in Children

Studies of school children show that the majority of children show leg length discrepancies and that the likelihood of the discrepancy increases with a child's age.[45] Pearson[46] radiographed a group of 1446 school children between 5 and 17 years of age, 80% had at least a 0.16cm (1/16-inch) discrepancy and 3.4% had a difference of 1.3cm (1/2 inch) or more. By comparison, in another study, 75% of elementary school children displayed a measurable leg length discrepancy, while 92% of similarly measured senior high school students showed measurable leg length differences. This suggests that differences in leg length tend to increase as children grow. Still other studies show that if leg length differences are corrected with heel lifts during childhood then the discrepancies often become smaller.[47-49]

Figure 19. Long Bones of the Newborn. [Reprinted with Permission. Adapted from *Grant's Atlas of Anatomy*, 7th Edition, by J. Anderson, Lippincott William & Wilkins, Philadelphia, PA.]

At birth[50] the bodies or diaphyses of the long bones in the lower extremities are largely ossified, but most of the ends or epiphyses are still cartilaginous (Figure 19). During the first two years after birth the epiphyses become ossified with only the articular cartilage and the epiphyseal plate remaining cartilaginous. Growth in the length of the long bones continues at this plate until it is replaced by spongy bone at 18-20 years of age. All together there are eight of these growth plates, two each for the femur and the tibia, in both the lower extremities. There are a number of references to asymmetric growth of the lower extremities, as being the cause of leg length discrepancies in the postural literature. Cathie[51] attributed leg length disparity to very slight epiphyseal injuries that disturbed normal bone growth. Schwab[52] thought that simple unequal growth was the most common cause of unequal leg lengths. Unequal growth may result from pathologic involvement of long bone epiphyses by infection, trauma, tumor, radiation and disease, the most notable being poliomyelitis. Furthermore, during growth or after completion of growth, leg length inequity may result from fracture.[53,54]

A broader and more consistent explanation of commonly found asymmetric leg lengths could be that it is the result of asymmetric pressure along the length of the long bones during growth. Kappler[55] reported that the pelvis typically side shifts towards the longer leg; hence, there should be more pressure over the long leg side. Morscher[53] and Gofton[56] argue convincingly that there is increased pressure upon the hip and leg on the long leg side. Some authors invoke Wolff's

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law as causative, and believe increased growth of the long leg is secondary to increased pressure. On the other hand, there is experimental evidence that shows decreases in pressure parallel to the growth axis in the long bones favor growth in length, whereas increases inhibit and may even stop epiphyseal growth.[57] Finally, other researchers have taken a middle road and have said, "between zero load and some limit, increasing loads increase growth".[58] Based on the clinical data, it would be reasonable to assume that increased epiphyseal pressure, within certain physiologic ranges, encourages growth. This raises the question. From an etiological perspective, is it the short leg syndrome, or the long leg syndrome? There needs to be further study to determine which leg in the growing child routinely has the most pressure and relate that to which leg either does or does not grow.

We have discussed several possible mechanisms that may explain the origin of the CCP (1) developmental fascial bias (2) birth trauma and (3) asymmetric leg growth. The latter factor resulting in leg length inequity, the most commonly found postural asymmetry.[59] In the following section we will examine the relationship between these developmental factors and the postural model.

8. Postural Asymmetries and the Postural Model

Commonly found postural asymmetries and their biomechanical relationship to one another are the basis of the current postural model.[60]

There are three primary regions of anatomic or postural asymmetry that have been studied with regards to the postural model. They are the *lumbosacral junction*, the *lower extremities* (including leg length, foot posture and foot arches) and the *craniocervical mandibular junction*. This last term, craniocervical mandibular may be unfamiliar, it was coined by dentists[61] and it reflects contributions from the other disciplines concerning posture. Dentists and orthodontist, as well as physical therapists have shown that occlusion and the mandibular rest position are also intimately related to the posture of the head and neck. As we investigate information from these fields we will see that commonly found postural asymmetries in all of these regions are also biomechanically interrelated. A conceptual overview of these regions and their relationship to one another is displayed in Figure 20. Each of the primary regions of postural asymmetry will then be examined in some detail.

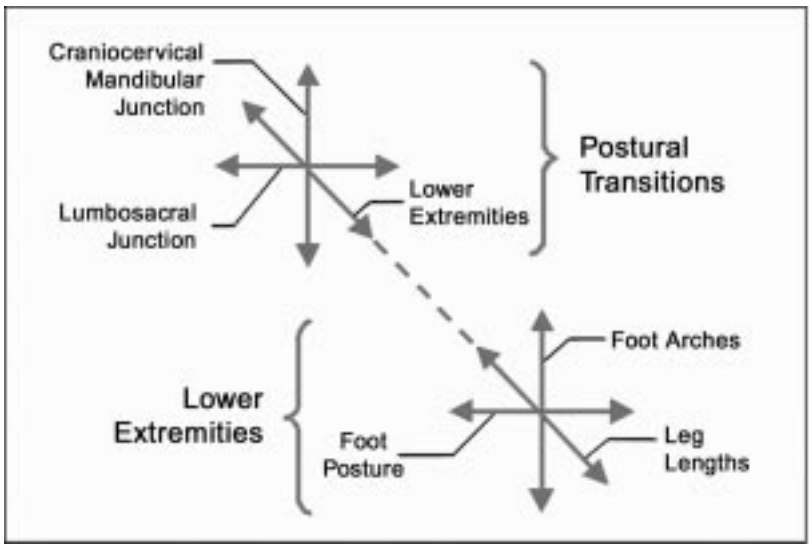

Figure 20. Primary Regions of Postural Asymmetry.

Lumbosacral Junction: Denslow and Chace[62] measured leg length discrepancy in 361 subjects. They found a higher incidence of low right femoral heads. In another study with 294 subjects they recorded the lateral curvature of the spine. This group demonstrated a high correlation between the direction to which the curvature occurred and the short leg with the lateral curvature most frequently occurring toward the short leg side. In yet another study these researchers measured pelvic rotation and discovered that pelvic rotation most commonly occurred contralateral to the short leg side. A composite of these findings produces the so-called "typical case" i.e., the most commonly found postural asymmetries. In the majority of cases where postural asymmetry is present Denslow and Chace[62] found that the lateral curvature is towards the short leg side with pelvic rotation towards the long leg side. This suggests a coupling of lumbopelvic mechanics, and they described two possible mechanisms for this coupling: (1) The two innominate bones and sacrum rotate as a block and (2) The two innominate bones rotate around the sacrum. Mitchell[63] definitively describes opposing rotation of innominate bones about a transverse axis through the lower sacrum as compensatory to leg length discrepancy with anterior rotation on the short leg side and posterior rotation on the long leg side. Denslow and Chace[62] further speculated that the high femoral head "drives" the anterior portion of the pelvis upward and backward, thus rotating the pelvis to that side and that the pelvis drops down on the low femoral head side. Thus unleveling the sacral base and producing a "buckling" of the lumbar segments.

Friberg[64] also described pelvic rotation as occurring opposite to that caused by lumbar coupling (Figure 21). He described the buckling or lat-

eral curve of the lumbar spine as a functional scoliosis secondary to the leg length inequity and the associated sacral base declination.

The lumbar spine follows Type I mechanics with side bending away and rotation towards the convexity, with an increase of backward bending. If one considers the pelvis as moving in block as described by Denslow and Chace, then the motion of the pelvis would also appear to follow Type I – like mechanics with side bending towards and rotation away from the short leg.

In the instance of the short right leg, the pelvis will then generally rotate to the left. This seemingly conflicts with the side bending and rotational pattern of the CCP; side bending and rotation both to the right. Furthermore, after observing obvious pelvic rotation to the left on a standing A/P film of the pelvis you can then manually test a patient for pelvic rotation in both standing and supine positions and find a clinically apparent rotational bias to the right. This disparity has certainly been a source of confusion for this author. How can these findings be reconciled? Since there is a great deal of plasticity in the pelvis, Zink[1] explained this disparity as a simple predominance of fascial twist (rightward fascial bias) over bony mechanics (left rotation) in the pelvis. Although if you conclude that motion testing of the pelvis follows Type I mechanics of the L/S junction you find that the disparity is resolved. The typical L/S junction test is performed with the patient prone, with the examiners' hand on the PSIS. The examiner lifts and medially rotates the pelvis to find ease of motion.[65] With the spine in the neutral position L-5 is sidebent left and rotated to the right. Rotation of the pelvis to the left is restricted by "facet locking" between L-5 and S-1. Thus, with motion testing of the L/S junction we could expect to find greater ease of motion to the right regardless of actual rotation of the bony pelvis. Another explanation for this paradoxical rotation involves the interaction of the lower extremities with the pelvis. Postural influences from the lower extremities include not only the leg lengths but also certain commonly found postures of the feet.

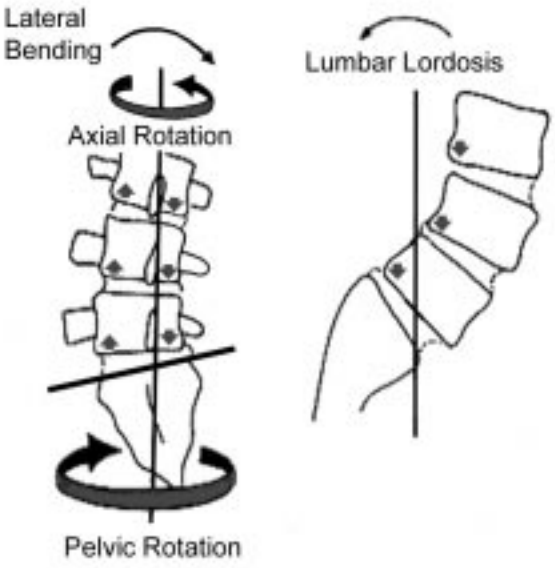

Figure 21. Lumbopelvic Coupling. [Reprinted with Permission. Adapted from *Spine*, 8(6): 643-651, by O. Friberg: "Clinical Symptoms and in leg length inequality", Lippincott William & Wilkins, Philadelphia, PA.]

Lower Extremities: The posture and architecture of the feet can have significant effect on leg length and the attitude of the pelvis. The most common asymmetrical foot position is the pronated foot (Figure 22), which is typically found on the long leg side and is considered compensatory to the long leg.[66] The supinated foot is also commonly seen and it is associated with the short leg.

A well-known result of foot posture is its capacity to affect the length

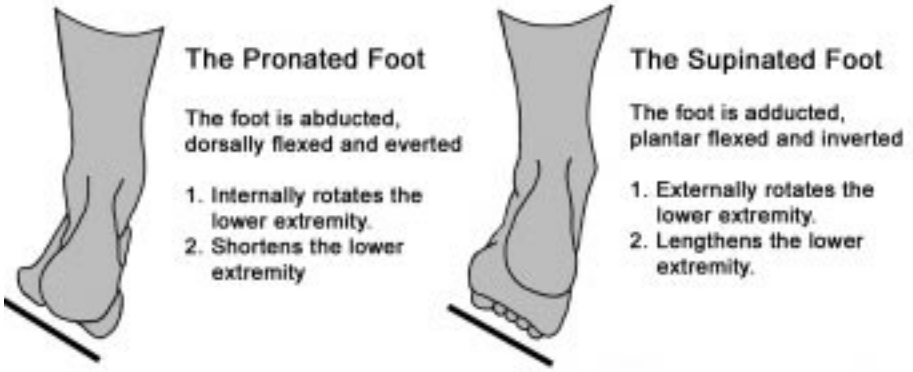

Figure 22. Foot Postures

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of the lower extremity.[66] The pronated foot acts to shorten the long leg and the supinated foot lengthens the short leg.[67] The pronated foot also causes internal rotation of the lower extremity and the supinated foot results in external rotation of leg and thigh.[68] Rotation of a lower extremity will also produce rotation of the pelvis. A supinated foot causing external rotation of the lower extremity will result in ipsilateral rotation of the pelvis. While on the other hand, with a pronated foot we find contralateral rotation of the pelvis. It is also reasonable to assume that rotation of the lower extremity causes change in the anteroposterior position of the femoral heads. The effect of forward position of one femoral head combined with posterior position of the opposite would result in an overall rotation of the bony pelvis.

The left side of Figure 23 depicts a posterior view of a person with a short right leg, a pronated left foot and a supinated right foot, while the right side of the figure shows cross sections of each corresponding level of the lower extremities and the pelvis.

The pronated position of the left foot causes internal (rightward) rotation of the left lower extremity and will result in a posterior positioning of the left femoral head. The supinated position of the right foot, resulting in external (also rightward) rotation of the lower extremity, causes an anterior positioning of the femoral head. Combined, one femoral head posterior and the other anterior, the result is rotation of the bony pelvis to the left or opposite to that of either lower extremity and thus provides an explanation for why the CCP fascial pattern differs from the bony radiographic presentation in the standing posture. This mechanism of anteroposterior femoral head position also helps to explain other clinical findings. For example, we commonly find patients with both feet pronated and with this we also observe increased lordosis. In this instance both femoral heads are positioned posteriorly which appears to translate the pelvis backward and results in a compensatory increase in lumbar lordosis. A corollary mechanism is bilateral supinated feet which results in an anterior translation of the pelvis. With this finding we clinically observe decreased lumbar lordosis or straightening of the spine. The pronated foot is generally associated with a subtalar joint (STJ) valgus and the supinated foot is associated with STJ varus. It should be kept in mind though that oftentimes you see a STJ varus with the pronated foot which can be the consequence of either an ipsilateral forefoot valgus or a tibial varus, or both. In other words, the position of the STJ and its coupling with lower extremity rotation depends upon an interaction between the rearfoot, the forefoot and the tibia.

Beyond these biomechanics there are also other fascial interactions between the arches of the feet and the attitude of the pelvis. Clinical experience suggests that bilateral pes planus is associated with a decrease in

Figure 23. The Relationship between Pelvic Rotation and Foot Postures

TABLE 3. A SUMMARY OF LOWER EXTREMITY EFFECTS

Postural Asymmetry	Sacral Base Declination	Pelvic Rotation	Pelvic Side Shift	Lordosis
Short Leg	Ipsilateral Low Base	Contralateral Rotation	Contralateral Side Shift	Increases
Unilateral Pronation	Ipsilaterally Lowers Base	Contralateral Rotation	Little or no effect	Little or no effect
Unilateral Supination	Ipsilaterally Raises Base	Ipsilateral Rotation	Little or no effect	Little or no effect
Bilateral Pronation	No effect	No effect	No effect	Increases
Bilateral Supination	No effect	No effect	No effect	Decreases
Supination & Pronation †	Towards Level	Decreases	Decreases	Decreases

† Typically the pronated foot is found on the long leg side and the supinated foot on the short leg side.

the lumbosacral angle and bilateral pes cavus is associated with an increased lumbosacral angle. Table 3 summarizes a number of the commonly found biomechanical interactions between the lower extremities and the lumbopelvis.

To reiterate in the postural model the body's response to lower extremity asymmetry are the commonly found somatic dysfunctions shown in Figure 24. These findings include (1) upslipped innominate on the left or downslipped right, (2) cephalad pubes left or caudad pubes right, (3) non-neutral FSR_L dysfunction at L-4 and/or L-5, and neutral S_LR_R at L-5 and (4) left on left sacral torsion.[69]

Other findings associated with the anatomical short right leg include a pronated left foot with a supinated right, an anteriorly rotated right innominate, and a posteriorly rotated left innominate. Functional rotoscoliosis is observed with a lumbar convexity to the right, thoracic convexity to the left and cervical convexity to the right.

To complete the postural model we should also examine the craniocervical mandibular junction and it's association with posture, because it has been known for a long time that structural and functional asymmetries at this junction can have profound effect on overall posture.

Craniocervical Mandibular Junction: Regarding fascia of the head and neck and its effect on the body as a whole Cathie[70] wrote, "Dental lesion and changes in the temporomandibular articulation are, singly or combined, capable of causing varied local and or distant disturbances." Conversely, we also know that fascial strains produced by structural asymmetries can directly contribute to craniomandibular dysfunction.[71-74] Magoun[75] summarizes this reciprocal relationship in the following manner, " While chronic postural

Figure 24. Common Structural Asymmetries

tension can be a major factor in the maintenance or recurrence of cranial lesion pathology, it is equally true that faulty cranial mechanics, often existing since birth, can adversely influence all the structures below."

This is not necessarily an easy relationship to understand. But if we look at head posture in the saggital plane (Figure 25) we see that when the head is in an ideal, orthostatic position, its center of gravity is slightly anterior to the vertebral column.[76] There must be balanced tension between the anterior and posterior craniocervical bony and myofascial structures in order for the head to remain erect. Any change in the structures anterior to the cervical spine will necessitate compensatory changes in either the cervical spine or the posterior myofascial structures or both.

The most critical anterior bony relationship is dental occlusion.[77-80] Thus in order for balance to be maintained there must be proper occlusion. For example it has been shown that with the Class II occlusion (overbite) is associated with cervical lordosis and forward head posture while the Class III occlusion (underbite) is associated with a straightening of the anterior or the normal anterior cervical curvature with a posterior head posture.[81] Several researchers have established a relationship between total posture and the stomatognathic system.

Using electromyography, Strachan and Robinson[72,73] showed that they could correct abnormal muscle firing sequences of masticatory muscles found in patients with malocclusion by correcting their leg length discrepancies with heel lifts. What's more, when they removed the corrective heel lifts, they recorded resumption of the abnormal electromyographic firing sequences. Thus demonstrating a relationship between correction of the short leg and correction of malocclusion. Wheaton[82] also found several relationships between the mandibular rest position, occlusion, and posture. Of these, the most significant positive correlations linked mandibular rest position with incisive position and the long leg. (The incisive position is a comparison of midline between the central maxillary and mandibular incisors in the occluded position.) In other words she found that the mandible tends to deviate in the same direction as the teeth and also toward the same side as the long leg.

Rocabado[83,84] put forth an influential conceptual model that states that ideal head posture is dependent upon three parallel lines of reference and these are the (1) bipupilar, (2) vestibular and (3) transverse occlusal planes (Figure 26). He surmised that the horizontal orientation of these planes would permit the visual gaze and vestibular system to remain level with the ground. He postulated that any change in the normal horizontal and parallel relationship of these planes to each other and to the ground would result in compensatory adaptations (flexion/extension, side-bending/rotation) by the incumbent spine.[83]

Huggare and others[85] studied the effect of scoliosis on head posture.

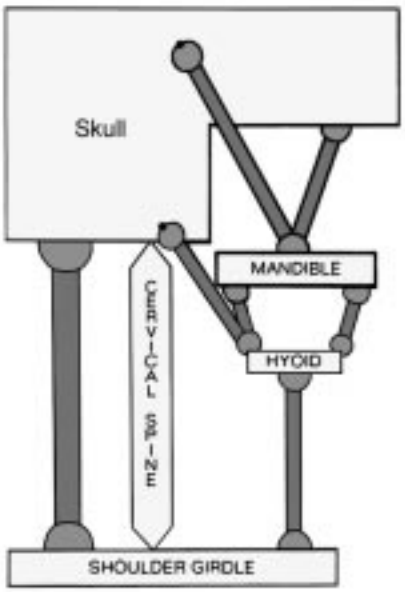

Figure 25. Saggital Head Posture. [Reprinted with Permission. Adapted from *New Concepts in Craniomandibular and Chronic Pain Management,* edited by Harold Gelb, Elsevier Science, Oxford, UK. Copyright 1994 Mosby Inc.]

Figure 26. Coronal Head Posture. Three parallel lines of reference: 1. Bipupilar Plane 2. Vestibular Plane 3. Transverse Occlusal Plane. Reprinted with Permission. Adapted from the *International Journal of Orofacial Myology,* 17(3): 8-10, D. MacConkey: "The relationship of posture and dental health"

Figure 27. Cephalmometric Studies. A composite of patients with scoliosis. Reprinted with Permission. Adapted from *Proceedings of the Finnish Dental Society,* 87(1): 151-8, by J. Huggare, P. Pirttiniem, W. Serlo: "Head posture and dentofacial morphology in subjects treated for scoliosis:

They found a high incidence of malocclusions in the scoliotic population, especially lateral malocclusion (crossbite). A composite cephalometric drawing of the location of these findings is shown in Figure 27. There was very little cranial tilting, but the overwhelming majority showed significant lateralization of the apical vertebra with compensatory craniocervical deviation to the opposite side. There was also increased rotation of the orbital, maxillary and mandibular planes in the frontal plane. Tilting of the mandibular plane, considered a vertical rotation in the frontal plane around a horizontal axis, is accompanied by a loss of posterior vertical dimension on one side of the bite with loss of anterior vertical dimension on the opposite side.[86]

Gelb[87] found that over time patients with a short right leg would develop left-sided loss of vertical dimension in the jaw. He found in these patients characteristic right-sided face changes that included (1) a higher eyebrow, (2) a higher and apparently larger eye, (3) a higher ear and (4) an up turning of the lips. Travell[88] noted that a useful clinical clue for identifying pelvic asymmetry and leg length discrepancy was that, "One side of the face is also smaller; this is most easily seen as a shorter distance between the outer corners of the eye and mouth". Relating to the remainder of the body Gelb[87] generally found the level of the shoulders, breast and hips to be lower to the right side. Royder[71] also found these common postural changes associated with the short right leg as well a number of others shown in Figure 28.

Royder[71] specifically mentioned that, "The flexible spinal mechanism allows the adjustment of the gravitational position of the head so that the eyes and the labyrinthine mechanism can remain level and stable". It follows that with left-sided loss of vertical dimension and concomitant cephalometric tilting that there is compensatory rotoscoliosis of the spine, cervical convexity to the right, thoracic convexity to the left and lumbar convexity to the right with a sacral base declination to the right. The muscle tightness and tenderness noted in the left cervicodorsal region are also consistent with the muscle imbalance patterns that are described by Greenman.[14] Royder also noted, as has been previously pointed out that, "Long-standing fascial strains, whether they come from above or below, soon become apparent throughout the entire body, and produce neural facilitation and somatic dysfunction. Therefore, malocclusion and mandibular dysfunction can be the result of somatic dysfunction resulting from structural imbalances in distant and seemingly unrelated parts of the body." He added, " Often TMJ pain and dysfunction can be traced back to sacral base declination through the fascial influences on cranial and mandibular function. Conversely, a torsion of the sphenobasilar symphysis will produce a torsion from the cranium caudad to the sacrum and on to the feet". Clinically, this author typically finds either sphenobasilar torsion or sidebending rotation cranial dysfunction associated with leg length discrepancy.

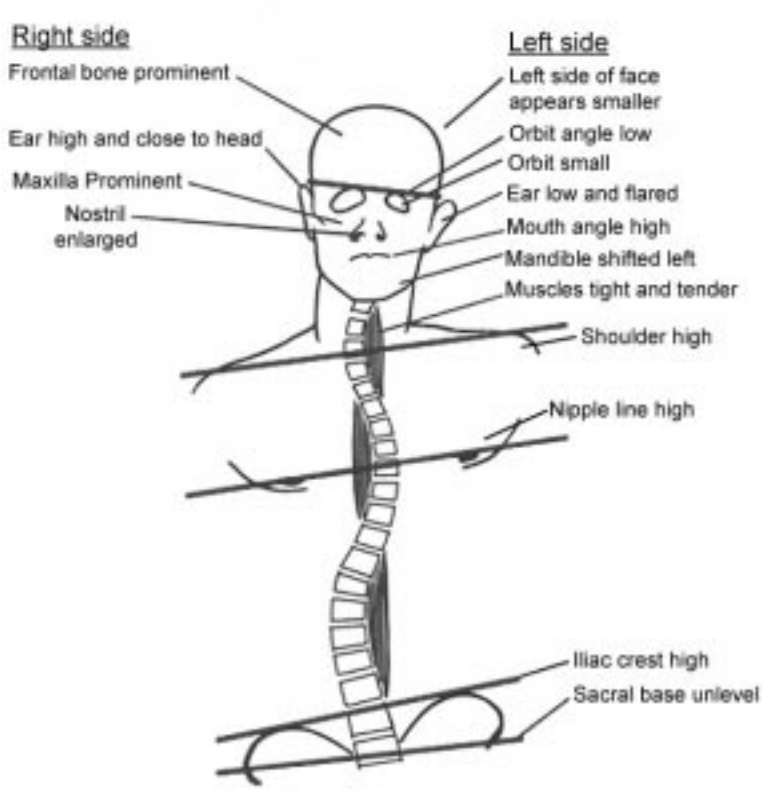

Figure 28. Short Right Leg Structural Findings. [Reprinted with Permission. Adapted from the *Journal of the American Osteopathic Association,* 80(7): 460-67, by James Royder: "Structural Influences in Temporomandibular Joint Pain and Dysfunction".]

Thus far we have examined Zink's circulatory / respiratory model, its origin and several biomechanical aspects of the postural model. Now let's look at specific relationships between these two models.

9. Relationships between the CCP and Posture

Regarding Zink's *compensated* patterns, there is evident agreement between the Common Compensatory Pattern and the common structural

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and functional asymmetries found in the postural model. Anecdotally, this author finds similar associations between the structural and functional findings of the short left leg and the Uncommon Compensatory Pattern. Zink [2] stated in the *ideal* pattern the patient presents with a level pelvis in both the horizontal and vertical planes and with equal leg lengths. In Kuchera's[89] description of Gravitational Strain Pathophysiology he said, "Gravitational force is constant and a greatly underestimated systemic stressor. Of the many signature manifestations of gravitational strain pathophysiology, the most prominent are altered postural alignment and recurrent somatic dysfunction." He went on to say that the signs and symptoms of gravitational strain pathophysiology "…often become apparent only after key host compensatory mechanisms are activated or overwhelmed. Zink's *uncompensated* patterns, associated with disease and lack of health, represent these patients whose ability to compensate has become overwhelmed.

It would seem that Zink's model and the postural model are fundamentally the same relationship seen from different perspectives. This hypothesis is the basis for a general postural model that is diagramed in part in Figure 29, with the complete model shown in Figure 31.

By substituting the specific term structural asymmetry found in the origin of the CCP relationship (Fig. 4), with the broader term *postural symmetry* you could derive a similar but more general relationship, the *origin of posture*. The reason for this substitution is that, as we have learned, human posture is not limited to structure. Clinical and experimental evidence suggests that developmental factors including third trimester fetal growth, birth trauma and cerebral lateralization can result in lifelong disturbances in structure and function of the human body. We have found that developmental influences acting on the human fetus along with its genetic potential come together to form a certain symmetry or asymmetry of structure and function in the adult. Postural symmetry is composed of three primary aspects. The first is symmetry of structure or anatomic mirror symmetry from right to left and vice versa. The second is symmetry of function, as in the phrase "symmetrical gait", used to describe equal use of the right and left sides of the body. The third is symmetry of mass, which is the attitude of the body from front-to-back and side-to-side. These three aspects of postural symmetry all under the influence of gravity directly relate to the concept of boundaries. Irvin[90] introduced the concept of boundaries by saying, "A tissue has the three qualities of structure, function, and conditions of boundary…" He further stated that, " the stability of the living system is a function of the boundaries within which proper structures perform, and is inversely proportional to the prevalence of accidents (somatic dysfunction and disease) that are consequent to suboptimal posture…" The words within parenthesis were added for context. The primary regions of postural asymmetry that were discussed in section 8 (Figure 20) are the same regions that determine the *boundaries of posture* and with this added perspective can also be related to human *function* and *structure*.

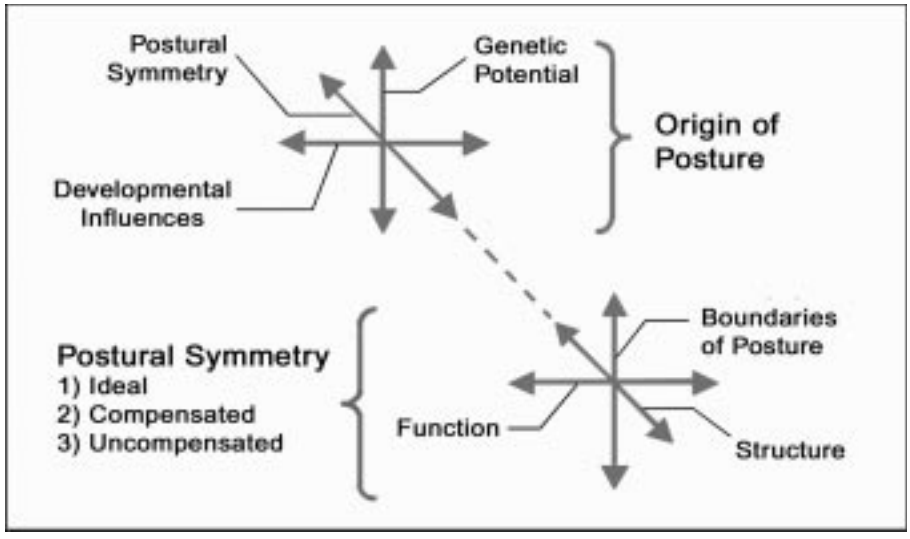

Figure 29. A General Postural Model (In Part)

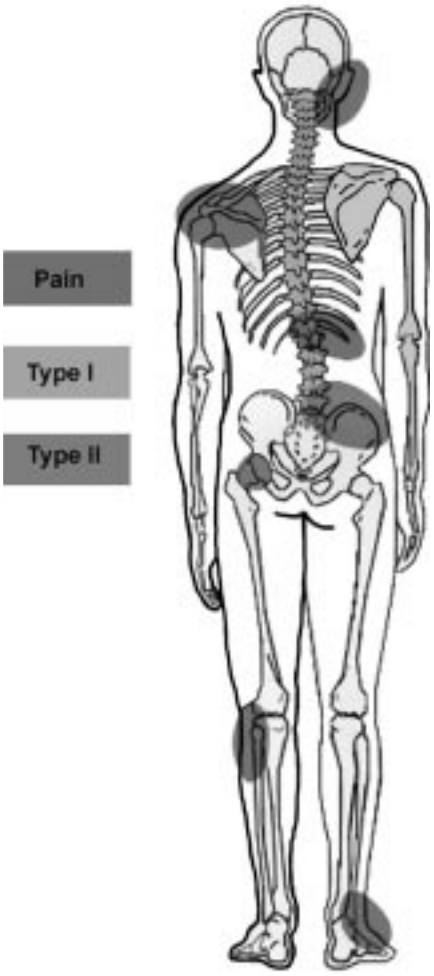

Figure 30. Common Pain Patterns

Having linked the origin of posture through the axis of postural symmetry to the thought that human structure and function are related through boundary conditions,[90] we can adopt Zink's nomenclature and characterized postural symmetry as ideal, compensated or uncompensated. These concepts organized in this manner allow for a general postural model. A model that takes into account the many varied aspects of posture and one that has a great deal of clinical utility.

10. Clinical Significance

Friberg[64] commented that the opposing torsional forces occurring at the L/S junction would cause significant stress to the numerous musculotendinous and ligamentous structures and result in inflammation and pain. Many clinicians[91-94] have noted that patients report pain accompanying these commonly found dysfunctions and postural asymmetries. Figure 30 illustrates some of the painful regions that are associated with a short right leg. In general, pain is reported at the junctional zones and associated with Type II mechanics. Foot and ankle pains are generally found on the right. Pain and osteoarthritis are frequently associated with the knee and hip of the long left leg. If shoulder pain is present, it is usually reported in the left shoulder. Additionally, if there is craniomandibular dysfunction and pain it is likely to be found on the right.[69] Ordinarily patients with postural asymmetry will describe their initial symptoms as recurrent. Then increasingly, the incidence of recurrence will become more frequent until finally their symptoms become persistent and their conditions then become subacute and chronic.

Treatment: In the approach to treatment of the patient with subacute and chronic pain of neuromyofascial-skeletal origin, clinical experience demonstrates that in general if the patient can achieve control in at least two of the three axes of postural symmetry then they will achieve compensation and cessation of painful symptoms.

Postural correction is used for treatment of the boundaries of posture. This includes the application of carefully crafted bite splints, foot orthotics, and heel lifts. For treatment of the functional axis you can prescribe specifically indicated strength, flexibility and neuromuscular re-education exercises. Finally, for the treatment of the structural axis we use osteopathic manipulative treatment (OMT). Nelson[95] stated that "the key to the entire relationship of posture to health lies in the entity of the osteopathic lesion, its production, maintenance and correction". He thought that postural imbalance produced and maintained somatic dysfunction and that its influence should be ruled out when considering treatment of any disease.

11. Conclusion

We have studied a number of the mechanisms thought to be responsible for the origin of the common compensatory pattern. Also based on a large body of theoretical, experimental and clinical evidence, we have described many relationships between the CCP and the Postural Model and discussed several factors that are common to both. There were several questions that were posed initially: Why do we see these same patterns over and over again? Is there a linkage between all of these commonly found clinical phenomena? What is the clinical significance of these patterns? We can answer these questions with the following simple conclusions.

- First with respect to their neurobiologic antecedents, Zink's fascial model and the postural model have the same genetic and developmental origins.
- Second that Zink's respiratory/circulatory model and the postural model are descriptions of the same phenomenon – human posture.
- Third that the two models can be combined to derive a general postural model.

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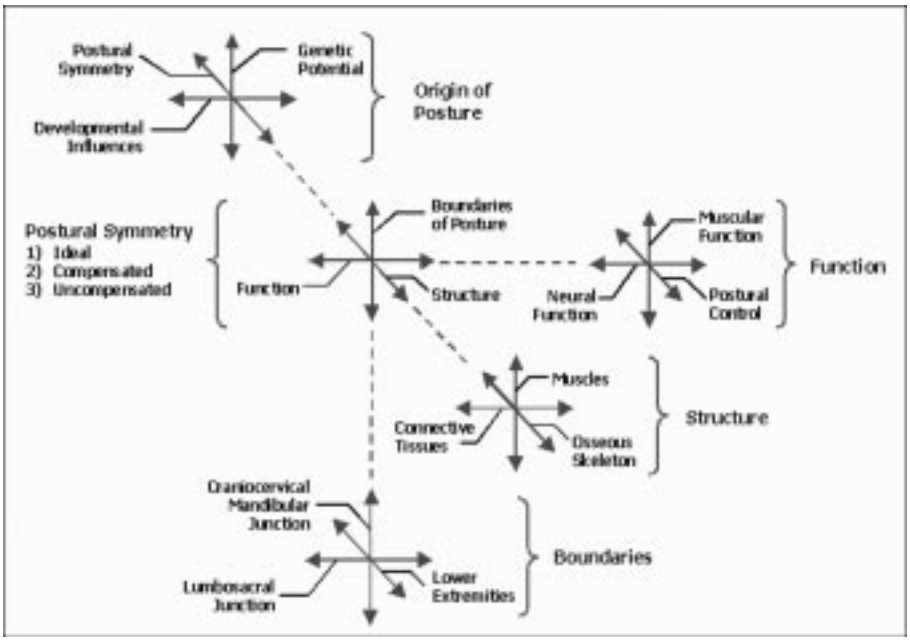

Figure 31. A General Postural

A general postural model (Figure 31) conceptually organizes what we know about commonly found structural and functional asymmetries.[96]

A General Postural Model: Clinical and experimental evidence suggests that genetic and developmental factors including third trimester fetal growth, birth trauma and cerebral lateralization can result in lifelong disturbances in structure and function of the human body. We find that these *developmental influences* on the human fetus along with its *genetic potential* come together to form a certain *symmetry* or asymmetry of structure and function in the adult. This can be abstractly represented in the familiar xyz-axes of the Cartesian coordinate system and are shown as such as the *Origin of Posture*. The most obvious structural asymmetries we see are the anatomic short right leg and the fascial bias throughout the body that was described by Dr. J. Gordon Zink as the common compensatory pattern.

There are also a number of commonly found functional patterns including recurrent patterns of somatic dysfunction and muscle imbalance. These well-known functional asymmetries are also related to motor dominance of the right hand and foot and postural dominance of the left leg.

Borrowing from Zink's work, we can characterize postural symmetry as ideal, compensated or uncompensated. The seminal thought that human structure and function are related through boundary conditions comes from Dr. Robert Irvin. [90] This general model recognizes three primary *boundaries of posture*: (1) the *craniocervical mandibular junction*, (2) the *lumbosacral junction* and (3) the *lower extremities*.

The interaction of these *boundaries* result in the commonly found pelvic types classified by Lloyd and Eimerbrink. [97] It should be noted that in this model the sacral base is not an independent variable. Rather, it is considered a part of the lumbosacral junction and its attitude is a resultant of the combined effects of the attitude of the craniocervical mandibular junction and the lower extremities. The latter including leg length, foot posture and to a lesser degree architecture of the foot arches.

If we expand along the axis of function in this model we can describe human function as an interrelationship between *neural* and *muscular function* and *postural control*. Similar treatment of the structural axis reveals a relationship between the support structures of the body. These include the *connective tissues* (composed of the fascias, ligaments, tendons and cartilages), the *muscles* and the *osseous skeleton*.

Finally a general postural model also allows us to conceptually link genetic and developmental factors to a number of commonly found clinical phenomena. The linkages within the model are summarized in Table 4.

Regarding the utility of a general postural model, Sir William Osler [71] once made the general statement, "In order to treat something, we must first learn to recognize it". Beyond that, Dr. Robert Kappler [55] specifically told us that, "Once the typical findings are defined and understood, then atypical postural balance patterns can be identified. If the patient has an atypical pattern, this alerts the physician to search for additional factors causing the patient's problem." Moreover a general postural model allows us to view human posture not as a simple static relationship between building blocks, one atop another, but as a lifelong interplay between genetics, development and postural symmetry.

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TABLE 4. CAUSAL LINKAGES IN A GENERAL POSTURAL MODEL

Factors	Linkage	Common Postural Findings
Genetic Factors	Cerebral Lateralization	Results in left cerebral dominance with right-sided motor dominance.
Prenatal Factors	Left Cephalic Fetal Lie Vestibular Lateralization	Results in a fascial bias that is consistent with the CCP. Resulting in left vestibular dominance left-sided extensor muscle dominance.
Birth Factors	Birth Trauma & Cranial Asymmetry	Results in the commonly found cranial assymmetries in infants that could in turn cause or reinforce the CCP in the adult.
Postnatal Factors	Growth and Development The cumulative effects of postural control, right sided motor dominance and left sided postural dominance.	Results in the long left leg and sacral base declination to the right with occipital tilting to the right. Also results in Gravitational Strain Pathology that includes recurrent somatic dysfunctions and muscle imbalances.

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