

This chapter explores the nature of the various aspects of the vertebral subluxation complex (VSC). It is beyond the scope of this writing to describe in detail all the possible entities included in the broad category of the VSC. The purpose of the material presented is to focus on selected aspects of the VSC relevant to chiropractic care.

Several distinct types of physical changes that occur in relation to the VSC have been described. These changes include those affecting kinesiologic, histologic, neurologic, myologic, biochemical, vascular, inflammatory, and connective tissue characteristics (1,2). This chapter will pay particular attention to the following clinical manifestations resulting from the physical changes listed above:

1. The relative positions of the vertebrae above and below an articulation involved in subluxation;
2. Intervertebral motion abnormalities in any or all of the six degrees of freedom of the motion segment; and
3. Neurophysiologic involvement caused by interarticular abnormalities.

Clinical presentation of a complex disorder can lead to treatment approaches that vary considerably, even within the same health field. One of the reasons for this variation results from approaching these problems from an isolated perspective of assessment. A multiparameter approach is requisite to provide for a working system of analysis and correction appropriate for each individual case presentation. One of the most important fundamentals of systematic full spine treatment is the recognition of the multiparameter nature of the VSC. The chiropractor should use examination procedures that are both sensitive and specific to all of the parameters of the VSC. Analysis procedures for the VSC will be covered in Chapters 4 and 5.

A complete understanding of normal anatomy and physiology of the structures involved is necessary to fully appreciate the clinical approach to the VSC. The subluxation is essentially an interarticular phenomenon (3). The reader is encouraged to pursue an in-depth study of normal articular structure and function and the effects of injury on the motion segment. Literature on the subject of spinal related conditions is extensive. Most research on spinal injuries has been performed on the lumbar spine. Many references for this chapter are taken from lumbar studies and may be cautiously extrapolated to other spinal regions.

POSITIONAL DYSKINESIA

Chiropractors have considered the interarticular alignment of spinal structures an important aspect of the VSC ever since the first chiropractic adjustment was given (4). The understanding of positional dyskinesia (misalignment of one vertebra on another) has evolved considerably since the original chiropractic “bone out of place” theory was formed.

The importance of the identification of positional dyskinesia is easily illustrated by the development of radiographic analysis of spinal structures. Widely taught at chiropractic colleges are literally dozens of methods of radiographic analysis (3). Positional dyskinesia is a factor in the etiology of neurophysiologic disorders, especially in the cervical and lumbar areas (5–11).

Etiology

Causes of positional dyskinesia have been postulated to involve the most basic circumstances of life, such as posture, the influence of gravity and cerebral dominance. The apparently high incidence of spinal subluxation of our species, in relation to other animals may be associated with the evolutionary theory of development from the quadruped to the upright stance (3). The spine is also under constant influence of cerebral dominance. The upper thoracic spine, for example, has the tendency for a lateral deviation with the convexity towards the side of the dominant hand (12).

Clinical Considerations

Positional dyskinesia is an important aspect of the subluxation. Malalignment of contiguous vertebral structures that support weight and guide movement, alters the ability of the involved functional spinal unit (FSU) to continue normal function. It is questionable, however, that positional dyskinesia by itself can cause direct neurophysiologic dysfunction. The association of positional dyskinesia with the degeneration of soft tissues and disruption of normal mechanics has been reported (13–15).

Mechanisms of Injury

It is important to recognize that positional dyskinesia can occur in any direction along the planes of possible move-
ment, and in any combination of directions, as are applicable to the articulation in question. The nature of positional dyskinesia varies with the shape and function of each articulation, and from region to region in the spine.

The mechanisms by which any given positional dyskinesia of the spine occurs is not always clear. The existence of positional abnormalities in the spine can best be explained by a discussion pertaining to interarticular soft tissue disruption.

Two probable mechanisms of positional dyskinesia of the spine will be presented. One mechanism involves a sudden application of a damaging force. The other is a mechanism occurring more gradually over a period of time. These mechanisms are put forth as probable but not as the only possible causes of positional dyskinesia.

MICROTRAUMA

The second suggested mechanism of injury involves the spine's reaction to long-term subthreshold (not causing irritant damage) forces. The forces of gravity on posture in combination with weakness or unbalanced contraction of postural muscles, inevitably take their toll on spinal structure (Fig. 3.2).

MACROTRAUMA

Sudden forces that overcome the strength of the paraspinal soft tissue can cause immediate damage to the joint capsules, disc, and ligaments that support a vertebral articulation (16). The nature of the damaging force, whether torsional, compressive, tensile, shearing, or a combination thereof, is the principle factor determining exactly which soft or hard tissue elements will be affected most (Fig. 3.1).

Interarticular injuries can damage any or all of its components. The main effects of injury are to the intervertebral disc, the two zygapophyseal joints, as well as the interspinous area. The reactions that occur in the spine in response to the different injury vectors have all been well described by various authors (17–19).

Figure 3.1. A, A common mechanism of sudden macrotrauma to the lumbar spine with resultant injury to the annulus and facet capsule depicted in (B).

Figure 3.2. Chronic asymmetrical loading of the spine resulting in scoliosis in a mail carrier.
The intervertebral joint loses stiffness after injury and the creep characteristics of the joint are changed. Creep is the gradual deformation of the intervertebral joint (ligamentous structures) under a constant load. Creep deformation occurs most in the direction of the injury, thus with a fractured end-plate the disc creeps to a reduced thickness. With torsional (Y axis) failure the joint tends to creep into the rotated position of injury (18).

Another example of the effects of long-term physical stress is seen as a compensation reaction. Anatomically normal FSUs are subjected to increasing amounts of stress during routine motions by compensating for other areas in the spine which are relatively restricted in their ability to move in one or more planes of normal motion (20). Intervertebral misalignment may well be the result of the injured spine's specific reaction to singular or accumulative traumatic forces.

**Retrolisthesis**

Retrolisthesis positional dyskinesia occurs most obviously in the normally lordotic cervical and lumbar areas and is best seen on the lateral spinal radiograph. Retrolisthesis is often accompanied by segmental hyperextension (−θX). This type of positional dyskinesia is capable of narrowing the spinal canal (21) and alters the weight bearing status of the FSU. This weight-bearing change sets the stage for compensatory weight bearing changes that can affect the entire spine, especially above the retrolisthesis. On the lateral spinal radiograph the shape of the disc space, the posterior edges of the vertebral bodies, as well as the interspinous spaces, can reveal the presence of −Z and −θX positional dyskinesia. Shifts in weight bearing with resultant changes in spinal curves as seen on x-ray can aid the clinician in determining which subluxated areas of the spine are affecting the overall structure. Subluxations that appear to significantly affect the functional, and postural status of entire regions of the spine are termed primary subluxations, and are of major clinical importance.

Retrolisthesis in the thoracic spine is usually less apparent and is commonly accompanied by flexion (+θX) misalignment. The theoretical importance of retrolisthesis in this area evolved from clinical observations of patient improvement after posterior to anterior adjudistic techniques.

Evidence of abnormal weight bearing by the facets is often seen on the lateral spinal radiograph, accompanied by −Z translation (retrolisthesis) of one vertebra on the segment below. This results in the degenerative changes (sclerosis) seen in the facet articular surfaces. Persistent −Z translation, or any other type of displacement, stretches the facet capsular ligaments, and would not occur without significant failure and destruction of intervertebral disc substance (Fig. 3.3). The classic "facet syndrome," and/or instability, is likely secondary to disc abnormalities (13). The management of patients diagnosed with what some practitioners consider facet syndrome must, therefore, include attempts to restore normal intervertebral disc mechanics (as opposed to symptom relief only) if correction is to be attained. After clinical observations, Gonstead theorized that the most significant direction of positional dyskinesia of a vertebrae from C2 to L5 is retrolisthesis (−Z) (22).

**Clinical Considerations**

The structural integrity of the spinal canal and lateral recesses are altered by positional dyskinesia. Encroachment on the neural and other soft tissue structures within

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**Figure 3.3A-B.** Retrolisthesis of L5 vertebra on S1, secondary to annular disruption.
the canal may lead to adverse mechanical tension on these structures. Stenosis secondary to persistent malalignment has been reported (5).

It is interesting to note that many manipulative procedures employ primarily rotational (Y axis) forces as a major component of the maneuver. These forces not only have the most potential for harm to the annulus and the posterior joints but are also ineffective in reducing retrosition or extension positional dyskinesia.

**Primum Non Nocere**

One of the foremost postulates of any health care practitioner, is to “above all do no harm.” Knowledge of the relative positions of vertebrae is necessary, to avoid the potential for further distortion of the neural elements and articular ligaments, cartilage, and disc during adjustments.

For example, if the L4 vertebra is rotated $\pm Y$ (spinous right) on L5 and an adjustment is performed at L4 in the $\pm Y$ direction, further torsional injury may result. One of the most common malpractice actions against chiropractors is related to incidents involving excessive axial rotation to the lumbar spine in the side posture position. To lessen the likelihood of malpractice litigation, one should consider the alignment characteristics of the involved FSU and apply manual forces in the opposite direction of the positional dyskinesia.

Positional information is of little use if one has no knowledge of the dynamics of the articulation. **No adjustment (i.e., grade 5 mobilization*) should be administered to an apparent positional dyskinesia without concomitant evidence of a relative decrease in mobility at that articulation.** It is well known that misaligned vertebrae may be freely movable (Fig. 3.4). These freely movable misalignments must be distinguished from articulations that exhibit a relatively fixed position when x-rayed in different postures (23). Fixation dysfunction is a dynamic component of the structural and functional characteristics of spinal subluxations. The knowledge of the presence of fixation dysfunction in combination with that of the relative positions of vertebrae surrounding the sublaxed articulation, is a prerequisite to the administration of the adjustment.

In summary, the chiropractor should evaluate positional dyskinesia by applying the same engineering principles that govern all structures under the effects of gravity. These principles must then be combined with the overall posture and the history of trauma of the patient, to properly establish the likely causes and clinical relevance of positional dyskinesia.

**FIXATION DYSFUNCTION**

Dysfunction, in strict definition, simply means ill operation of the described entity (24). Applied to the functional spinal unit, the term dysfunction implies that abnormal motion characteristics are present.

Abnormal structure, such as anomaly, positional dyskinesia, etc., can lead to dysfunction, and dysfunction can lead to abnormal structure, such as articular degeneration. The motion coupling of the lumbar spine, for example, is dependant on the lordotic posture of that area (See Chapter 7).

Fixation is a term describing a specific type of dysfunction that is applied to a FSU that is restricted in any or all of its six degrees of freedom.

Besides the limitation of the intersegmental range of motion, fixation dysfunction can be manifested by alterations in the instantaneous axis of rotation (25). A restriction in one direction of the articulation may be accompanied by an increased range of motion in the opposite direction.

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*Grade I-IV mobilization descriptions:
Grade I: Small amplitude movements performed at the beginning of the range
Grade II: Large amplitude movements that do not reach the limit of the range
Grade III: Large amplitude movements performed up to the limit of the range
Grade IV: Large amplitude movements performed at the limit of the range
Grade V: Movement into the paraphysiologic range
Clinical Considerations

Fixation dysfunction is an important aspect of vertebral subluxation and it must be analyzed, corrected, and managed, along with the alignment and neurophysiologic aspects of the VSC. Prolonged fixation can cause abnormal somato-autonomic and somato-somatic reflexes (26), compensatory hypermobility at other spinal articulations (20), and spinal articular degeneration (27).

The importance of the intervertebral disc to spinal function has been discussed in an earlier section. One major mechanism involved in the maintenance of homeostasis of the disc is the process of imbibition. The physical properties of the intervertebral disc have been conceived as depending mainly on the water binding capacity of the nuclear pulp. The hydration of the nucleus is predominantly due to the imbibition pressure exerted by its mucopolysaccharide gel (17). The influx of nutrition, and the effusion of waste products from the disc, relies on imbibition due to the disc's inherent lack of blood supply (12). Prolonged fixation can be a cause of degenerative change of the disc by interfering with its ability to maintain itself through imbibition (Fig. 3.5) (19). The lack of motion impedes the flow of fluids through the intervertebral articulations.

Interarticular movement is necessary for the prevention of contracture and adhesion formation, as well as for the proper orientation of collagen fibers. Extracellular water loss, glycosaminoglycan depletion, and collagen cross linking accompany persistent immobility. The above response is uniform throughout ligaments, capsules, tendons, and fascia (27).

Compensatory Hypermobility and Instability

Hypermobility dysfunction is seen when any particular range of motion of the articulation(s) has increased beyond what is considered normal for articulations within the same region of the spine (28). It is not uncommon for hypermobile segments to be the cause of symptomatology in an individual (28).

It has been said that for every subluxation, there is compensation (22). Hypermobile articulations have been considered as a compensatory phenomenon, secondary to the presence of fixation dysfunction elsewhere in the spine (usually at adjacent or nearby segments) (20). Compensatory hypermobility above the level of surgical fusion is well known (29). The explanation given for the compensation has been that when an individual attempts to move the spinal region near the fixation dysfunction, other articulations nearby are forced to move through a greater range of motion. The total spine will attempt to maintain normal global or end-range of motion, sometimes sacrificing the integrity of an individual spinal unit.

It has been reasoned that the stress of compensatory hypermobility and abnormal weight bearing results in the breakdown of the interarticular soft tissues. This degeneration is commonly seen on the lateral radiograph of the cervical spine in the form of traction osteophytes at spinal levels above the area of subluxation, usually between the C4 and C6 FSUs.

Case studies have described adjustments applied to areas of hypomobility that have resulted in increased movement of previously restricted articulations (30–33). Jirout (30) describes compensatory hypermobility reduction from adjusting nearby levels involved in fixation dysfunction.

We saw earlier in the chapter that persistent positional dyskinesia in one area can result in compensatory alignment changes elsewhere. Here we have presented the compensation mechanisms involving motion characteristics. It can be seen that specific adjustments, applied to reduce positional dyskinesia and improve mobility, have biomechanical effects that extend to spinal regions beyond those directly treated.

Etiology

Much is to be learned about the factors involved in restricted vertebral mobility. A literature review by Rahlman (34) describes fixation dysfunction as, “acute joint fixation, locking, binding, blocking” which includes localized muscle spasm and can be ameliorated immediately after a manipulation.

Theories of fixation dysfunction must encompass not only those mechanisms involved in acute, but also chronic fixation. It is common for the VSC to occur and progress insidiously. Many acute presentations, therefore, are actually an acute stage of a chronic condition.

It is likely that most occurrences of articular fixation are multifactorial. Possible mechanisms of fixation include muscle spasm, meniscoid entrapment, articular adhesions, edema, and disc derangement. Contracture of the ligaments, muscles, and tendons may also resist intersegmental motion. Abnormalities in articular structure, especially in the facets, may affect joint function and be mistaken for fixation dysfunction (See Chapter 7).
MENISCIOIDS

An entrapped meniscus-like body in the apophyseal joints called a meniscoid is an attractive theory behind vertebral fixation. A meniscoid is attached at its base to the articular capsule of the zygapophyseal joint with a free end that invaginates into the articulation (35). Meniscoids have been found in all regions of the spine. Meniscoid entrapment may not only lead to the restriction of motion, but also to pain and muscle spasm secondary to joint capsule traction.

Further investigation by Bogduk and Engel (36) into the structure and the arrangement of meniscoids reveals that they are a weak combination of fibrous connective and adipose tissue. The type of strong tissue necessary to cause joint locking and generate tension in the joint capsule was not observed in their own work or in the studies they cited. Several other investigators have studied meniscoids and their conclusions are variable as to the significance of meniscoid entrapment relating to joint lock.

Meniscoids may contribute to some part of joint derangement in specific cases, especially in the cervical spine where the facets are large relative to the three joint complex. Capsular proliferation secondary to degeneration of a large facet joint may result in a larger meniscoid. Derangement of a cervical facet may hypothetically have a greater influence on the function of the three joint complex than in the thoracic or lumbar spine.

Some cases may begin with a pinching pain that initiates a cycle of events and produces restricted intervertebral range of motion accompanied by painful and spasmed musculature. The degenerative changes associated with meniscoid entrapment may be similar to that of the detached cartilage of knee joint derangement. Although meriting further research, the meniscoid theory of joint fixation does not appear to be a mechanism that is a major contributor to fixation dysfunction found at primary sites of the VSC.

MUSCLE SPASM

Muscle spasm is known to reduce spinal mobility. The possible mechanisms eliciting muscle spasm are many and varied. Reflex mechanisms involving muscle are never unisegmental; that is to say that they are relayed to segments above and below the level of the direct initiation of the neurologic impulse (37). Muscle spasm often exists at multiple spinal levels. This phenomenon is often elucidated on stress radiography by the presence of restricted mobility of a section of the spine, which may consist of a few to many vertebral articulations. This type of grouped hypomobility is different from the unilevel intersegmental fixation that the chiropractor must look for in determining specifically which articulation is to be adjusted.

Muscle spasm is not likely the chief cause of most cases of intersegmental hypomobility (35). In one experiment, evidence of fixation dysfunction existed even after patients were completely under anesthesia, which included myorelaxants. Movement restriction was even more recognizable during narcosis, as the patients were totally relaxed (38).

Muscle spasm has been implicated as one possible reason for fixation dysfunction. The reflex mechanism involved in muscle spasm may be interrupted by the adjustment, leading to an instantaneous restoration of motion. We must look further, however, to uncover the mechanisms that are primarily responsible for intersegmental fixation dysfunction.

ADHESIONS

Disorganized fibrous cross-linking (scarring) between parallel collagen fibers is termed adhesion formation (27). Adhesions may form in ligaments, cartilage, muscles, tendons, or fascia. Articular adhesions are the by-product of the process of degeneration, and may result from trauma or immobilization (35). Ligamentous and cartilaginous tissue disruption involves healing mechanisms that replace normal tissue with fibrotic tissues of a lesser grade (17). Degeneration often begins at an early age and may result from persistent malalignment and fixation. The acute case presentation usually involves an acute stage of a chronic underlying condition. It is common that considerable disruption of supportive spinal soft tissue exists by the time most patients seek care for their condition.

Articular adhesions are likely to be part of the manifestation of intersegmental fixation dysfunction, especially as degenerative changes progress. The breaking up of adhesions from a spinal adjustment could account for some of the reason that an instantaneous change in intersegmental motion may occur after an adjustment is given. In cases of post adjustment pain, the cause may be inflammation in reaction to tissue damage from the tearing of adhesions.

The retraction of adhesions may progressively restrict interarticular movement. This process occurs over a relatively long period, thus necessitating recurrent adjunctive interventions. Adhesion formation and gross soft tissue disorganization are the likely reasons that prolonged supportive chiropractic care may be necessary for patients with chronic VSC.

INFLAMMATION

The effects of inflammation on restricting mobility of an articulation is exemplified by the painful restriction of edematous joints. In the acute stage, intersegmental motion may be inhibited by the presence of inflammatory fluids in the joint space (Fig. 3.6).

Movement of fluids away from the articulation, when indicated during the acute stage of injury, can be accomplished by “pumping” the articulation in the direction of
relatively large interarticular surface area occupied by the disc, as well as the fact that the disc supports most of the weight of axial compression forces, illustrates its potential to be a major factor in the manifestation of fixation dysfunction.

Both the insidious progression of major biomechanical disturbances of the spine, and the permanent effects of subluxation on spinal integrity, can be explained by disc related mechanisms. Instantaneous improvement in intersegmental mobility from spinal adjusting may be explained by the release of entrapped and sequestered disc material (39).

**Hypermobility.** Disc degeneration may lead to hypermobility. It is safe to say that hypermobility of the motion segment is impossible without significant disc dessication. Kirkaldy-Willis (28) describes a process of lumbar spine degeneration that involves a gradual progression of variable events which eventually leads from dysfunction to instability to stabilization. The incidence of hypomobility versus hypermobility resulting from progressive disc degeneration is not clear.

It is not to be assumed that abnormal discs are the only factors necessary to be considered in subluxation. Intervertebral disc derangement, however, is of paramount importance to the biomechanical aspects of the VSC.

**CONTRACTURE**

Soft tissue adaptation in the form of contracture (shortening) of muscles, ligaments, and tendons may act to restrict intervertebral motion (17). Reduction of contractures is probably one of the mechanisms involved in the restoration of joint movement and improvement in spinal curvatures. Prolonged fixation dysfunction from other mechanisms, however, likely precedes gross contracture.

**DISC PROTRUSIONS**

Abnormal disc changes can and do affect any and all levels from C2 to L5 (5). The highest incidence of clinically significant protrusions occur in the lower lumbar discs. Any disruption in the disc must be accompanied by altered mechanics of the three-joint complex. Marked disc protrusion is seen as an advanced stage of the process of degeneration, which begins very insidiously from an early age (16).

Substantial controversy exists regarding the indications for chiropractic treatment for patients with disc protrusions. Before advanced imaging techniques (e.g., MRI, computerized tomography), many patients with protruded discs were adjusted without consideration for the extent of the underlying disc derangement. Some chiropractors appear to be shifting the emphasis in therapeutic approach away from manipulative procedures for these
cases. Clinical experience, however, has been that most intervertebral disc displacements are manipulable lesions (39,43). The omission of the adjustment in treatment of many cases of disc protrusion likely results in unnecessary patient suffering.

Some feel flexion distraction techniques are preferable to manipulation for treatment of disc lesions. Others point out that the only studies showing efficacious manipulative treatment for disc protrusions used long lever rotational manipulations (44). The potential danger of worsening a disc protrusion by rotational maneuvers is related to axial torsion damage or failure. Flexion distraction’s proposed effect is longitudinal traction of the posterior annulus and posterior longitudinal ligament (PLL) in an attempt to produce a negative pressure within the disc thereby creating a centripetal effect on the nucleus (45). The flexion distraction techniques apply tension to nerves and ligaments that may be disrupted. Most of the damage to the annulus occurs posterior to the nucleus pulposus; therefore, traction would have the effect of stretch on the sprained ligaments. It is likely, however, that substantially more people with pain from disc protrusions have been helped than hurt via both of the above approaches.

The specific adjustment can be used in attempts to directly push the displaced nucleus anteriorly. We advocate specific, short lever adjustments, primarily in the +Z (posterior to anterior) direction for posterior disc displacements whenever possible (Fig. 3.8). Tolerance by the patient is most important. Any increase in leg pain during set-up or thrust should be considered a contraindication for that position and an alternative position or contact should be attempted. Each patient must be matched with the correct table and position. The spinous contact (at least initially) is preferred, as this contact produces the least rotational lever while maximizing +Z force (See Chapter 7).

![Figure 3.8. A, Displaced disc material with concomitant retrolisthesis of L5 vertebra. B, The +Z force applied during a specific adjustment attempts to influence the position of the displaced disc material.](image)

![Figure 3.9. The graph demonstrates in a qualitative way, the sethold feature of the specific adjustment.](image)

Creep properties of ligaments are time dependent (46). A force applied for a long duration, as in flexion distraction or posterior to anterior (+Z) long amplitude maneuvers, is likely to create a greater effect on the intervertebral ligaments when compared to short duration loads. A combination of the direct adjustment along with long duration forces is most likely to maneuver the sequestered disc material. In administering an adjustment, the doctor is advised to set, then hold pressure forward for several seconds, followed by a gradual release (Fig. 3.9).

The reduction of a protrusion as viewed with MRI or CT is not necessary for a substantial resolution of the patient’s signs and symptoms (47). Disc protrusions, though of great importance, do not always result in the direct production of symptoms (16,17). The disc can be the cause of pain without protrusion, as derangement may stimulate the recurrent meningeal nerve. Disc protrusion can reportedly account for lumbar pain anywhere from one to thirty percent of the time (48). Intervertebral dysfunction is also common with disc derangement in the absence of protrusion.

**OPEN WEDGE**

The original Gonstead subluxation theory revolved around the intervertebral disc and the changes it undergoes in response to trauma (22). The shape of the disc space on X-ray is used to infer the state of the disc. **Locating an “open wedge” on the antero-posterior (AP) radiograph or a degenerated disc on the lateral projection is not, however, pathognomonic of subluxation.** Many of the most misaligned FSUs are likely compensations for subluxations elsewhere in the spine (22).

It is incorrect to assume that the appearance of an open wedge on the AP radiograph indicates that the nucleus has moved towards the open side of the wedge. The direction of a nucleus shift or protruding annulus
may well be on the side opposite the open wedge. MRI, CT, or obvious clinical findings indicative of protrusion are much more accurate in determining the location of displaced disc material.

Vertebral end-plate invaginations may reveal the location of the nucleus (49). A variant of this is the classic Schmorl’s node (Fig. 3.10A-B). (5). Please refer to Chapter 7 for disc space findings in relation to spondylolisthesis and base posterior sacrum subluxations.

Examination

The potential cause of fixation-dysfunction must be determined if proper management of the VSC is to be achieved. To determine the cause of spinal joint fixation in a presenting patient, the chiropractor must first have an understanding of all the known causes of fixation as well as expertise in the analysis of spinal motion.

One of the greatest potential pitfalls in the differential diagnosis of the VSC relates to determining aberrant motion characteristics via palpation. Despite repeated attempts, investigations into joint end-feel motion palpation of the lumbar spine have yet to show good levels of interexaminer reliability (50). Intersegmental range-of-motion palpation (both passive and active) has not been researched adequately (51). Perceived results of an intersegmental range of motion assessment determined through palpation should not be weighed heavily relative to other means of assessment of vertebral motion (e.g., stress x-ray, videofluoroscopy). Radiography has been suggested as the most objective method of assessing intersegmental motion abnormalities (52). The risks associated with excessive x-ray exposure limits radiography as a means of frequent follow-up assessment. See Chapter 4 for a detailed description of the chiropractic examination.

NEUROPHYSIOLOGIC DYSFUNCTION

Any attempt at providing a complete, concise summary of the ramifications of a neurologic disorder is lacking, due to the inherent want for knowledge that exists with respect to the central nervous system. What is presented here is a working theoretical basis for the approach to the neurologic interference involved in the VSC.

Space Occupying Lesions

Neurologic ramifications from tumors, fractures, and other pathologies may be severe but are likely much less common than those occurring from degenerative changes of the intervertebral articulation. Neurologic interference can be seen as both a direct and an indirect result of subluxation. Lesions of a persistent space occupying nature predispose the nerve root complex to direct irritation (3). These conditions are exacerbated by the effects of tension placed on nervous tissue during movement and from pressure secondary to edema (10). A decrease in the cross sectional area of the spinal canal and intervertebral foramen has been shown to occur as a result of vertebral positional dyskinesia and disc displacement, as well as from changes accompanying the inflammatory and degenerative processes (28). The persistence of fixed positional dyskinesia at levels of subluxation ensures the longevity of the existence of the space occupying aspect of the lesion.

Figure 3.10. A, Lateral radiograph of a cadaveric specimen exhibiting a lucency of the inferior vertebral body of L4 indicative of an invagination of nuclear material through a fractured end-plate. Notice the subtle invagination in the L5 inferior vertebral body. B, A sagittal slice of the specimen in A exposing the gap in the L4 end-plate. The remains of the degenerated nucleus of the L5 disc are seen under the upward invagination of the L5 inferior end-plate.
Hadley (5) has suggested that abnormal constriction in the size of a normal intervertebral foramen, if not actually causing nerve root pressure, nevertheless decreases the reserve safety cushion space surrounding the nerve and may predispose it to pressure. The subsequent development of edema, hemorrhage, disc pressure, or movement of adjacent structures may be sufficient to produce radicular symptoms. He found evidence that cervical, thoracic, and lumbar articulations could produce intervertebral foramen encroachment when persistent malposition of one vertebra on another existed.

Kirkaldy-Willis (28) has suggested that nerve compression in the lumbar spine is most commonly associated with disc degeneration, either by itself, or in combination with degenerative and/or developmental stenosis of the spinal canal. Once stenosis occurs, the neurologic elements are more susceptible to insult from relatively small changes in disc displacement. This apparent predisposition to nerve involvement may explain why some individuals, with relatively minor physical exam findings present with more discomfort than others with apparently more objective evidence of dysfunction. The radiographic, CT, and MRI evaluations often illustrate this stenosis component.

**Adverse Mechanical Tension**

The effects of tension on the nerve elements must be considered when studying the neurologic ramifications of the VSC. The primary source of meningeal and neural tension is the lengthening of the spinal canal on forward bending and lateral flexion (53). Limb movements will also transmit tension to the nerve roots, causing piston-like movements of the root complex within the intervertebral foramen (17). Normally, the soft tissues adapt freely to these skeletal movements, but in the presence of space occupying lesions involving the spinal canal and intervertebral foramen, and when there are sclerotic or fibrotic lesions that restrict the mobility or extensibility of nervous and meningeal tissues, the tension may be greatly increased (17,53). Even when the lesion appears to be exerting an essentially compressive effect, the resulting deformation leads to a local increase in tension. An important cause of functional disturbances, both of the nerve access cylinders and the blood vessels, lies in the reduction of their cross-sectional areas, resulting from tension and/or compression (53).

Grieve (17) cites over fifty investigative reports of conduction block, ischemia, and post-ischemic paresthesia. "Severe and prolonged compression blocks the nerves' blood supply and produces other damage. It then loses its ability to conduct impulses. Prolonged inflammation appears to produce the same effect. Temporary compression will produce temporary loss of conduction, from minutes to days, depending on the degree and duration of the compression. Intermittent compression or mechani-

cal irritation may lead to inflammatory changes with space occupying effects produced by edema, and thus some or all of the changes and clinical features after inflammation. Traction of sufficient force to disrupt the nerve, will cause irritation and consequent neuritis." Swelling of the nerve root elements may be associated with some cases of neuritis (54).

Persistent and prolonged fixation, positional dyskinesia, and ensuing degeneration can create a stenotic predisposition to nerve involvement. Disc displacement, inflammation, and movement of nerve elements through the stenotic spinal canal and foramenal areas are all significant factors contributing to the manifestation of neurologically related conditions of the VSC.

**COMPRESSION EFFECTS**

Let us pursue some basic information about the nature of abnormal nerve function under compression and how the restoration of function ensues when pressure is removed. The "all or none" law refers to the principle that nerve tissue will either respond to stimuli completely or not at all. This law is applicable to the individual nerve fibers only. Studies of nerve compression have demonstrated that blockage of only some nerve fibers in the nerve is possible. The remaining fibers in a nerve root complex under compression respond to stimulus normally (21). The importance of this phenomenon is that the manifestation of compressive effects depends on those parts of the nervous system that are being affected. Conscious awareness of nerve involvement by the patient would only be possible if nerve pathways under voluntary control were involved. When objective findings of subluxation exist in the absence of subjective complaints, the necessity for the correction of the subluxation still exists. This necessity is not only for biomechanical reasons but also for the underlying potential for neurologic involvement that may result without notice.

There are reversible and irreversible conduction blocks that result from compression. A completely reversible conduction block seems to leave the nerve undamaged. The usual explanation of the reversible block is anoxia. The nerve deprived of oxygen ceases to conduct in 16 to 35 minutes. If neither axons nor blood vessels are damaged during the constriction, conductivity returns soon after the compression is removed (17).

The irreversible conduction block is characterized by disturbances in nerve continuity and degenerative structural changes in nerve and or vascular tissues. A greater amount of constriction and/or compression over a longer time period is necessary to produce an irreversible conduction block (17).

Neurologic findings that are almost instantaneously improved after the administration of an adjustment, such as deep tendon reflexes, motor power as measured by grip strength, and dermatomal sensory loss (55), may be due
to the removal of constriction in the area of a completely reversible conduction block.

Different types of nerve fibers appear to react differently to compression and its consequences. The number of fibers affected in the nerve varies with the degree of constriction (17). When a patient presents with seemingly irreversible signs of nerve damage, it should not be assumed that attempts at the correction of the subluxation will be of no neurologic value. Interruption of the constriction, although not likely resulting in the healing of tissues already lost to degeneration, may help to prevent further degenerative changes of nerve tissue by relieving compression and tension on elements not yet permanently affected.

CLINICAL CONSIDERATIONS

Compensatory hypermobility of the motion segment can be a potential cause of nerve irritation and degeneration. The major difference between compensatory and subluxated levels in their ability to produce direct nerve involvement is that compensatory areas are highly moveable and are not likely able to produce fixed stenotic lesions. Symptoms are often reported by patients in areas of compensation. These symptoms are usually temporary in nature and are generally easily abated by rest and/or applying corrective measures to areas of primary subluxation.

Denervation Supersensitivity

Injury or pathology may result in peripheral nerve or dorsal root ganglion (DRG) damage. Ensuing nerve fiber degeneration mainly takes the form of either axonal or segmental degeneration. Trauma induced axonal degeneration leading to secondary myelin sheath breakdown is termed Wallerian degeneration (56). Segmental demyelination occurs when Schwann cells and the myelin sheath are damaged without effect to the axon anatomically, yet the conduction of impulses may be impeded (57). After denervation, changes occur in the muscle and receptor sites that are characteristic of denervation supersensitivity (DS).

SIGNS

Denervation supersensitivity of peripheral nerves can be, at least to some degree, related to the VSC. Muscle and peripheral receptors become hypersensitive to circulating neurotransmitters and other stimuli after denervation of some neurons. Associated signs and symptoms of this phenomenon include cutaneous and myalgic hyperalgesia, autonomic dysfunction, trophic changes and increased muscle tone (57). These findings have been reported in "low back sprain" patients without otherwise obvious physical findings (58).

MUSCLE CHANGES

Denervation resulting from degeneration and inflammation local to the spine may lead to peripheral supersensitivity at the neuromuscular receptors. The area of response to acetylcholine spreads from that immediately adjacent to the receptor end-plate, along the surface membrane, and sometimes involves the entire nerve fiber (59). Normal muscle presents as relatively soft and undefined at rest (resting tonus is present). In the presence of DS, palpation reveals hypertonicity of resting muscle due to hyperexcitation of the muscle by the spindle. The muscle may become abnormally shortened because of this effect (57). Asymmetrical muscle shortening may eventually contribute to skeletal asymmetry.

MOTOR POINTS

The "motor point" is that skin region overlying the terminals of the neurovascular hilus at the point where the principle blood vessels enter the deep surface of the muscle. These motor points are not normally tender. Mild tenderness at motor points may be found in normal individuals after exercise. In patients with DS, the amount of tenderness at motor points is directly proportional to the severity of the radiculopathy (57).

TROPHIC CHANGES

The "boggy" or "doughy" characteristics of tissues overlying a subluxed FSU's paraspinal area, detected through palpation, may be due to a phenomenon termed trophedema. Partial interruption of a peripheral nerve causes gradual fibrosis of the subcutaneous tissue (60).

AUTONOMIC CHANGES

Vasomotor, sudomotor, and pilomotor dysfunction may be caused by DS. "Mottling" of the skin, palor and cyanosis, lower skin temperature, and rarely, pigmentation resembling erythema ab igne are vasomotor disturbances that have been related to DS (57). Hyperhidrosis resulting from DS may be seen beyond the confines of the involved nerve's distribution. Extensive sweating areas may include the axillae, palms and soles (57).

"Goose pimples" may occur as a result of brisk cool air exposure (as the patient undresses) over the skin of an affected area. This reflex may only be observable for a brief moment and can be a pilomotor effect of DS in the dermatomes of the involved nerves (57). This phenomenon can only be detected in the absence of a generalized pilomotor response.

Autonomic nervous system dysfunction via neurologic involvement at the spinal cord, nerve root and inter-
connections with autonomic ganglia, are also important considerations in the analysis, correction, and management of the subluxation.

From the standpoint of organic dysfunction resulting from subluxation, the autonomic nervous system has long been the focus of attention in chiropractic management. Imbalanced input from the parasympathetic and sympathetic nervous system has been implicated in the clinical manifestation of disease (27). The management of clinical disorders resulting from subluxation must include a full understanding of the nature of injury and healing of the spinal cord, nerve roots, and peripheral nerves. The aspiring clinician is encouraged to pursue an in-depth study into these areas to facilitate proper patient care.

**Facilitative Lesion**

Aberrant activity of the sympathetic nervous system (SNS) may be present at traumatized vertebral levels. This aberrant SNS activity has been implicated as a central factor in somatovisceral reflex disorders resulting in end-organ dysfunction (26). A hyperactive spinal cord level or “facilitated segment” may be the source of aberrant SNS signals. Sustained sympathicotomy may be related to kinesiologic disorders as present in the VSC (61). The basic mechanism involves the nerves supplying the mechanically traumatized FSU sending distorted information via the interarticular sensory apparatus to the corresponding segment of the spinal cord. This reflex activity results in increased postsynaptic response of a synapse to successive stimuli (62). The spinal cord segment becomes hyperirritable; relatively few impulses result in responses that are greater than would have normally resulted from the original impulse. All of the connected pathways involved in reflexes with the facilitated segment are affected similarly. Increased muscle tone and sudoriferous gland activity, pathology of the viscera, and blood vessel and skin temperature abnormalities, have all been postulated as resulting from this phenomenon.

Involvement of the ventral and dorsal nerve roots, the spinal cord and cauda equina, and the autonomic nervous system can result from subluxation in all areas of the spine (3). Virtually every part of the human body, therefore, can be affected by VSC.

**Spinal Learning**

The nervous system has the capability to become more or less sensitive to repeated exposure to the same stimulus. These are short-term alterations in responses termed behavioral plasticity and involve internuncial pathways (63,64). Habituation occurs most when mechanoreceptors are selectively stimulated. Sensitization occurs most when nociceptor input is maximum (64,65).

A review by Slosberg (66), asserts that spinal (as opposed to cortical) learning is possible via alterations in interneuronal pathways in response to pairing conditional with unconditional stimuli. A conditional stimulus (CS) is one that normally does not initially illicit a nervous system response. An unconditional stimulus (US) consistently evokes a response.

Slosberg’s review suggests that the interneuronal pathways are capable of a form of associative learning. For example, if a CS (e.g., light pressure) was paired repeatedly with a US (e.g., strong shock), the interneuronal mechanisms “learned” to associate the CS with the US and later responded to the CS alone as if it were a US.

**CLINICAL CONSIDERATIONS**

The implications of spinal learning as they relate to the VSC are potentially widespread. Spinal learning may be able to explain long-term hyperactive muscle and sympathetic tone (67).

Spinal learning in relation to the VSC could result from the following hypothetical situation. A long-term bout of severe pain results in chronic nociceptive involvement at the segmental level involved. Muscle spasm often results from such pain. Motor pathways that were once stimulated for an extended period of time (i.e., hours or days) in response to the nociceptive bombardment may be reexcited later (e.g., hours to months) by a CS in the absence of significant pain. The CS may be mild mechanoreceptor input or sympathetic effector activity (66). Even though the patient’s initial painful etiology no longer exists, relapsing spasm (or other responses) may occur as the result of a stimulus that would normally go unnoticed.

The more prolonged the bombardment by nociceptors, the more lasting the effects of spinal learning. It is unclear whether the effects of severe alterations produced by intense patterns of input can be completely reversed (68). The long-lasting, possibly irreversible effects of these mechanisms constitute the most plausible rationale for the supplementation of chiropractic care with physiotherapeutic modalities. The restoration of the mechanical function of the joint through the adjustment may fall short of normalizing the neurologic component. Consider, for example, in the case of a patient with severe degeneration at the level of the VSC combined with several months of severe secondary extremity pain. After a reasonable trial of spinal adjusting, if no change in the patient’s signs and symptoms has occurred, a judicious trial of a modality such as electrical stimulation may potentially have merit. It is possible that such modalities may act to reprogram pathways that have been altered by nociceptive bombardment. The authors’ clinical experience has been that the numbers of patients that would fall into this category are relatively few. Unfortunately, the need for physiologic therapeutics is often based on the
potential for financial gain rather than on the patient’s needs. The lack of controlled trials to support the indications in individual cases complicates this picture.

**Viscerosomatic Reflexes**

Components of the VSC may develop without primary involvement of the ligamentous tissues. Factors of the VSC such as pain, spasm, and movement deficits may result from primary visceral etiology. Somatic reflections of visceral dysfunction have been described (69). The transmission of pain on the afferent fibers of the pharynx, esophagus, thoracic viscera, stomach, intestines, kidney, ureter, gall bladder, and bile ducts, in response to electrical stimulation pass primarily by the sympathetic nerves, no: the vagus (12). The exact nature of these afferent pathways are not completely understood. Malliani et al. (69) “conclude that visceral pain may reflect the abnormal involvement of a sensory substratum subserving usually the mediation of reflex functions and of vague sensations, so important to feelings. Mechanical stimuli, chemical substances, interactions like ‘sensitization,’ circumstances like ‘inflammation,’ may all contribute to that abnormal quantity in the receptive process that causes the unpleasant experience called pain.”

Organ dysfunction of sufficient magnitude to initiate noxious afferent bombardment of the spinal cord segment may create compensatory mechanisms in the spinal column (antalgia, spinal learning, facilitation) which alters the normal muscle physiology of the region. If the bombardment is of sufficient duration, the muscle involvement may result in interarctical effects similar to those of primary joint trauma. Immobilization caused by spasm may lead to degenerative changes as previously described herein. The above theoretical scenario would necessitate initial stimuli of sufficient strength and duration to create prolonged, self propagating nervous system involvement.

Consider the following example of this theoretical mechanism. A severe episode of appendicitis (without previous spinal concomitant) of two days duration creates excruciating abdominal pain. The pain is accentuated by movement. The myriad of symptoms include acute constipation, vomiting, and fever. Visceral afferent pain transmission referred to the lower thoracic spine segmentally may result in interneuronal bombardment, thus affecting other normally uninvolved nerve pathways. This bombardment lowers the reflex threshold of the musculature associated with that spinal segment. Normal spinal movements may now cause exaggerated neuro-

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**Figure 3.11.** Trauma may create a cascade of events (some cyclic in nature), causing pain via diverse interrelated mechanisms.
muscular responses similar to those involved in spinal learning and facilitation, thus perpetuating the cycle of events (Fig. 3.11). Related symptomatic such as the forceful muscle contraction involved in vomiting, general myalgia associated with infection, and the psychologic stress of physical illness may further promote the cycle of events via both afferent and efferent pathways.

Pain

The clinical presentation of pain should be understood in the management of VSC related disorders. The diversity of the possible causes and ramifications of pain may obscure the clinical picture (See Figure 3.11). A simple model for pain production and its effects is not available. Theories that oversimplify this complicated phenomenon are attractive, but are seldom accurate.

NOCICEPTIVE REFLEXES

Noxious impulses from nociceptive stimulation of free nerve endings in an area of chemical or mechanical irritation may be a primary etiologic factor in somatic dysfunction (25). Nociceptive distribution includes all connective tissues of the body, excluding the brain stroma. Periosteum, peritoneum, dermal and subdermal tissues, joint capsules, ligaments, muscles, tendons, and muscle fascia are all recipients of nociceptive free nerve endings. Contrary to the beliefs of some (70), the discs have a nerve supply to the outer one-third to one-half of the annulus (71). It is important to remember that there is nociceptive supply to the meninges, stroma of all internal organs, and all blood vessel stroma (excluding capillaries) (72–77). Specific segmental pain patterns will be discussed in subsequent chapters.

There is variation in the sensations of pain from individual to individual and within the same individual at different times. Characterizations have been described as sharp or knife-like, throbbing and specific or diffuse. Nociceptive impulses may generate reflexes that modulate somatic and visceral physiologic behavior (25). The fact that nociceptive fibers are so vastly distributed throughout the body indicates their overall influence on somatic dysfunction. The physiologic convergence and divergence of stimuli further illustrates their ramifications on the VSC.

GATING MECHANISMS

Wall and Melzak (78) have described the gate control theory of pain. Impulses concerned with pain transmitted from the first central cells of the spinal cord depend on three factors:

1. The arrival of nociceptive messages;
2. The convergent effects of other peripheral afferents which may exaggerate or diminish the effects of the nociceptive messages; and
3. The presence of control systems within the CNS which influence the first central cells.

Injury-produced afferent signals pass through a gate control where they are modulated by other peripheral events and by the sensory posture set by the CNS (Fig. 3.12). This system changes slowly after injury so that central connections can provide control. Wall and Melzak believe that as theories of pain evolve, it will become necessary to reintegrate pain as one of the main modes exhibited by a single sensory emotional behavioral system (78).

DENERVATION SUPERSENSITIVITY AND PAIN

Dermatome changes resulting from DS include paresthesias such as numbness, deadness, and tingling. The patient may experience muscle pain and tenderness. Scleratogenous pain from DS would be dull, aching or boring, deep, and poorly localized.

Sensory disorders, including pain secondary to DS, may involve the nociceptor system in the vasculature. The sensory disorders may be atypical to segmental associations. The double innervation of all blood vessels by sympathetic and nociceptive nerves makes them very susceptible to DS.

Causalgia is an example of the late stage of DS. Many atypical pain presentations can be an indication of early and subtle manifestations of DS.

Correction of Nerve Involvement

Much is yet to be discovered about the effects of nerve interference as well as how chiropractic adjustments may decrease aberrant nerve activity. Extensive clinical experience has been contributed by the chiropractic profession which provides a wealth of information on the clinical management of vertebral subluxation and its neurophysiologic effects. Later chapters will attempt to correlate and present case management information exemplifying those clinical observations. An adjustment can potentially result in the reduction of nerve interference via more than one mechanism.

MECHANICAL EFFECTS

Certain aspects of vertebrogenic interference at the subluxated articulation involve stenotic factors. The fixation that can hold an articulation in a mechanically stenotic configuration has been shown to respond to manipulation (30,55). Restoration of normal motion characteristics reduces the likelihood of persistent stenosis. The tendency toward prolonged compression and adverse mechanical tension on nerve and perineural tissue would be diminished if the pliability of movement returns to the involved articulation.
Intervertebral disc displacement is a major contributor to compression. The design of specific adjustments feature an emphasis on disc integrity. Posterior to anterior forces applied to a vertebra through its center of mass when indicated, are utilized to influence position of the disc material that has been displaced posteriorly into the area occupied by the nerve elements, without compromising annular integrity. It has been reported that disc protrusions showed reduction immediately after an applied manipulative force (long lever Y axis rotation) (79). Traction has been shown to have only temporary effects on diminishing displacement of disc material (39,80).

Recurrent dynamic nerve entrapment may occur at hypermobile FSUs (28). Reducing the need for movement at these unstable motion segments by increasing movement at restricted FSUs can possibly limit the occurrence of this type of nerve involvement. Abnormal mechanoreceptor stimulation may also decrease if hypermobility is reduced.

REFLEX EFFECTS

One mechanism that is thought to be an essential concept in manual therapy is the presynaptic nociceptive inhibition by proprioceptors after mechanical stimulation of group I, II, and III proprioceptors, primarily in zygapophyseal articulations (81). A spinal adjustment may selectively stimulate the proprioceptors, while not affecting the nociceptors, thereby blocking the perception of pain (See Figure 3.12). Somatovisceral, viscerosomatic and somatosomatic reflexes may all be affected by the above mechanisms.

INFLAMMATION

Ameliorating inflammation may also reduce adverse tension or chemical irritation to neural elements. Eliminating mechanical irritation by correction of abnormal biomechanics likely interrupts the inflammation cycle. Increasing motion in an area of fixation dysfunction with edema may help circulate inflammatory by-products and reduce pressure and chemical irritation.

Chiropractors must be careful not to hastily accept the theoretical mechanisms involved in concepts formulated to explain the effects of spinal adjustments. It is likely that the underlying mechanisms involved will become illuminated and clarified with further research. Our limitations in our ability to aid patients with their vast array of health conditions remains unclear.

MYOLOGIC CHANGES

Nerve, muscle and tendon changes are interrelated. Muscle degeneration occurs secondary to fixation dysfunction and contributes to joint degeneration. Alterations in muscle tissue after immobilization include those of gross structure, morphology, metabolism, connective tissue, biomechanics and contractile and neurologic/bioelectric properties (2). Immobilization has been associated with adhesion formation between the tendon and its sheath, decreased muscle extensibility (82) and a reduction in the number of sarcomeres (83). Muscle changes from fixation dysfunction are often completely reversible depending on the type of muscle and the duration of the immobilization (2).

Spindle Effects

Immobilization can lower the threshold and increase the sensitivity to stretch of muscle spindles of the muscles related to the affected articulation (84). Spindle degeneration begins within one week of the onset of joint immobilization (85). One might speculate that these muscle spindle changes may account for the results seen in a study whereby spinal adjustment reduced the hyperactivity of paraspinal musculature (86).

Clinical Considerations

The potential importance for chiropractic care with respect to limiting muscle degeneration secondary to the VSC should be obvious. The cause and effect relationship of many of these mechanisms, however, remains unclear. A common misconception of the etiology of the VSC proposes that hyperactive muscles directly damage the ligaments of the FSU by pulling vertebrae out of alignment. Mathematical models have proposed that the muscles of the lumbar spine cannot exert enough force to damage the ligamentous elements (87). Most damage to the intervertebral joint results from failure of the muscular and ligamentous systems while attempting to resist external loads.

Stress

Abnormal mental states may be perpetuating factors in muscle pain (88). It can be convenient for unknowing
doctors to assume a primary psychogenic etiology in patients with confusing clinical pictures involving muscle pain. It is doubtful that psychologic factors represent the primary etiology in most of these cases. Prolonged physical dysfunction, however, will almost assuredly involve some psychologic component and vice versa. Referral for concomitant psychologic counseling should be considered in the management of these patients.

Chiropractic intervention with regards to viscerosomatic dysfunction revolves around normalizing the primary visceral etiology (See Chapter 13). Psychologically induced somatic dysfunction may involve similar mechanisms as viscerosomatic conditions. Overall chiropractic spinal management may be indicated in these conditions, possibly reducing the likelihood of exacerbations of previously present subluxations during the acute phase, and contributing to the overall patient well-being (89). Future research is needed to clearly define the role of chiropractic care in aiding the patient in the acute and recovery stages of these disorders. It is possible that early mobilization of the concomitant spinal areas may minimize the exaggerated muscular responses and subsequent production of pain. Referral for medical care should be considered in all of these cases. A primary visceral etiology should be suspected in patients that do not otherwise respond to adjustments. It is the authors' opinion that primary visceral causes of the VSC are relatively rare; however, these mechanisms should be considered in light of the potentially devastating consequences from mismanagement.

INFLAMMATION

Interarticular inflammation from trauma causes edema that reduces joint movement. Immobilization can cause degenerative joint disease (DJD) (90). Arthritis or DJD is usually diagnosed radiologically by the presence of bony proliferation and decreased soft tissue at the joint space. Chronic or repeated episodes of inflammation may eventually result in ossification of paraspinous ligaments (91). The restoration of motion leads to a decrease in the degenerative process (2).

Inflammation results in hyperexcitable nerves and causes ganglia to continue firing long after stimulation has ceased (2). Inflammation is a potentially powerful enhancer of pain production and prolongation, as well as increased muscle tone (which also may restrict movement) through hyperexcitation of the nerves exposed to inflammatory byproducts.

VASCULAR CHANGES

Alterations in the arterial supply and venous drainage of the spinal column has been suggested as a contributing factor to interarticular degeneration after immobility (2). Experimental occlusion of the arterial supply and/or venous drainage can lead to joint stiffness (91). Lantz (2) suspects that when venous stasis occurs the reduced rate of removal of cellular toxins leads to inflammation and accelerates the degenerative process. Immobilization, disc herniation compressing on epidural veins (92), or SNS dysfunction creating arterial constriction may be involved in VSC progression. Blood flow restriction may heighten the effects of trauma and the interarticular dehydration that follows immobilization.

ARTICULAR NOISE

Evidence suggests that the cracking sound of the articulation that occurs in response to manipulation is due to coaptation of articular gases in synovial joints (93). When the separation of the joint surfaces is great enough to coaptate the articulation, an abrupt liberation of gas from the synovial fluid occurs (Fig. 3.13). Once this coaptation of gas has occurred, there is a refractory period during which the articulation experiences a greater degree of interarticular freedom of movement. Manipulation of the joint during the refractory period may not produce another audible crack. It takes approximately twenty minutes for the gas to slowly redissolve and end the refractory period (93).

Repeating a thrust into an articulation after fully coaptating the joint by a prior thrust may result in ligamentous strain more readily than would be expected to occur in an uncoaptated joint. The coaptation forces are thought to provide an elastic barrier of resistance to gapping of the articulation (94). It requires less force to overcome coaptative forces in an unrestricted joint than in an articulation restricted by previous interarticular changes (95). Specific adjustments aimed directly at restricted articulations are indicated in preference to long lever, regional manipulations for the above reason.

![Figure 3.13. The graph depicts the progressive application of force to separate a synovial joint (arrow = the point of coaptation).](image)
CORRECTION OF THE VSC

The clinical application of our understanding of specific full spine chiropractic management is illustrated in the following examples of the VSC.

Example One

Consider one common type of subluxation pattern encountered in the cervicothoracic spine. A patient is suffering from midline, lower neck pain, and objective signs of the VSC (as will be discussed in Chapter 4) are found on the patient at the C7-T1 motion segment. The static upright neutral A-P x-ray of the area involved reveals the presence of $-\theta Z$ (left lateral flexion) and a $+\theta Y$ (spinous process rotated to the right side) alignment of the C7 vertebra on T1. The lateral radiograph exhibits the presence of $-\theta X$ alignment (an extension misalignment) of C7 on the T1 vertebral with concomitant C7 $-Z$ alignment. The translation into Gonstead nomenclature of the above positional dyskinesia is PRS-inf (22). The motion characteristics of the C7-T1 articulation are analyzed, and the C7 vertebra is found to be restricted in $+\theta Z/-\theta Y$ (coupled right lateral flexion and contralateral spinous rotation) and $+\theta X$ (forward flexion) motions.

The positional dyskinesia and the fixation dysfunction are considered secondary components to the disruption of the normal functional and structural characteristics of the FSU. The alignment of the joint will help to determine the mechanism of injury. The trauma in this example most likely involved forces of compression ($-\theta Y$), left lateral bending ($-\theta Z$) and or axial rotation ($+\theta Y$) of the head and neck, and possibly flexion or extension ($\pm \theta X$). This then led to the motion segment assuming a position of $-\theta Z$ (P), $+\theta Y$ (R), $-\theta Z$ (S), and $-\theta X$ (inf). The effects of weight bearing and posture help to further the accelerated plastic deformation of the damaged interarticular soft tissue. The vertebra(e) settles into the direction of the ligamentous weakness. Inflammation and scarring with fibrous adhesions add to the articular dysfunction.

An adjustment chosen to correct such a subluxation would include pretensioning and applying a force in the opposite direction of the positional dyskinesia, usually with the patient's neck in $+\theta Z$ (right lateral flexion) (Fig. 3.14). The thrust would be from posterior to anterior, with inferior to superior lifting of C7 on T1, from the right side contacting the right posterior inferior portion of the spinous process (Fig. 3.15).

The goals of the application of force in this maneuver are to improve mobility in the direction the articulation was lacking (fixation dysfunction) and to normalize the interarticular alignment (positional dyskinesia). After the adjustment, the motion is reanalyzed to determine if improvement occurred. Alignment changes, if any, are seen on comparative radiographic examinations.

CLINICAL CONSIDERATIONS

As mentioned earlier, many cases are acute presentations of a chronic underlying condition. The fixation dysfunction in the above example likely represents the combination of nuclear-annular disrelationships, adhesions, malaligned joint surfaces, edema and muscle spasm. In the presence of nuclear displacement it is important to first separate the vertebral end-plates before applying the force aimed at directing the nuclear material towards a more

Figure 3.14. This line drawing illustrates the effects of prepositioning the spine for a specific adjustment designed to correct a C7 PRS subluxation.

Figure 3.15. A photograph demonstrating the correct set-up and multiple directions of forces that combine to make-up the desired correction of a C7 PRS subluxation.
normal position. Because of the extensive connections of the annulus to the vertebral body, forced translation of one vertebra on another can potentially affect disc position. Rotary manipulation has undergone controlled clinical trials showing effectiveness in the management of patients with disc lesions (47). More research in this area is necessary. If function is to remain more normal after a correction is made, intradiscal healing must occur, sealing the fissures and preventing nuclear remigration. Repetitive adjustments, and possibly nutritional supplementation, may be necessary to aid the FSU in its return to normal function, because of the slow repair process of the tissues involved. Proper patient ergonomics are imperative to this healing process. Over time the disruption of function and persistent malalignment will lead to degeneration of all supportive soft tissues which further ensures the persistence of dysfunction.

Example Two

A common subluxation of the lumbosacral articulation involves a posterior nucleus shift through radial fissures in the annulus. This eccentric nuclear position creates blockage to extension movement at L5-S1. The lateral radiograph will show the discal abnormality, by exhibiting a more parallel or open posterior configuration of the usually anteriorly wedged appearance of the L5-S1 disc space (Fig. 3.16). Severe low back pain and concomitant sciatica or referred leg pain (at times bilaterally), can accompany the above lumbosacral subluxation.

Correction of this type of subluxation (termed a base posterior sacrum subluxation, See Chapter 7) is easily accomplished in the side posture position, but can also be achieved in the knee-chest or prone position. The doctor manually contacts the posterior-superior aspect of the sacrum at the S1 or S2 tubercle and applies a +Z (posterio to anterior) force, following through with an inferior to superior arcing motion of the contact hand. The anterior aspect of the L5-S1 disc space is separated and the base of the sacrum is pushed forward, allowing disc material to be moved forward away from the spinal canal. The inferior to superior motion at the end of the forward thrust is aimed to further force nuclear material out of the posterior aspect of the disc space. L5-S1 is then re-evaluated for the presence of extension movement. Clinical observations suggest that the positional dyskinesias of the base posterior sacrum appears reduced on x-rays taken after a series of corrections have been made, relatively more often when compared to other positional dyskinesias.

The above examples represent only a small sample of the potential subluxation configurations encountered on a daily basis in a chiropractic practice. Much of what is applied in chiropractic practice is based on the inductive reasoning from clinical observations in combination with known experimental results. The paradigm is far from complete however.

SELECTIVE ADJUSTING

Mixing Systems

A common question proposed by chiropractic students, that is not often clearly answered, pertains to when it is acceptable to “mix (autonomic) systems.” The clinically relevant aspect of this question centers on the separation of adjustments applied to the lower cervical, thoracic, and lumbar vertebrae, from those delivered to the upper cervical or sacral regions. To address this question completely, one must consider more than just parasympathetic nervous system (PNS) and sympathetic nervous system (SNS) effects.

Gonstead advocated the separation of adjustments affecting both of the main divisions of the autonomic nervous system. Affecting either the PNS or the SNS is considered especially important in the management of patients with a visceral concomitant. When a specific visceral effect is desired (e.g., increasing intestinal motility), selective adjusting of subluxations influencing only one autonomic division at a time may be indicated (See Chapter 13).

Reducing Variables

During the initial phase of chiropractic care, reducing the number of VSCs adjusted will diminish the number of variables the doctor must consider in evaluating the response to treatment. By adjusting less motion segments each session, it is easier to determine the apparent cause and effect relationships between the patient’s response and the treatment applied. This concept is also applicable to mixing types of treatments as with chiropractic and
adjunctive therapies. If a patient responds favorably to a treatment administered then all is well. If, however, the doctor applied more than one type of treatment or adjusted more than one FSU (especially in close proximity) and the patient did not respond favorably, it may prove difficult to determine what steps to take next. It is important to remember that treatment is partially diagnostic. The “shotgun” approach of applying multiple therapeutic modalities together may prove to relieve initial symptoms more rapidly than spinal adjusting alone (96). In long-term corrective care, however, a step by step systematic approach to treatment will be more valuable. Through the limitation of variables, the chiropractor progressively learns about specific adjustments and their outcomes. Eventually, more levels can be adjusted at one time or additional care can be prescribed when adequate information has been obtained from the treatment trial.

Selectively separating lumbar adjustments from those directed to the sacroiliac joint may be useful not only for separating autonomic divisions but also for mechanical reasons. Lumbar subluxations can refer pain to one, or less commonly to both sacroiliac articulations. An example of reducing the number of variables in the initial treatment phase may occur when the patient presents with signs and symptoms of a VSC in both the lumbar and sacroiliac areas. By limiting treatment to one area or the other, the potential mechanical effects of the adjustment can be more clearly delineated.

In instances where the patient is seen on an infrequent basis, multilevel adjustments, when indicated, may be more effective since a lack of attention to some motion segments may result in a deterioration of the patient’s condition. In some cases, normalizing the structural and neurologic balance in the body by correcting subluxations in multiple regions of the spine can be advantageous.

Proprioceptive Effects

The upper cervical area is richly endowed with proprioceptors. This abundant supply is responsible for head-to-body proprioceptive balance (97,98). The potential neurologic effects of upper cervical adjustments is evidenced by the fact that nerve dysfunction at this level may potentially affect any spinal cord pathway. To more accurately assess the effects of upper cervical adjustments, it is often necessary to adjust this region alone.

SUMMARY

The understanding of mechanisms involved in the VSC will evolve with further knowledge, which is a by-product of continued investigation. The practical application of chiropractic has developed empirically after millions of clinical observations. It is likely that many of these applications will withstand the test of time and clinical research; those that do not should be refined or discarded.

This work is but a foundation open to the addition of any new developments related to the care and maintenance of spine related disorders.

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