3

Components of the Vertebral Subluxation Complex

The word "subluxation" may have been the term of choice for the better part of 100 years, but, as discussed in the previous section, it doesn't really distinguish itself from other uses of the same term. Hence, the term Vertebral Subluxation Complex (VSC) has come into favor in more recent years.

The updated name easily implies a more complicated situation than a simple partial dislocation. While the partial dislocation may do an adequate job describing the orientation of the articular surfaces, the basis of chiropractic is seated in the effect the loss of articulation has on the surrounding tissues, particularly the neurological receptors.

Initially, the VSC had been broken down into components and popularized independently by Joseph Flesia[1] and L. John Faye[2] in 1982 and 1983 respectively. The original model had five components:

1. Kinesiopathology
2. Neuropathology
3. Myopathology
4. Histopathology
5. Pathophysiology

The components of the VSC obviously describe unique facets of the complex, but each of the components are not equally detrimental, some are reversible whereas others are not, and not all components become apparent at the same time.

1. Kinesiopathology

Based on a breakdown of the term alone, kinesiopathology refers to the biomechanical condition of the vertebrae: basically, any and all alterations in normal movement, including excessive motions beyond normal range of motion and deficits in what would be expected to be normal. Chronologically, the kinesiopathology will be the first component to manifest. All of the others are a consequence of it and without the kinesiopathology component, there would be no means of chiropractic intervention.

There are five aspects or variants of the kinesiopathology component.

i. *Hypomobility*—Segmental blockade/fixation and abnormal restriction of joint. During hypomobility, certain vectors of motion may be maintained as normal, while one, more, or all may show deficits. It's considered a general loss of movement in at least one range of motion.

Hypomobility of an individual segment is most likely to be reflective of a loss of overall motion. That is, sedentary periods are more likely to cause segmental hypomobility over time.

It is the kinesiopathology component that makes the VSC a chiropractic condition; this statement pertains to any hypomobility present.

Hypomobility is the component upon which all other components are based. Without hypomobility, any cascade of events is not considered a chiropractic condition.

ii. *Hypermobility*—As the prefix suggests, hypermobility indicates an increase in joint motion. In a similar fashion to hypomobility, hypermobility indicates that a vertebral segment has one or more of its ranges of motion in excess of its normal values.

It's important to keep in mind that both the hypomobility and hypermobility variants of kinesiopathology can coexist in the same vertebral segment. That is, a particular vertebrae could be hypomobile in right rotation, but hypermobile in left rotation. This is one of the reasons for the importance of adjustment specificity. Identifying the segment to be adjusted is not enough, as the individual three dimensional orientation and asymmetries of motion makes the vector of force applied during the adjustment as important as the identification of the vertebrae itself.

Hypermobility may be the consequence of a significant traumatic situation. Severe whiplash can and will cause significant strain on the muscle and ligamentous structures of the vertebrae, resulting in a loss of supportive integrity. It should be noted that pure hypermobility is not a chiropractic condition, as it lacks the prerequisite *loss* of vertebral motion.

iii. *Compensation reaction*—There is a phenomenon that can be observed when looking at changes in biomechanics of the human frame. Any alteration or aberrant function sets off a cascade of changes in order to compensate and minimize the overall effect of the original change. Changes in function of one joint leads to an opposing change in adjacent segments.

This can be seen on a grand scale in individuals who have a loss of function at the sacroiliac or iliofemoral joint on one side of the body. During ambulation, the contralateral arm tends to swing at a greater amplitude than the ipsilateral arm. In this instance, an inability to smoothly and effectively flex the hip while walking will limit the distance of the foot travelled prior to the heel strike phase of gait. To minimize this loss, the contralateral arm will swing at a greater degree and force in order to create the leverage needed to help the hip perform closer to normal.

On a more focal scale, it's common to find a hypomobile vertebrae create a hypermobile vertebrae adjacent to it, and vice versa. Nowhere is this seen to such an extent as the effects of a surgically fixated vertebrae on the adjacent spinal segments above and/or below the fixation.

Long term hypomobility as part of the VSC causes the joint above (usually) and the joint below to become hypermobile—i.e., function is adapted rather than lost across multiple joints.

Compensatory changes tend to be a domino effect throughout the spine and the rest of the skeletal structures, with structures being close to the original aberrant function being affected initially, then the longer the altered function mechanics is present, the wider spread the compensatory changes will become, in various patterns of hyper and hypo mobility. In this fashion, throughout the spine, some of these mechanical changes will be true vertebral subluxation complexes while others will be compensatory changes, secondary to the VSC.

iv. *Loss of joint play*—The loss of normal joint play involves hypomobility on the vertical plane.

This variant adds some of the directionality that was implied during the hypomobility variant section. As the facet joints glide over one another during flexion/extension, a fixation in the joint at various points could create a posteriority or anteriority, depending if the fixation occurs more in extension or flexion respectively.

v. *Loss of rotational motion*—The loss of normal play so that the joint becomes hypomobile with respect to the central axis of motion.

The remainder of the implied directionality from the hypomobility variant comes from the loss of central axis of motion. Loss of movement in this plane adds the rotational component to the VSC—that is, as per the Gonstead nomenclature, a spinous right or spinous left malposition.

At this point, we have addressed the anterior/posterior and both rotational aspects of VSC orientation, but have not mentioned the laterality aspect. The laterality, in this model is attributed to a combination of the asymmetries seen in the loss of joint play during flexion/extension *and* loss of rotation.

The coupled motion observed at adjacent vertebral segments will dictate that any rotational movement is accompanied with a lateral movement, creating a wedging of the IVD space. In the cervical spine for example, movement of the spinous process to the left is going to be accompanied by lateral flexion of the same vertebrae to the right.

vi. *Positional dyskinesia*—Joint misalignment throughout the entire range of motion of the involved joint.

Dyskinesia involves a fixation within the normal range of motion of a spinal segment that leads to an adaptation of the proprioceptors in the joints and soft tissue associated with that segment. Because of limitations in the range of motion at the segment, there is a loss of stimulation in the phasic receptors that are specialized for detecting kinetic movement.

Therefore, the loss of proprioception from the fixated spinal segment leads to a direct loss in the body's awareness in positioning, increasing altered mechanics at adjacent segments and the direct link to the neuropathology component of the VSC.

Positional dyskinesia can and does occur outside the normal, physiologic range of motion. In these instances, the dyskinesia is associated with varying degrees of integrity loss of periarticular tissues such as the IVD, articular capsule, and/or paravertebral ligaments. This loss of integrity can vary from mild degenerative, physiologic changes to complete failure.

It cannot be overstated that it is the hypomobile aspect of the kinesiological component that chiropractic addresses. Whether it is the primary aspect of the altered biomechanics or a compensation to a hypermobile segment adjacent to it, the chiropractic adjustment targets a hypomobile segment.

Each of the subsequent components of the VSC is the direct or indirect consequence of the kinesiopathology component (Fig. 3.1). All of the effects on the surrounding and/or related tissues are rooted in the ultimate loss of vertebral motion. Without segmental movement, muscles atrophy, sensory feedback is lost from the immobilized vertebrae, and the loss of mechanics is compensated by adjacent joints, resulting in increased physical strain and accelerated degeneration.

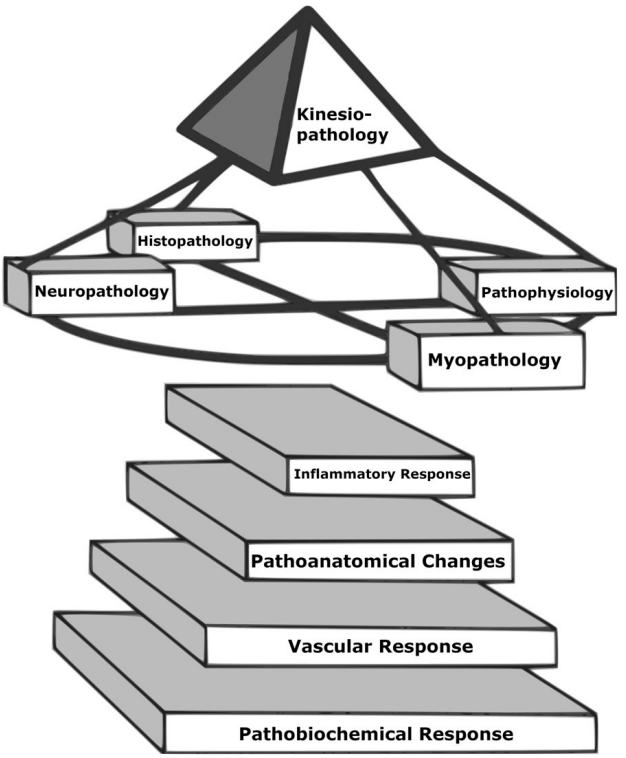

Figure 3.1—Hierarchy of Vertebral Subluxation Components. While any of the above components can exist in the human body under different circumstances, from the chiropractic context, the kinesiopathology component leads to the neuropathology, myopathology, vascular pathology, and connective tissue pathology directly. The remaining components are considered to be more long term complications. Make particular note that as far as chiropractic is concerned, the beginning of the cascade is the kinesiopathology.

2. Neuropathology

This component of the VSC will be the major topic for the majority of this text; the description immediately to follow is in a more general sense than the sections to come.

Very quickly following the loss of vertebral movement, there will be a loss in sensory feedback from the tissues associated with that segment.

Once the joint complex has a reduction of movement, the small, stabilizing musculature undergoes a dramatic reduction in contraction, relaxation, and stretch. Muscle spindles can no longer provide sensory changes from muscles that span a single spinal joint while Pacinian corpuscles and Ruffini endings sensing joint position is no longer able to sense changes in said position.

While the loss of activity at the receptor level of the nervous system may be the immediate and most directly related neurological consequence of joint fixation, the more intriguing neuropathological changes will be the effects that this loss of afferent activity causes in the central nervous system.

The loss of sensory input results in facilitation at the dorsal horn of the spinal cord, increased activity at the reticular formation, heightened sympathetic tone, and altered motor function.

The VSC neurology is discussed as a facilitative lesion rather than a compressive or chemical one. The concept of a compressive force (as seen with a protruding IVD) being a contributor to the neurological aspect of the VSC is often misplaced. The compressive load on any neurological tissue is a subsequent result of the initial VSC and while any neurological changes observed are clinically important, they are the result of loss of structural integrity of the tissues surrounding the delicate neuroanatomy, not part of the neurological bases of the VSC. Likewise, a chemical lesion with respect to the VSC arises secondary to inflammatory/chemical changes that arise with local tissue changes.

The impact on the spinal nerve secondary to a compressive or chemical load

is really a consequence of the degenerative, histopathology changes to be discussed later. At the point in time when these components are present to the degree with which to result in physical or chemical stress on the adjacent neural tissue, the facilitative lesion has already since been long established.

3. Myopathology

The consequences of the VSC on the muscle tissue occurs very shortly after the onset of the kinesiopathology component and involves the weakening, atrophy, increased tonicity, and/or alterations in neuromuscular habit patterns of the muscle tissue associated with the fixated spinal segment. This quick response to the effects of the VSC comes as no surprise, considering the close relationship between the vertebral articulations and the muscles that are meant to move and stabilize them. This relationship is so close that injury to muscle can be a primary cause of the VSC itself. That is, fixated joints impact muscle function and vice versa, altered muscle function can result in joint fixation.

The first of such muscle tissue changes will be in tonicity. Loss of joint movement results in a decrease in muscle spindle activity, with in turn, an increase in muscle tone. (See ch. 19—Motor System.)

Muscle tissue is an adaptive tissue, in the sense that it responds to physical stress by growing and strengthening in order to better handle it. Likewise, a lack of physical demand on the tissue has a stark and opposite effect. Disuse atrophy and weakening is seen in any muscle that has decreased use. With respect to the spine, we are able to see disuse atrophy in the more superficial musculature such as the erector spinae, iliocostalis lumborum and longissimus thoracis, but this has been at least partially attributed to individuals with associated pain as a limiting factor and as a result, increased sedentation results in muscle weakening. The superficial muscles span several joint structures, and loss of functional mechanics at one part of the body is generally compensated by altered mechanics at other areas. This compensation reduces the effect on superficial musculature, as demand on the muscle can still be applied through grossly maintained active range of motion. Deeper spinal musculature, however, does not get this pass. Muscles such as the multifidius, intertransversii, interspinalis, and rotators only span 1–2 joint complexes in a rather short span. Immobilization will affect the muscles associated with the joint more if these muscles only span that particular joint—i.e., as seen in the gastrocnemius of a fractured leg that has been casted. Likewise, atrophy and weakening will be seen in muscles that span only a single, immobilized joint as seen in the VSC. With the ratio of muscle to joint being much, much lower than the superficial musculature, any immobilization of the joint has a more dramatic effect on the weakening and atrophy of those muscles. To make matters worse, each of these muscle groups plays a role as the body goes through the general ranges of motion and activities of daily living. Superficial muscles tend to be stronger and take the spine through larger ranges of motion and aiding the body through activities such as lifting heavier objects. Deeper muscles, however, take on the role of stability of the spine as the body goes through these activities. Lack of stability as the deeper muscles weaken increases the likelihood of sprain/strain injuries and episodic spinal pain syndromes with even mild/moderate modes of onset.

Altered muscle activity on one side of the spine in comparison to the other as a result of the VSC can be seen clinically by way of certain types of instrumentation.

Increased muscle activity undergoes increased metabolism which dissipates heat to the surface of the skin as a result. These changes in surface temperature on opposite sides of the spine can aid in the identification of the VSC.

4. Histopathology

Histopathology concerns the physical and chemical changes seen in the tissues associated to the VSC. In 1989, Charles Lantz[3] sought to expand the five-component VSC model into a nine-component model. The main area for expansion came to the original histopathology component. Lantz examined the most common tissues involved and the response the tissues have in the presence of the VSC.

The subdivisions of this component include:

I. Connective Tissue
II. Vascular
III. Inflammatory
IV. Anatomic

I. Connective Tissue

The connective tissues most in question are the hyaline cartilage surfaces and intra-articular meniscoid tissue of the facet joint, and the fibrocartilage of the intervertebral disc.

With respect to the connective tissues involved, there is a balance that must be met in order to maintain optimal tissue health. Accelerated degenerative changes can be seen in joints that are experiencing excessive physical stress, but unfortunately there is also an increase in the rate of degeneration seen in immobilized joints. Because there is no direct blood flow, IVD's require a certain degree of mechanical stimulation to maintain normal health via osmosis. Intersegmental immobilization reduces this necessary movement while at the same time results in compensatory hypermobility at adjacent segments.

This is the component that would be most accurately represent factors of the compressive lesion model of the VSC, as IVD degeneration, spinal stenosis, and ligamentum flavum buckling physically impact nerve roots.

II. Vascular

Blood flow to and around the joint structures becomes altered in the presence of the VSC. In fact, A.T. Still hypothesized that the most significant detrimental effect of an immobilized vertebrae was the changes it had on vascularity. Injured areas will have an increase in blood flow (as part of the inflammatory process to be discussed momentarily). In the absence of acute injury, altered muscle tone causes ischemic compression of blood vessels. Altered blood flow makes it difficult for tissues to maintain homeostasis as nutrients and oxygen have a more difficult time getting to the tissues while carbon dioxide and other waste products tend to show elevated levels. It is this ischemic compression that contributes to tender trigger points commonly found in tight muscles.

III. Inflammatory

The inflammatory process has three basic phases. An inflammatory response, a repair/regeneration phase, and remodeling/maturation phase.

During the inflammatory response, increased blood flow to the injured area transports cells to initiate the healing process. Any damaged cells are removed and new collagen is produced. This phase is usually accompanied by swelling, pain, and increased tissue temperature.