The pathophysiology of thoracic disc disease

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Nucleus pulposus herniations are far less common in the thoracic spine than at the cervical and lumbar regions. Traditionally, diagnosis of thoracic disc herniations has been challenging because the signs and symptoms are often subtle early in their course. As a result, delays in diagnoses are common. Because they are uncommon as well as difficult to diagnosis, the neurosurgical community has sparse data on which to base good clinical decision making for the treatment of these herniations.

In this review the authors seek to place the phenomenon of thoracic disc disease into the context of its pathophysiology. After a careful evaluation of the available clinical, pathological, and basic science data, a case is made that the cause of nucleus pulposus herniations in the thoracic spine is similar to those occurring in the lumbar and cervical regions. The lower incidence of herniations is ascribed primarily to the reduced allowable flexion at the thoracic level compared with the lumbar and cervical levels. To a lesser extent, the contribution of the ribs to weight-bearing may also play a role.

Further review of clinical data suggests that thoracic disc herniations, like herniated cervical and lumbar discs, may be asymptomatic and may respond to conservative therapy. Similarly, good surgery-related results have been reported for herniated thoracic discs, despite the more challenging nature of the surgical procedure.

The authors conclude that treatment strategies for thoracic disc herniations may logically and appropriately follow those commonly used for the cervical and lumbar levels.

KEY WORDS • thoracic spine • intervertebral disc herniation • degenerative disc disease

Despite the decreased mobility and increased stability of the thoracic compared with the cervical and lumbar spine, it is still subject to the overall stresses applied to the spine as well as the processes of aging. Although less common than in the cervical or lumbar region, cases of degenerative disc disease are seen in the thoracic spine, and the resulting morbidity can be significant (Fig. 1). As in other spinal levels, this degeneration can manifest as osteophyte formation or disc herniation. Radicular pain, back pain, long track signs, spasticity, and bowel or bladder dysfunction are all common manifestations of thoracic disc disease.

Knowledge of the anatomy and physiology of the normal intervertebral disc serves as the basis for understanding the pathophysiology of degenerative disc disease. Ultimately this knowledge and understanding provides the necessary foundation for the appropriate management of thoracic disc disease.

ANATOMY AND PHYSIOLOGY OF THE DISC INTERSPACE

Embryological Development

The intervertebral disc can be divided into two main constituent parts: the nucleus pulposus and the annulus fibrosus.15 These two structures can be traced back to the early development of the human embryo. The nucleus pulposus is derived from the notocord, and this primitive structure appears at approximately Day 19 of development, including the formation of the vertebral column. Ultimately the notocord involutes as the VBs form. A small remnant, however, does remain and ultimately forms a portion of the nucleus pulposus. The cells of the notocord persist until after birth but gradually undergo a mucoid degeneration. This as well as mucogelatinous degenerative material of the inner fibrocartilage of the disc is generally thought to contribute to the very gelatinous matrix of the nucleus pulposus observed at birth. In general, the notocordal remnants are more prevalent at the high cervical and low lumbar regions of the spine. This likely represents another reason why thoracic disc disease is relatively less common than other forms.

The annulus fibrosus forms from a dense concentration of mesenchymal cells derived from the sclerotome of the somites. The somites are segmented structures that appear in the embryo at approximately Day 20 of development. Mesenchymal cells of the somites differentiate into the dermatome, myotome, and sclerotome. As their names imply, the first two groups develop into dermal and muscle tissue, respectively. The sclerotome ultimately forms all of the muscle, cartilage, and connective tissue of the spine, and it migrates over the dorsal aspect of the neural tube, into the body wall, and around the notocord. These three groups of cells then give rise to the vertebral arch, the ribs, and the anterior vertebral structures, respectively.
The more caudal portion of each somite ultimately forms the VB, whereas the more cranial portion forms the annulus fibrosus. Initially these cells have little structure and are rounded in appearance. As development proceeds, the cells become elongated and arranged in concentric layers. Each concentric layer is oriented obliquely to the layers above and below and runs completely between the two adjacent VBs (Fig. 2). This development is completed prior to birth and is thus not influenced by movement or gravitational stress.

**Anatomical Features**

The thoracic spine plays an integral role in the support provided by the axial skeleton. In addition to resisting gravity, the spine at this level allows for movement, primarily via flexion and extension. Spinal stability is provided by both the bone structure of the VB and by the joints. Like the cervical and lumbar regions, the thoracic spine has two types of joints. The intervertebral discs are synovial joints, meaning they have no synovial membrane. The facet or zygapophysial joints, in contrast, are lined by a synovial membrane and are termed diarthrodial joints, or synovial joints. Unlike the lumbar and cervical spine, however, the thoracic spine also shares articular surfaces with the ribs at each level, which are also diarthrodial. These diarthrodial joints offer movement with minimal resistance. In contrast, the intervertebral disc primarily plays a that of load-bearing and shock-absorption role, although it also allows for movement despite its lack of a synovial membrane. Both the facet and costovertebral joints do add some stability to the entire spinal region as well. The costovertebral joints, however, also limit the overall flexion of the spine at the thoracic levels. Some authors have postulated that a decrease in flexion would be expected to result in an overall decrease in disc disease at the thoracic levels.

The thoracic spinal cord is approximately 6.5 mm deep and 8.0 mm wide, whereas the thoracic spinal canal is approximately 16.8 mm deep and 17.2 mm wide. The canal does widen distally, and as a result, there is a minimal clearance space of 9.2 mm laterally whereas there is 10.3 mm of clearance in the AP direction. In comparison, cervical spine clearance space is 11.3 mm laterally and 7.0 mm in the AP plane.

At the time of birth, the nucleus pulposus and annulus fibrosus are already present and functional as the intervertebral disc. The disc itself is a flattened and cylindrical structure. The combined disc height represents approximately 25% of the overall height of the vertebral column, although it represents only approximately 20% at the thoracic spine. The discs are nearly equal in height anteriorly and posteriorly in the thoracic spine. This means that the characteristic thoracic kyphosis is almost entirely the result of a difference in height of the VBs. In fact, the thoracic VBs are typically 1 to 2 mm greater in height posteriorly. The ALL forms the anterior border of the disc where its deepest fibers merge with the fibers of the annulus. This ligament can be found adjacent to the discs and VBs from the atlas to the sacrum. The deeper fibers connect disc to VB, whereas the more superficial fibers run over and connect up to five vertebral levels. Thus, although the ALL is thick, it is not completely continuous. The PLL, which forms the posterior border of the disc, is often thin in comparison to the ALL. It is, however, continuous. In the thoracic region, the PLL is wider over the disc and thinner over the VB (Fig. 3).

The outer annulus makes up approximately 60% of the disc volume, whereas the inner and slightly posteriorly directed nucleus pulposus makes up the remainder. Above and below this are the cartilaginous endplates. These thin layers of hyaline cartilage form the inferior and superior surfaces of the VBs. The lamina cribosa connects these layers to the VBs and, as its name suggests, contains fine pores through which nutrients may diffuse. The fibrocartilage of the annulus connects to the cartilaginous endplates in all but the outermost layer. This layer, known as Sharpey fibers or perforating fibers, attaches directly to the epiphyses of the VBs. The annulus tends to be thicker anteriorly and thinner posteriorly.

The inner nucleus consists of cartilage cells, physaliphorous cells, loose connective tissue, and notocordal remnants. In early life these notocordal cells may be identified, but with aging they become more fibrous and resemble chondrocytes or fibroblasts. There is a high water content in the disc at birth, and this gradually dissipates and is replaced with dense connective tissue. Although the
disc has a blood supply at birth, this involutes over the course of the first 1 to 2 years of life. Likewise, there is no innervation of the interior portion of the adult disc.9

The sinuvertebral nerves (or meningeal branch of the spinal nerves) are thought to provide innervation of the outer portion of the disc.9 This nerve is given off by the gray ramus communicans shortly after the ramus arises from the spinal nerve. The nerve courses medially, through the intervertebral foramen back into the spinal canal, where it turns in a cranial direction, around the pedicle, and then moves further medially to run along the PLL (Fig. 4). The nerve provides innervation for the annular fibers of the posterior portion of the disc, the PLL, the epidural blood vessels, the dura mater, and the periosteum. As a result, it is logically assumed to be important in the production of back pain.11 The anterior portion of the disc is supplied directly by the gray ramus, as is the ALL.5

Biochemical Features

Common molecules found within the annulus fibrosus and the nucleus pulposus include proteoglycans and collagen types I and II.5 Collagen is actually a family of related connective tissue proteins, the name of which is derived from Greek, meaning to produce glue. All varieties of collagen share a triple helix configuration with substantial cross-linkage, which leads to a very high tensile strength. Approximately one third of collagen is composed of glycine, because it is seen regularly at every third amino acid residue. Proline is also highly represented whereas two other amino acids seen in collagen, hydroxyproline and hydroxylysine, are virtually absent form all other proteins. The three individual alpha helices of the triple helix are arranged in a left-handed fashion and are tightly wound. This tightness is enhanced by the high concentration of glycine, the smallest amino acid.19 The tightness of the helix also enhances the strength of the fiber by allowing increased hydrogen-bond formation between adjacent residues within the helix. The types of collagen are determined by the makeup of these individual alpha helices, which are then arranged into a right-handed super helix. The strands are referred to as tropocollagen which can be as long as 300 nm, making collagen one of the longest proteins known. The tropocollagen is then arranged into fibrils with additional disulfide bonds between them. As these fibrils are assembled into connective tissue, the strength increases dramatically. To break a fiber with a 1 mm diameter requires the application of at least 10 kg of force.19 Type I collagen is found in bones and tendon and is primarily present in the annulus fibrosus. Type II collagen is more commonly present in articular cartilage and is the collagen type seen almost exclusively in the nucleus pulposus.

Proteoglycans are formed from a combination of polysaccharides and proteins, the polysaccharides being much more prevalent. Most proteoglycans are composed by the attachment of a central core of hyaluronic acid to various side chains of glycosaminoglycans. The most common glycosaminoglycans in the nucleus pulposus are chondroitin sulfate and keratin sulfate (Fig. 5).19 These molecules contain negatively charged acidic groups. In the case of keratin sulfate there is one net negative charge, whereas chondroitin sulfate has a net negative charge of two. Such negative charges attract small cations such as sodium, calcium, and magnesium to remain electrically neutral. As a result, higher concentrations of these cations are required in the disc space, compared with plasma. As these cations move into the disc matrix, a significant osmotic gradient is created, causing water to move into the disc matrix as well. The overall result is that the disc swells.5

Physiological Characteristics

The motion of the thoracic spine is limited by a number of factors. The presence of the rib cage tends to decrease movement in all directions. The attachment of the ribs to
the sternum and their articulation with the VBs both serve to limit rotation and lateral bending of the thoracic spine. As previously discussed, costovertebral articulation also limits flexion of the thoracic spine. In addition, the relatively small size of the thoracic intervertebral discs themselves decreases the overall mobility of the spine at this level. This underscores the role of the disc as a joint and its importance to movement in general.

The intervertebral disc functions primarily to absorb the force of shock and dissipate the compressive load applied to the spinal column by gravity. The disc achieves this function by converting the vertical pressure to a horizontal thrust. The elastic nature of the annulus fibrosus allows for an increase in the diameter of the disc (Fig. 6). An increase of 0.5 mm has been measured in the annulus when a 50-kg load is applied and 0.75 mm when a 100-kg load is applied. This relatively small amount of movement in the horizontal plane helps provide spinal stability.

The response of the nucleus pulposus is more complex. As previously described, water is drawn oncotically to the matrix of the nucleus pulposus and is in a dynamic fluid equilibrium with the plasma. This can be depicted mathematically by the following equation:

\[ \text{extradiscal hydrostatic pressure} + \text{intradiscal oncotic pressure} = \text{intradiscal hydrostatic pressure} + \text{extradiscal oncotic pressure}. \]

Increased hydrostatic pressure forces this equilibrium to favor loss of water from the matrix to the plasma. At rest, the biochemical composition of the matrix, especially the proteoglycans and glycosaminoglycans, attracts water, causing the disc to swell. These forces equilibrate completely at 800 kPa. Thus, during recumbency, when the force on the spine is less than 800 kPa, water flows into the disc leading to an increase in disc height. This event can actually be demonstrated by an increase in stature immediately upon waking. Likewise, when assuming an erect position, the force on the disc space greatly exceeds 800 kPa, and water is forced out of the disc to compensate for the increased compressive force.

**PATHOGENESIS OF DISC DEGENERATION AND SPONDYLOSIS**

Degenerative disc disease may be best thought of as a failure on the part of the species to evolve the necessary adaptations to make erect posture more favorable. Despite the large number of primates capable of a bipedal posture, humans assume it preferentially and in a prolonged fashion. Moreover, it is achieved with the knees extended, which prevents these joints from possibly aiding in load reduction. Consequently, all stress is placed on the spine, and there is a dearth of structures available to participate in load reduction. In fact, the thoracic region is the only portion of the spine where there are additional structures available to aid in weight bearing—namely the ribs. This may help, once again, to explain the decreased incidence of disc disease at these levels. Nevertheless, the degenerative process affects all levels of the spine with a conserved pathophysiology.

**Effects of Dehydration**

Given the significant role that water plays in the dissipation of compressive force on the disc space, it logically follows that loss of water would lead to significant pathological changes. Indeed, the water content of the disc is nearly 90% in early childhood and decreases to less than 70% by the eighth decade. This decrease in matrix water content may partially be explained by a change in the biochemical composition of the matrix itself. Aging is associated with an increase in the deposition of insoluble collagen within the disc matrix, and this decreases the gelatinous nature of the nucleus pulposus as well as the elastic nature of the annulus fibrosus. As the annulus becomes less elastic, the frequency of tears within it increases. The frequency varies directly with age.
Anatomical considerations may also contribute to decreased hydration of the matrix. Although the disc has a vascular supply at birth, this gradually involutes until it disappears at approximately the 2nd year. 9\textsuperscript{,}13 Because this corresponds to the developmental point at which a child assumes an erect posture, it has been suggested that the loss of vascular supply is secondary to the increased stress applied to the disc. Regardless, the overall result is that the chondrocytes of the disc space rely exclusively on diffusion for the provision of metabolic substrates, resulting in an overall decrease in access. 15\textsuperscript{,}16 This relationship is then exacerbated by a concomitant decrease in the size of pores available for diffusion in the lamina cribosa.

Perhaps a more significant contributor to the decrease in matrix water is a decrease in the overall amount of proteoglycans as well as a change in the ratio of chondroitin sulfate to keratin sulfate.\textsuperscript{9} Keratin sulfate, with only one net negative charge, tends to increase as compared with chondroitin sulfate, which has two.\textsuperscript{19} Because it attracts fewer small cations, the osmotic gradient into the disc is decreased and subsequently the overall water content is reduced as well. With decreased water content, the disc loses height and some ability to expand. As a result, more of the load is borne by the annulus fibrosus. This, in turn, increases the likelihood of injury to the annulus and the overall rate of its degeneration. The disc’s ability to expand does not begin to decrease until the fourth decade. Later in life significant decreases in the expansile nature of the nucleus actually decrease the incidence of herniation.

It is likely that coincident findings cause the incidence of all disc herniations, including those occurring in the thoracic spine, to peak during the fourth and fifth decades of life. It is at this time that the intervertebral discs experience a slight decrease in nuclear expansion, causing increased stress on the annulus fibrosus, and the decreased elasticity of the annulus makes it more susceptible to injury. An increase in annular tears coupled with a persistently expansible nucleus then results in a higher incidence of disc herniation (Fig. 7).\textsuperscript{5\textsuperscript{,}14} This would be especially true for active individuals who place additional loads on the spine.

**Disc Deformation**

Herniation of the nucleus pulposus must be caused by a combination of high pressure within the nucleus and weakness of the annulus fibrosus. With the aforementioned decrease in water content, the ability of the nucleus to dissipate downward-moving pressure decreases, and the intradiscal pressure rises in response to axial loading. This pressure will be transmitted, in turn, to the annulus, whereas the nucleus itself will migrate away the point at which where the pressure is applied. This event will not necessarily cause a disc herniation on its own but may lead to a disc bulge.

Logically, the annulus fibrosus is more likely to become disrupted in areas where it is thinner or has less reinforcement. The slightly eccentric placement of the nucleus pulposus posteriorly previously discussed results in the annulus being thinner posteriorly. In addition, the PLL is thicker at the midline and thinner toward the lateral aspects of the disc. The expectation that posterolateral disc herniations would be most common has been borne out in repeated clinical observations, although many thoracic discs have been described as “centrolateral” in their location. 4\textsuperscript{,}7\textsuperscript{,}18

### NATURAL HISTORY OF THORACIC DISC DISEASE

**Incidence of Thoracic Disc Disease**

Thoracic disc herniations are certainly rare in comparison with either cervical or lumbar disc herniations. The incidence of thoracic disc herniation is estimated to be from 0.25 to 1\% of all disc herniations. Analysis of population studies suggests that the overall incidence of thoracic disc herniations is approximately 1 per 1,000,000 patient years. 5\textsuperscript{,}7\textsuperscript{,}18 There is approximately a 1:1 male/female ratio in presentation. The majority of thoracic herniations have been noted to be central or centrolateral, with a minority of herniations truly lateral. 4\textsuperscript{,}13 Many herniated thoracic discs are shown to be calcified at presentation (Fig. 8). Despite studies in which calcification is reported to occur in 30 to 70\% of cases of thoracic disc herniations, the cause of this phenomenon remains unclear. Calcification is an important consideration, however, because approximately 5 to 10\% of calcified discs are associated with intradural extension.\textsuperscript{18}
The authors of some studies have shown that there is a tendency for thoracic disc herniations to be multiple in nature and that they are commonly identified in patients in whom herniated discs are present at either the cervical or lumbar levels as well.\(^3,4\) The majority of thoracic herniations are seen during the third to fifth decades, a finding that mirrors the age range in which cervical and lumbar disc herniations are found as well.\(^4,7,18\)

The vast majority of herniated thoracic discs are found below the level of T-7. Interestingly this corresponds to the last level of individually fused ribs. The eighth, ninth, and 10th ribs are joined to the sternum via fused cartilage, whereas the 11th and 12th ribs are “floating” without any attachment to the sternum, and this is thought to make these vertebral levels more flexible than the higher thoracic levels.\(^20\) The greater degree of flexion allowable at each level should, indeed, predict a higher incidence of nucleus pulposus herniations. The graphs in Fig. 9 show the allowable degrees of flexion at each vertebral level as well as the incidence of nucleus pulposus herniation at each vertebral level. The increase in incidence of herniated discs demonstrated at the more mobile (lower) thoracic vertebral levels lends further support to the theory of a common pathophysiology for disc herniations at all levels.

**Presentation of Thoracic Disc Herniations**

Although early reports on thoracic disc herniations focused on a traumatic cause our discussion of pathophysiology thus far suggests that this is not necessarily the primary cause. Careful reviews of the literature have supported this view, suggesting that traumatic injury is associated with approximately 25% of cases of such herniations.

In a 1998 paper in which they discussed surgery-related outcomes, Stillerman, et al.,\(^18\) divided the presentation of thoracic disc herniations into three distinct symptom complexes: localized thoracolumbar pain, radicular pain, and myelopathy. Their classification was based on the frequency of presenting symptoms and the response of these symptoms to surgical intervention. The most common presenting symptom, occurring in 76% of their patients, was pain; second most common was myelopathy. Despite the fact that many of the patients did present with pain, it is equally interesting to note that nearly 25% did not present with any symptomatic pain.

Less common but associated symptoms include bladder dysfunction (24%), sensory impairment (61%), and motor deficits (61%). The most common characterization of bladder dysfunction was urgency. Sensory disturbances usually consisted of paresthesias, dysesthesias, or complete sensory loss. If there was motor impairment, it was three times more likely to be a paraparesis than a monoparesis (Stillerman and Chen).

**Herniated Disc–Related Pain**

The perception of pain remains poorly understood. In classic reports the authors describe the presence of unmyelinated, free nerve root endings seen in many structures surrounding the intervertebral disc.\(^9,11\) These structures include the ALL and PLL, the synovial capsule, the vertebral periosteum, the ligamentum flavum, and the supra- and interspinous ligaments. Such fibers are also seen in the outermost layer of the annulus fibrosus but are not found in the deeper layers or within the nucleus pulposus.

Experimental studies have been performed to help elucidate the origin of pain. The injection of hypertonic saline has been shown to cause pain similar to back pain when injected into the ligamentum flavum, the facet joints, the interspinous ligaments, and the disc proper.\(^11\) In one study, Compere and Cloward\(^8\) induced pain by pinching the
annulus or ALL. Smyth and Wright showed that pain is induced by applying traction to the nerve root. This latter experiment supported the notion that some degree of distortion or injury, perhaps leading to inflammation, plays a role in the production of pain. Conversely, others, including Fisher, have noted that the application of pressure on a nerve commonly produces numbness or paresthesias but rarely produces pain. In addition, it is known that asymptomatic patients may harbor radiographically detectable disc herniations particularly in the thoracic region. Furthermore, in approximately 38% of patients treated successfully by conservative means no radiographic change or even worsening of their disc herniation will be demonstrated.

Regardless of the controversy surrounding the cause of pain, the spinal nerves themselves travel out through the intervertebral foramina in close proximity to the disc space. Spinal nerves are, therefore, likely candidates for compressive lesions. Within the intervertebral foramen, the nerve root takes up virtually all of the AP space but does leave the cranial one third open for the passage of vascular structures and areolar tissue. Consequently, any space-occupying lesion that takes up any of the AP diameter may very well be symptomatic. Each nerve in the thoracic spine, however, does travel approximately one level caudally prior to exiting through the foramen. Thus, a central protrusion is more likely to cause symptoms in one neurological level lower, whereas a lateral protrusion may affect the nerve directly.

Stillerman, et al. divided the pain reported at presentation into three categories: localized thoracic back pain, axial pain radiating down the spine, and radicular pain. Greater than 90% of their patients with axial and radicular pain patterns responded to surgery, whereas 82% of those with localized back pain responded to surgery.

Long-Term Outcomes

Much like those in the cervical and lumbar regions, thoracic discs may herniate without causing any symptomatology at all. Similarly, thoracic disc herniations may respond to conservative measures and never require any surgical intervention. In 1991 Awwad, et al., demonstrated that asymptomatic thoracic disc herniations were a common finding in patients undergoing myelography for symptoms at the cervical or lumbar levels. On 433 myelograms, they identified 54 asymptomatic thoracic disc herniations in 40 patients. This represented 9.2% of the patients, which they believed was an underestimation of the true incidence because not all of the thoracic levels were examined. They estimated the true incidence to be 11.1%, although they noted that the population could be unrepresentative, as degenerative disease of the spine at other levels had at least been suspected in these patients. Nevertheless, the study does demonstrate that asymptomatic disc herniations can be common.

In a retrospective study, Brown, et al., demonstrated that herniated thoracic discs do respond to conservative measures. Thoracic disc herniations were identified in 55 patients, 40 of whom underwent conservative therapy alone. Of these, baseline level of function was recovered in 77%. Of note, the authors of neither the former nor the latter study could correlate outcome with preoperative radiographic appearance. Brown, et al., did observe that in the majority of the 15 patients who ultimately underwent surgery the herniated discs were located below the T-9 level, whereas in the majority of those in the conservative therapy group the herniations were between T-6 and T-9. This led the authors to speculate that more distal herniations are more likely to yield lower-extremity symptoms and hence require surgical intervention, as vertebral levels demonstrate greater flexion.

Surgical intervention has been thoroughly discussed by Stillerman, et al. In their report in which they demonstrated that their findings were comparable with other modern reports on the surgical treatment of thoracic disc herniations, they attained excellent clinical outcomes. Myelopathic symptoms resolved in over 95% of cases whereas pain resolved in 87%. Resolution of bladder dysfunction and motor weakness was less robust; the former improved in 76% of patients, whereas the latter improved in only 58%. The overall complication rate was 12.6%; the rate of major complications in this series made up only 3.6%. These results compare well with the overall 6.1% rate of major complications demonstrated in other series. Nevertheless, this complication rate highlights the need to undertake surgical intervention judiciously, especially in light of the good results obtained with nonoperative management in the aforementioned reports.

CONCLUSIONS

This review of the anatomical, physiological, and pathological features of the nucleus pulposus in the thoracic region, as well as the pathophysiology of disc herniations in this region, allows for several conclusions. First, the structure of the nucleus pulposus is similar in all regions of the spine. It is the nature of this structure that leads to disc degeneration and herniation in a well-defined group of patients, most commonly those in the third to fifth decade of life. Second, the allowable movement, and more specifically flexion, at each individual vertebral level appears to correlate well with the incidence of disc herniation located at that level, an observation that suggests that flexion is a significant factor in the development of disc herniations at all levels. This also provides an excellent explanation for the significantly decreased incidence of disc herniations seen at the thoracic level: the thoracic spine has much less allowable flexion because of the presence of the ribs. Finally, appropriate clinical decision making in the management of herniated thoracic discs should be individualized but can be based on the same principles as those applied when treating cervical and lumbar disc herniations. The decreased incidence of thoracic disc herniations does not indicate a different pathological phenomenon but is rather a manifestation of similar pathological mechanisms seen at other spinal levels.

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