

REVIEWS OF THE LITERATURE



A Review of Biomechanics of the Central Nervous System—Part I: Spinal Canal Deformations Resulting from Changes in Posture

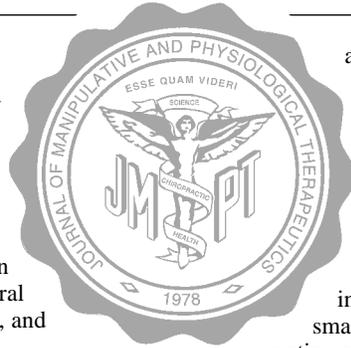
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ABSTRACT

Objective: To discuss how the spinal cord deforms as a result of changes in posture or biomechanical alterations of the spine.

Data Collection: A hand search of available reference texts and a computer search of literature from the Index Medicus sources were collected, with special emphasis placed on spinal canal changes caused by various postural rotations and translations of the skull, thorax, and pelvis.

Results: All spinal postures will deform the spinal canal. Flexion causes a small increase in canal diameter and volume as the vertebral lamina are separated. Extension causes a small decrease in canal diameter and volume as the vertebral lamina are



approximated. Lateral bending and axial rotation cause insignificant changes in spinal canal diameter and volume in cases without stenosis.

Conclusions: Rotations of the global postural components, head, thoracic cage, and pelvis cause changes in the diameter of the spinal canal and intervertebral foramen. These changes are generally a reduction of less than 1.5 mm in extension, compared with a small increase in flexion of approximately 1 mm. These small changes do not account for the clinical observation of patients having increased neurologic signs and symptoms in flexion. (*J Manipulative Physiol Ther* 1999; 22:227-34)

Key Indexing Terms: Posture; Spinal Cord; Biomechanics; Stenosis

INTRODUCTION

The spinal column is a complex mechanical structure. Alterations in the biomechanical alignment of this structure over time, as seen with abnormal asymmetrical posture, results in degenerative changes in the muscles, ligaments, and bony structures.¹⁻⁶ Asymmetrical posture may also be directly or indirectly responsible for spinal pain syndromes.⁷ Because of this, health care professionals are interested in correction of biomechanical dysfunctions and alterations of upright human posture as a primary focus or goal of care.^{8,9}

More important, alterations in posture, especially in the sagittal plane, have both direct and indirect effects on the central nervous system (CNS) and its associated structures.¹⁰⁻¹³ There is a large body of literature supporting the hypothesis that the static and dynamic deformations of the spinal column are directly transmitted to the CNS.¹⁴⁻²³ Knowledge of the stresses and strains in the CNS may lead to an increased understanding of altered neural physiology,

degenerative disorders, and acute or chronic pathologic processes occurring in the nervous system. To fully appreciate this, one must understand the biomechanics of the CNS. This body of knowledge provides insight into the way mechanical forces influence and interact with the anatomy and physiology of the neural tissue system. The purpose of this 3-part review is to provide evidence for the link between structural alignment of the spine and forces on the CNS. We hope that clinicians will gain further understanding how the structure of the spine affects the function of the nervous system. For Part I, a review of spinal canal dimensional changes is provided.

To discuss forces on the CNS, disks, vertebrae, and ligaments, an agreed upon Cartesian coordinate system is needed for communication. Fig 1 illustrates the directions for the positive x, y, and z axes suggested in 1974,²⁴ which will be used in this article. Panjabi et al²⁴ chose the y-axis to be vertical instead of the conventional use of the last letter of the alphabet z. Thus their right-hand screw system has different directions of the positive axes compared with mathematicians, engineers, and NASA.²⁵ Therefore when reading articles authored by the latter group, care must be taken to change coordinate axes for proper determination of rotations and translations causing spinal cord deformations.

DISCUSSION

Four Types of Structural Loading

Engineers are taught to analyze stresses and strains occurring in the four basic types of loads applied to structures.²⁶ We recall that stress is force per unit area. Because force is a

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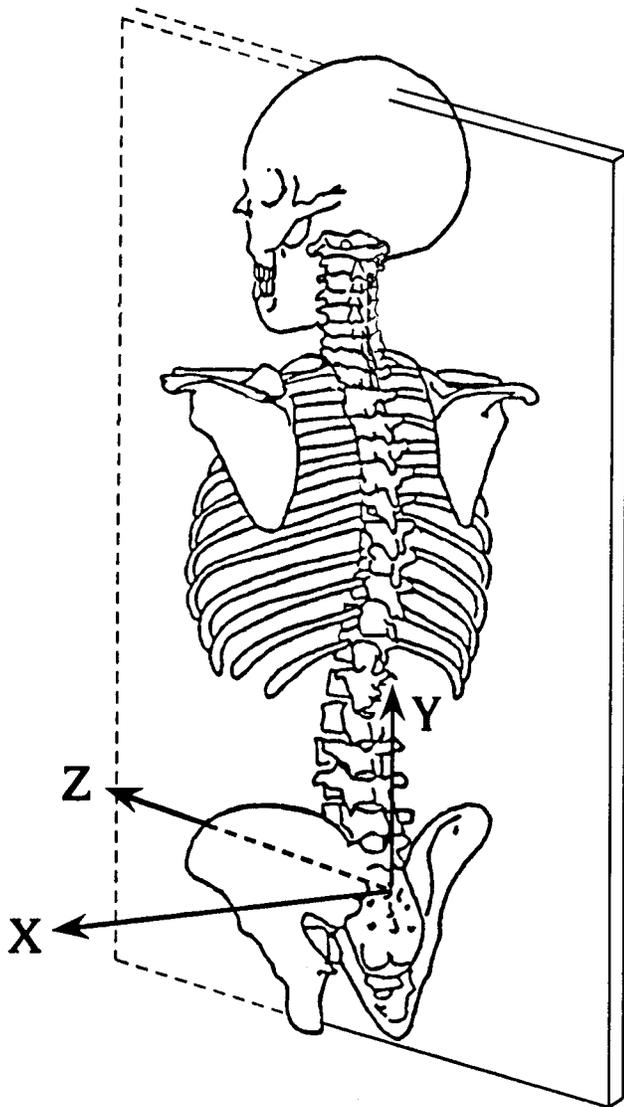


Fig 1. Cartesian coordinate system. Arrows depict the positive directions for the x, y, and z directions suggested for human biomechanics in 1974. This is a right-hand system.

vector, stress is a vector. Engineers use a stress tensor (matrix) composed of 9 stress vector components to completely analyze internal forces on an object (Fig 2). We will restrict our discussion to the components of the stress vector. There are two types of stress components: normal (longitudinal) and shear (cross-sectional). Fig 2 illustrates that an arbitrary force on a structure results in both normal and shear stresses. Fig 3 illustrates stress vector components resulting from the four basic types of loads: axial, transverse, torsion, and pure bending. It is important to note that stresses cause strains. Strain is a measurement of deformation, and there are two types: axial (also called *normal*), which is expressed as a percent change or some decimal value, and cross-sectional, which is expressed as an angle or configuration change. Physicians need to be familiar with these concepts to understand forces applied to spinal struc-

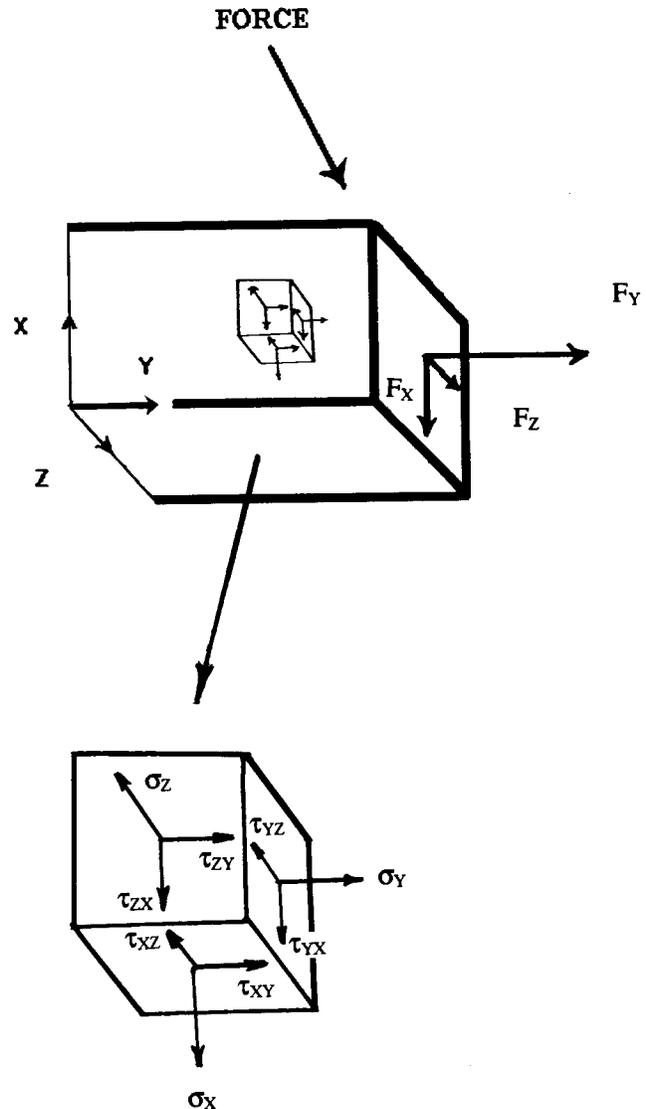


Fig 2. Applied force causes 9 stresses. Engineers keep track of stress components in all directions at once with a stress tensor, which can be considered a 3×3 matrix with 9 elements. An arbitrary force applied to a structure will cause a 3-dimensional system of stresses; 6 shear stresses, τ_{yx} , τ_{yz} , τ_{xy} , τ_{zy} , τ_{zx} , τ_{xz} and three normal stresses, σ_x , σ_y , and σ_z . The shear stresses cause angle changes (deformation) in the cross-section, whereas normal (longitudinal) stresses cause length changes in tension or compression.

tures, including the CNS. The compressive force of gravity on the upright spine is an example of axial loading, but longitudinal traction (tension) is also considered an axial load. Forces causing x-axis and z-axis translations of the skull to thorax and thorax to pelvis are examples of transverse loads. Shear is a special type of transverse load where application of the load and the support vectors are close together but in opposite directions. Axial torque causing axial rotation, y-axis rotation in Fig 1, is an example of torsion in the spine, whereas z-axis rotations (lateral flexion) and x-axis rotations (flexion-extension) are examples of pure bending.

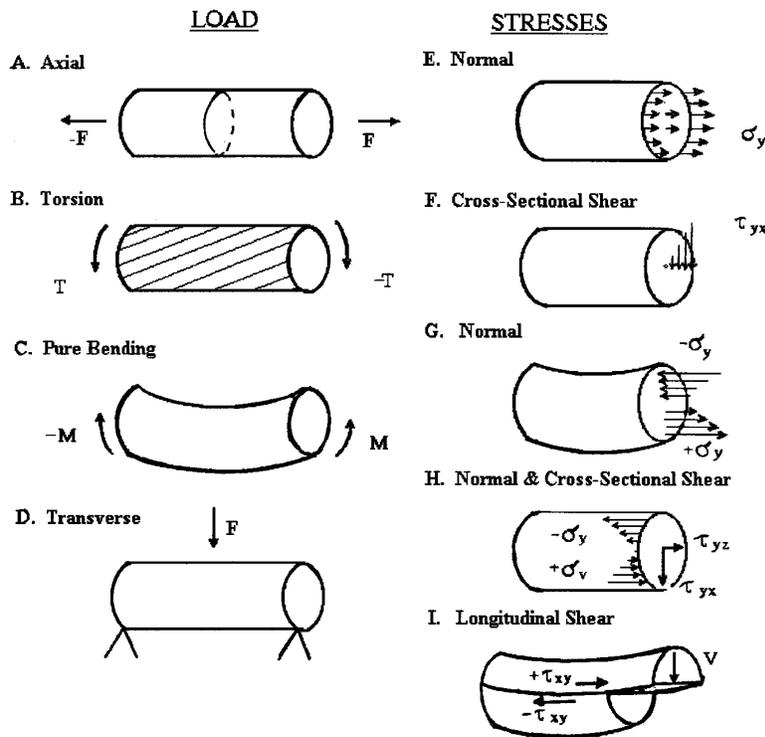


Fig 3. Four types of loads and their induced stresses. (A) The axial load ($F = \text{force}$) on a cylinder is tension, and the stress induced (B) on a vertical cross-section is a normal stress σ_y . There is a maximum shear stress at a cross-section of 45 degrees not shown. (C) Torsion ($T = \text{torque}$) is applied to a cylinder and the stresses are shear stresses, τ_{yx} , in the plane of the cross-section (D). (E) A bending moment, M , is applied to a cylinder and the stresses are normal, σ_y , but there exists a neutral plane with compression ($-\sigma_y$) in the top portion and tension ($+\sigma_y$) at the bottom (F). (G) A transverse load, which is applied to a cylinder ($F = \text{force}$), can be decomposed into a shear, V , in the cross-section and a bending moment with compression on top and tension on the bottom (H). It is known that transverse loads always cause an additional longitudinal shear, τ_{zy} (I), which is equal in magnitude to the vertical shear force, V .

With these engineering concepts in mind, the stresses applied to the hindbrain, brain stem, spinal cord, and nerve roots can be evaluated in various postural positions (loads on the spinal structures). To begin, some general observations concerning the spinal canal during postural movements are presented, and then finally, some specifics of CNS stresses resulting from postural positions will be reviewed in Part II of this review in a future issue of *Journal of Manipulative and Physiological Therapeutics*.

Flexion and Extension

The spinal canal changes in length during physiological movements or alterations in posture. However, only the traditional planes of motion (Rx, Ry, Rz) have been studied for their effects on the spinal canal and spinal cord. With the exception of axial tension (Ty), the translations (Tx and Tz) of the head and thorax have been ignored in the literature. Flexion and extension (Rx) lead to the largest changes, but scoliosis, lateral bending (Rz), and axial rotation (Ry) also lead to significant deformations of the spinal canal. The axis of motion for single rotations is normally in the disk or vertebral bodies.²⁷ Consequently, during flexion of the spinal column,

structures anterior to this axis will shorten and be exposed primarily to compressive forces, whereas tissues posterior to the axis will lengthen and be exposed mainly to tensile forces. The total change in length of the spinal column from extension to flexion was estimated by Breig¹⁰ to be 5 to 7 cm, whereas Louis¹¹ suggested 5 to 9.7 cm. This change in length is greater at the posterior aspect of the canal because of its increased distance from the axis of motion. Thus the strain in the posterior portion of the canal contents will be larger than the strain of the anterior structures.

Beginning in maximum extension and moving to maximum flexion, the anterior portion of the cervical canal increases in length 0% to 24%, whereas the posterior canal increases by 28% to 61%.^{10,12,13} Because of the rib cage, the thoracic spinal canal has a decreased sagittal plane mobility, although an increased kyphosis will increase the length of the canal up to 3 mm.¹¹ Similar to the cervical spine, the lumbar canal has a relatively large increase in length during flexion. Here the central axis of the canal increases in length by 20%.¹⁰

Holmes et al²⁸ measured spinal canal volume in neutral and flexion-extension of the lower cervical spines (C2 to C7) of 10

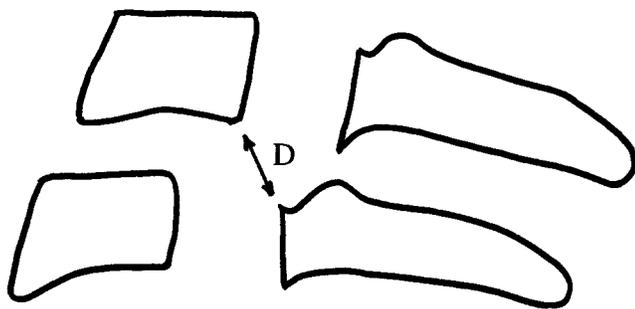


Fig 4. Dynamic sagittal diameter of the spinal canal. In extension, the dynamic sagittal diameter (D) of the spinal canal is the distance from the upper crest of the spinous-lamina junction of the inferior vertebra to the posteroinferior body corner of the superior vertebra.

cadavers. The volume of liquid displaced increased with flexion and decreased with extension, but the average total change from extension to flexion only amounted to 1.9 mL.

Axial Rotation

Axial rotation (R_y) of the spinal column or extremities sets up a physiological stress inside the pons-cord tract, although no overall lengthening of the bony canal occurs.^{10,13,27} Depending on the segmental level, however, increases and decreases in the canal dimensions do occur. This is a result of the segmental coupling patterns from axial rotation. For example, during axial rotation of the cervical spine, the upper three vertebra (C0 to C3) will extend, whereas the lower cervical vertebra (C4 to C7) will flex.²⁹ At the same time, the upper cervical vertebra will laterally bend in an opposite direction of the lower cervical vertebra. The same phenomenon exists in the lumbar spine as well.³⁰ This information will be applicable when the specifics of biomechanics of the nervous system are discussed in relation to canal strains.

Lateral Bending

Lateral bending (R_z) results in an increase in canal length on one side of the canal and a decrease on the opposite side. The axis of rotation for lateral bending is still debatable; however, the current thought is that it lies anterior to the spinal canal closer to the anterior aspect of the vertebral body and perhaps in the sagittal center.²⁷ Regardless of the exact location of the instantaneous axis of rotation (IAR), it is quite obvious that the ipsilateral side of the spine in lateral bending is shortened and under compressive loading whereas the contralateral side lengthens and is subjected to tensile forces. The spinal canal shows similar deformations during lateral bending.^{27,31} However, because of the rotational coupling (R_y) patterns caused by lateral bending, torsional stresses and strains will occur and the magnitudes depend on the segmental level in question. Holmes et al²⁸ measured spinal canal volume in neutral and left/right lateral bending of the lower cervical spines (C2 to C7) of 10 cadavers. The volume of liquid displaced in lateral bending was much less than flexion/extension and only amounted to 0.2 mL.

Combined Postural Loading

It is important to note that the previous material on canal deformations is only for single postures, such as a lateral flexion of the skull compared with a fixed thorax. With combinations of postures, lateral flexion of the skull plus an axial rotation of the skull, the canal deformations may be much larger than simply the combination of the two loads. This is because a shift in the axis of rotation occurs with combined loads. For example, Pope et al³² studied the combined loads of R_y and R_z in the lumbar spine. Here there were no uniform distributions for the IAR; in many instances the IAR was well outside the vertebral body.³² This will be applicable when combined postural loading is discussed in relation to the CNS deformations.

Canal Cross-sectional Area Changes Resulting from the Disk and the Ligamentum Flavum

The cross-sectional area and volume of the spinal canal varies with posture positioning. Fig 4 illustrates that the dynamic sagittal diameter of the spinal canal is measured from the upper crest of the spinous-lamina junction of the inferior vertebra to the posteroinferior body margin of the superior vertebra. These changes are of significance to clinicians when dealing with patients who have canal stenosis. The effects of canal stenosis on neural tissue is well documented.³³⁻³⁸ We do not deny the clinical significance of this issue, nor do we intend to minimize it. However, we believe that an equally important and more prevalent issue has typically been underappreciated by many clinicians and researchers, this issue being neural tissue stress and strain in the absence of stenotic conditions. Because stenotic canals are present in only a minority of individuals, we believe that the latter condition may encompass a larger volume of patients and neurologic conditions. This is because altered alignment of upright posture, viewed as rotations and translations in three dimensions, and poor ergonomics are relatively common place among today's patient populations.^{5,8,39,40} This review is presented with emphasis on this position.

During flexion the spinal canal diameter is increased as the slack is taken out of the soft tissues surrounding the canal. On extension the canal's anterior to posterior width is reduced, but this is usually a small amount, less than 1 mm.²⁸ This reduction is principally due to two mechanical events, deformation of the intervertebral disk (IVD) and deformation of the ligamentum flavum.^{10,27,28} Extension of the spinal column causes the IVD to bulge into the spinal canal, whereas flexion creates tension in the annular fibers posteriorly and compression anteriorly causing an anterior bulge. This bulge is different from the shift of the nucleus pulposus, which in a healthy disk will shift minimally toward the convexity or remain in the neutral position. The annular fibers always bulge into the concavity in a healthy disk. In the lumbar spine, the IVD bulge may approach 1 mm on flexion and extension, whereas in the cervical spine it is significantly less. Obviously, in degenerative conditions and traumatic disk injuries, the strains will be larger.

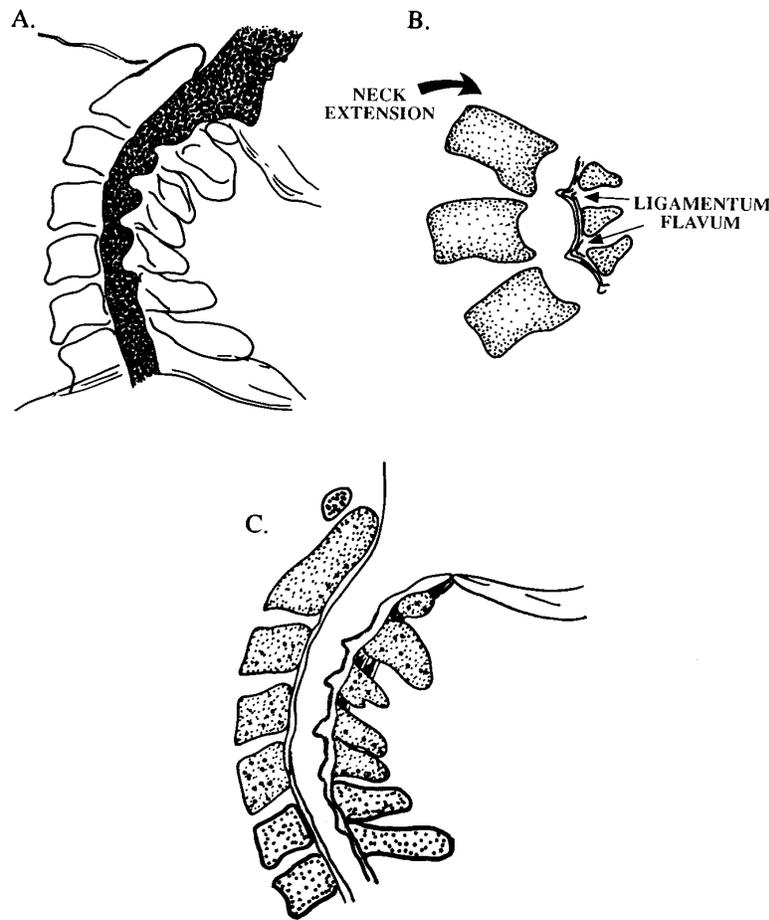


Fig 5. *Folding of the spinal cord in extension. Early myelography research showed posterior indentations in the cord during extension (A). Before Breig's experiments published in 1960,³¹ it was believed that the ligamentum flava was the cause of these bulges into the posterior cord upon extension (B). However, when Breig dissected the ligamentum flava away from the spinal canal, these bulges still existed (C). This led to his discovery that the spinal cord folded on itself in extension.*

The ligamentum flavum may also affect the size of the spinal canal during flexion and extension (Rx).^{10,27,28} The strains in the ligamentum flavum during flexion/extension motions have been measured. In the neutral posture the flaval ligament has a 10% pre-strain; in full extension there is a decrease in length of approximately 13%. This results in a 3% compression, which potentially may bulge into the canal.²⁷ In healthy spines this 3% deformation of the ligamentum flavum and up to 1-mm bulge of the disk on extension are not enough to cause compression of the spinal cord or nerve roots. For instance, Holmes et al²⁸ measured the change in width of the cadaveric cervical spinal canal in flexion and extension. They found the largest reduction in width at C5 and C6, with a magnitude of only 0.7 mm decreasing to 0.4 mm at C3 and C4, which is insignificant. Clinically, this will change in degenerative conditions or in older patient populations. However, the difference from neutral to extension may still only be 1.1 to 1.5 mm and would only compress the spinal cord and nerve roots in the presence of canal stenosis.³³ Originally, it was assumed that the irregular folds or undulations of the dura and cord visualized on extension of the

cervical spine was a pathologic mechanical compression of the spinal cord resulting from the inward bulge of the ligamentum flavum. In contrast, however, several studies have shown that this is normally not the case. A review of these studies is provided for a thorough understanding of the effects that this may have on the neural and vascular structures.

Breig and El-Madi⁴¹ performed gas myelography on patients with cervical myelopathy and spondylosis to visualize the effects of flexion and extension of the cervical spine on the cervical cord. In extension there was a wavy dorsal contour of the dura. This wavy contour was obviously not caused by the folding of the dura over the ligamenta flava. In this reference⁴¹ all cases except one, which was because of arachnoidal adhesions, had a clear air stratum in the subarachnoidal space between the dorsal contour of the cervical cord and that of the cervical canal. Additionally, the ligamentum flavum was dissected away from the spinal canal and the wavy contour or folds of the dura and cord still existed on extension of the cervical spine (Fig 5).³¹

In another study, the cervical spines of 11 cadavers were fixed in an extended position.¹² Bone spurs were present in 8

Table 1. Mean sagittal diameters of the ventral and dorsal arachnoid spaces during 50 degrees of flexion, neutral, and 30 degrees of extension of the cervical spine

Level	Ventral diameter (mm)			Dorsal diameter (mm)		
	F-50°	N	E-30°	F-50°	N	E-30°
C2	4.5	5.1	5.5	2.9	2.4	2.2
C3	3.0	4.0	4.3	2.9	2.3	2.0
C4	2.3	3.6	3.7	3.3	2.3	2.0
C5	2.2	3.5	3.8	3.3	2.1	1.8
C6	2.4	4.2	4.5	3.4	1.8	1.6
C7	3.0	4.6	5.0	3.8	2.3	1.9

F-50°, 50 degrees of flexion; N, neutral; E-30°, 30 degrees of extension.

Adapted from Muhle et al. Biomechanical aspects of the subarachnoid space and cervical cord in healthy individuals examined with kinematic magnetic resonance imaging. Spine 1998;23:556-67.

of the 11 on the anterior surface of the canal. After dissection 14 hours later, deep grooves were present in the anterior surface of the spinal cord and the posterior surface of the cord was humped opposite these grooves. However, no impressions were found on the posterior surface of the spinal cords, which would have indicated contact with the laminae or ligamenta flavum. It was concluded that the anterior grooves were due to the weight of the relaxed cord, caused by the extended neck posture, sagging down as a result of the force of gravity because the spines of the cadavers were fixed in the prone position.

Okumura and Homma⁴² studied the effects of flexion and extension in 7 patients with juvenile compression myelopathy in the cervical spine. Myelography, computed tomography, and magnetic resonance imaging (MRI) in the extended position were negative for cervical cord compression in all of the patients. In contrast, the flexed cervical spine position showed cord compression (actually a combination of tension, compression, and shear), and cervical cord atrophy was localized to this same level.

Houser et al⁴³ evaluated 93 patients with cervical spondylotic stenosis and associated myelopathy by use of computed tomographic myelography. Hypertrophy of the ligamenta flavum was present in roughly 25%. However, displacement of the posterior spinal cord in hyperextension was present in only two cases. This study suggests that approximately 2% of individuals with canal stenosis or 8% of individuals with hypertrophy of the ligamentum flavum may have an abnormal cord compression during hyperextension. The authors concluded, "although occasionally substantial, the hypertrophy of the ligamenta flava did not deform the dorsal aspect of the spinal cord in most patients."⁴³

In the lumbar spine, Penning and Wilmlink⁴⁴ believed that the ligamentum flavum forced the dural sac ventrally in seated lumbar extension and the implications for canal stenosis were discussed. Myelography showed an anterior depression in the dural sac at the levels of L3 to L4, L4 to L5, and L5 to S1 disk spaces, which was believed to be due to a forward bulging pressure from the ligamentum flavum. However, there was no posterior depression of the dural sac from invagination of the ligamentum flavum. This ligament

Table 2. Mean sagittal diameters of the cervical spinal cord during 50 degrees of flexion, neutral, and 30 degrees of extension

Posture	Cord diameter (mm) at different cervical levels					
	C2	C3	C4	C5	C6	C7
F-50°	6.5	6.5	6.5	6.3	5.9	5.7
Neutral	7.2	7.2	7.2	7.0	6.7	6.6
E-30°	8.1	8.0	8.0	7.8	7.7	7.6

F-50°, 50 degrees of flexion; N, neutral; E-30°, 30 degrees of extension.

Adapted from Muhle et al. Biomechanical aspects of the subarachnoid space and cervical cord in healthy individuals examined with kinematic magnetic resonance imaging. Spine 1998;23:556-67.

can invaginate into the canal during lumbar extension, but in extension the thoracolumbar fascia loads the ligamentum flavum in tension, through the interspinous ligament, preventing it from bulging into the spinal canal.⁴⁵ Only with extensive degenerative changes would it be likely for the ligamentum flavum to significantly deform the spinal cord.

Historically, it has been believed that the spinal cord shrinks after death and any minor changes in spinal canal diameter, with the increased size in life, might cause stenosis. However, in a recent study, Fountas et al⁴⁶ used post-myelography computed tomography scanning on 102 live subjects and reported that the diameter of the cervical spinal cord in live subjects is 15% to 20% smaller than in measurements from autopsy data.

Subarachnoid Space

In 1966 Penning and van der Zwaag⁴⁷ reported the occurrence of adaptive changes in the subarachnoid space in flexion/extension. The subarachnoid space was smallest at extension as compared with neutral and flexion, whereas the diameter of the cervical cord decreased at flexion and increased in extension. In 1998 Muhle et al⁴⁸ used MRI in 40 healthy individuals to evaluate functional changes in the cord and subarachnoid space. The sagittal diameter of the dorsal subarachnoid space changed at maximum of 1.6 mm from neutral to flexion at C6. Values for flexion, neutral, and extension at each level are listed in Table 1. It is noted that from neutral to extension, the diameter had a maximum change at C7 of only 0.4 mm. A similar small change was reported for the sagittal diameter of the ventral subarachnoid space in moving from neutral to extension. Table 2 provides the diameter of the cervical spinal cord observed by Muhle et al.⁴⁸ Note that Poisson's effect of narrowing the cross-sectional area during tension is observed in flexion, whereas extension caused a full and relaxed cord with its largest cross-sectional area.

Deformations of the Intervertebral Foramina

The dimensions of the intervertebral foramina (IVF) will also change with sagittal plane posture.^{10,27} Breig¹⁰ showed that the IVF will decrease in vertical height by 33%, and the cross-sectional area of the space will decrease as a result of the impingement from the zygapophyseal joints on extension.

Others have found that the intervertebral space increased by 24% in flexion and decreased by 20% in extension.²⁷

The dimension of the IVF will change during Rz as well. The ipsilateral side decreases in total area, whereas the contralateral side will increase slightly. These changes are not as significant as those observed with flexion and extension. Experiments have shown that one of the main reasons for the reduction in area of the IVF is the bulge of the disk, which occurs in the lateral and posterolateral direction as a result of a combination of lateral bending and compression. However, the magnitude is only 1.5 mm in healthy functional spinal units.²⁷

Stenosis can occur in the IVF, as well as the spinal canal. Humphreys et al⁴⁹ studied cervical IVFs of 43 subjects from 20 to 60 years of age with MRI. Although foraminal heights, widths, and areas were larger in symptom-free subjects than in those with symptoms, they reported that the inferior facet hypertrophy tended to decrease the width of the foramen in aging patients.

Several experiments have reported on the effects of mechanical compression on the dorsal nerve roots. Some of these effects are disturbance of blood flow,^{50,51} change in impulse propagation,⁵²⁻⁵⁴ inflammation,⁵⁵⁻⁵⁷ increase in microvascular permeability,^{58,59} and formation of an intra-neural fibrotic scar.^{60,61} Compression of the dorsal root ganglion has been associated with IVF encroachment. The positions and clinical relevance of the cervical and lumbar dorsal root ganglia have been examined.⁶²⁻⁶⁶

CONCLUSION

It is possible that clinicians have misconstrued the reports of canal narrowing in extension by not reading the extremely small values reported in extension of less than 1 mm of narrowing, which is insignificant. Unless severe pathologic changes are present, slight extension is the preferred position of the spine as far as adverse mechanical stresses and strains in the CNS are concerned.

When one considers the separation of adjacent posterior vertebral elements upon flexion, such results are not surprising. These small changes cannot account for symptoms in these positions. In a point of view in 1998, Dvorak⁶⁷ summed it up best when he stated, "Volume decreases during extension while increasing during flexion, results which are not surprising, do not explain the clinical observation of having more symptoms and signs during flexion (Lhermitte sign) as the authors correctly point out." Thus one must look elsewhere for the cause of neurologic symptoms in the absence of stenosis. This will be addressed in Part II of this review.

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