ROTHMAN—SIMEONE

# THE SPINE

# THE SPINE

## Volume II

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1975

W. B. SAUNDERS COMPANY
*Philadelphia • London • Toronto*

W. B. Saunders Company: West Washington Square
Philadelphia, PA 19105

12 Dyott Street
London, WC1A 1DB

833 Oxford Street
Toronto, Ontario M8Z 5T9, Canada

The Spine—*Volume II*

ISBN 0-7216-7717-7



Last digit is the print number: 9 8 7 6 5 4 3 2 1

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# Lumbar Disc Disease

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## INTRODUCTION

Back pain has plagued man for many thousands of years. Descriptions of lumbago and sciatica are found in the Bible and in the writings of Hippocrates. Despite the long history of awareness of this problem, a reasonable and scientific explanation of the source of low back and leg pain did not emerge until 1934 with the publication of the classic paper by Mixter and Barr.[78] These investigators for the first time delineated prolapse of the intervertebral disc as the etiologic agent in the production of these symptoms. It is commonly acknowledged today that derangements of the intervertebral disc represent the great majority of cases of back pain and sciatica.

Human disease assumes importance as a cause of either death or disability. Degenerative disease of the spine, for all intents and purposes, is a nonlethal entity, and its priority must rest on a determination of its prevalence in the population and its impact on this population in terms of pain and disability.

## INCIDENCE

In Sweden, each member of the National Health Insurance, in order to receive compensation, reports his or her illness by telephone to a central bureau. Thus, excellent statistics are readily available in terms of population analysis. Back pain has been reported in 53 per cent of light workers and 64 per cent of those engaged in heavy labor.[60] An extensive investigation by Horal showed that low back pain of a significant degree begins in the younger age groups, with a mean age of onset of 35 years.[58] Of the individuals complaining of low back pain, only 35 per cent will develop sciatica. After subsidence of the original attack of low back pain, 90 per cent have a future recurrence. The recurrent attacks tend to be more prolonged than the original episode. It is of interest that 50 per cent of individuals in this study of the population with low back pain also had cervical spine discomfort but that the age of onset was, on the average, 6 years later. Twenty per cent also had pain in the thoracic spine. It would appear that at least half of the adult population at some time in their lives is incapacitated with low back pain secondary to degenerative disc disease.

A clinical and radiographic survey of the British town of Leigh revealed that in males between the ages of 55 and 64 years, 83 per cent showed evidence of significant lumbar disc degeneration.

Several excellent reviews on the impact of spine disease on industrial disability are found in the medical literature. The Department of Labor and Industry of the State of Washington carefully analyzed its experience in 1965 and was able to come to the following conclusions: 24 per cent of days lost from work were due to low back disorders. Of those individuals who were out of work for greater than six months, only 50 per cent ultimately

returned to work. Of those off work for longer than one year, only 25 per cent ultimately returned to work. Although the average industrial claim was 25.7 days, the average days lost per back claim were 123. The implication of these figures is obvious and has been repeated in every major analysis of industrial back disease.

## NATURAL HISTORY

Intelligent treatment of lumbar disc degeneration must be predicated on a thorough knowledge of the natural history of this disorder. If this information is not available to the treating physician and to the patient, they will be unable to honestly and effectively make the decisions necessary in the management of this disorder. All too often, decisions either for or against surgical intervention are based on distorted concepts of disc disease. A group of 583 patients were studied at the Karolinska Institute after their first attack of sciatica. Surgery was undertaken in 28 per cent of the group, and both the operative and nonoperative patients were followed for an average of seven years.[46] The results of this study indicated that the acute episodes of sciatica ran a relatively brief course in most cases, regardless of whether the treatment was conservative or surgical. However, the subacute or chronic symptoms secondary to disc degeneration, although less dramatic, were prolonged and had a profound effect on the patient's life. At the end of the follow-up period approximately 15 per cent of the conservatively treated group continued to have a reduced working capacity, a restriction in leisure activities and regular sleep disturbances. Twenty per cent of the conservatively treated group continued to have pronounced residual sciatica. Colonna studied a group of 28 patients with radiographically proved disc herniations for a period of up to eight years.[15] Fifty-seven per cent of the patients had intermittent disability for five years or longer. Pearce has reported a group of 91 patients with a diagnosis of lumbar disc protrusion who were followed conservatively.[83] At the time of follow-up evaluation eight years later, 26.5 per cent were free of pain, 41 per cent were troubled by mild pain and 32.4 per cent were having minor to severe pain.

Disability from lumbar disc disease must be considered in terms of back and leg pain with its attendant limitation of function. Neurologic deficits including motor weakness, although helpful diagnostically, are not necessarily compelling surgical factors, since residual weakness is not markedly different in patients treated operatively or conservatively.[46] Bowel and bladder dysfunction affects a relatively small percentage of patients but assumes greater significance in terms of surgical urgency.

With this background, the treating physician and his patient must take their decision for the role of surgery. If, after careful diagnostic evaluation, a firm diagnosis can be established, and a concerted course of conservative treatment has failed, and the treating surgeon feels that his operative intervention would with certainty shorten the disease process, then surgery can be recommended.

## PATHOLOGY OF THE LUMBAR INTERVERTEBRAL DISC

The biochemical, autoimmune and genetic factors in disc degeneration have been discussed in detail in Chapter 7, and the reader is referred to that section for a discussion of those aspects of lumbar disc disease. The lumbar intervertebral disc undergoes a type of degeneration which is different in many respects from the pattern found in the cervical spine. Chronic cervical disc degeneration is the major cause of neck pain and radiculitis. Herniations of soft nuclear material are the exception rather than the rule. In the lumbar spine herniations of soft disc material are frequent and, indeed, are the most common cause of acute sciatica in conjunction with low back pain. This is not to say that acute disc herniations do not occur in the cervical area or that chronic disc degeneration cannot produce sciatica, but that it is not the usual case.

In both the cervical and lumbar areas, disc degeneration is not usually due to one major traumatic insult. Approximately 25 per cent of patients presenting with back pain and sciatica will give a history of trauma. Injury will most often play a precipitating role in what is truly a chronic degenerative process.

### Ballooned Disc

Ballooning of the intervertebral disc is characteristically seen in association with dis-

eases which weaken the body of the vertebra. Classically, osteoporosis, more accurately termed osteopenia, will sufficiently weaken the vertebral body to allow expansion of the intervertebral disc into the upper and lower plates of the body. For this to occur the disc must still have its elastic gelatinous nucleus.[17] If the disc has lost its integrity, collapse and wedging of the vertebral body will occur rather than ballooning of the disc. Malignant processes such as multiple myeloma will produce sufficient weakness of the vertebral body to allow this ballooning to take place (Fig. 9–1).

Figure 9–1. X-ray of the lumbar spine revealing severe osteoporosis with ballooning of multiple discs into the softened adjacent vertebral body.

## Intra-Spongy Nuclear Herniations

**(Schmorl's Node)**

It has been noted for over 100 years that the intervertebral disc can herniate through the cartilaginous end plate into the cancellous bone of the vertebral body. This herniation of disc material takes place through a defect in the cartilaginous plate which may represent the point of passage of blood vessels from the body of the vertebra to the disc during early life. These herniations are irregular in size and shape, and ultimately will be surrounded by a rim of bony sclerosis. These defects are seen throughout all adult life. The adjacent disc space frequently exhibits thinning (Fig. 9–2).

Figure 9–2. X-ray of the lumbar spine illustrating multiple intra-spongy nuclear herniations into the central portion of the adjacent end plate of the vertebral body.

## Osteophyte Formation

Peripheral osteophyte formations, anteriorly and laterally and to a lesser extent posteriorly, are often found in the bodies of lumbar vertebrae associated with disc degeneration. These osteophytes represent pathologic stimulation of new bone formation at the attachment of the longitudinal ligaments of the annulus to the bodies. This stimulation may be due to hypermobility of the vertebral body or abnormal distribution of stresses on the annulus and ligaments associated with degeneration of the intervertebral disc. MacNab has

differentiated the traction spur from other osteophytes.[72] It is horizontally directed and arises two millimeters away from the discal border of the anterior and lateral surfaces of the vertebral body. MacNab feels that it denotes segmental instability. The decreased incidence of posterior osteophytes as compared to those in the anterior and lateral position may be explained by the absence of a strong attachment of the posterior longitudinal ligament to bone. This osteophyte formation in association with disc degeneration has often been termed lumbar spondylosis, which has its counterpart in cervical spondylosis.

## Thinned Discs

Three distinct situations are present which may be associated with thinning of the disc space. The first situation occurs in the presence of a transitional fifth lumbar vertebra (Fig. 9–3). Between the transitional vertebra and the sacrum, a vestigial disc is often found which is devoid of a nucleus pulposus.[48] This loss of height and disc space is not usually the product of degenerative changes. When the transverse process of this transitional vertebra is incompletely sacralized on one side and normal on the other it is possible that the caudal intervertebral disc may be degenerated. More often there is a broad sacralization of the transverse processes, either unilaterally or bilaterally. In these situations one should look for disc degeneration above this transitional vertebra rather than below it.

A second group of thinned intervertebral discs is associated with the degeneration of the nucleus pulposus, with or without herniation. This mechanical loss of disc material is often associated with invasion of granulation tissue through rents in the annulus. Progressive dehydration will contribute markedly to the diminished volume of the disc.

Disc thinning may also occur subsequent to infection of the disc space, and is more completely discussed in Chapter 12, Infections of the Spine.

## Disc Protrusion

The process of nuclear herniation and annular protrusion is caused by a combination of biochemical factors previously discussed, chronic degenerative structural changes and superimposed mechanical stress. The pathologic cycle has been described in detail by Armstrong.[6] Prior to actual displacement of disc material, the nucleus and annulus undergo certain well-defined structural changes. Radiating cracks in the annulus fibrosus develop in the most centrally situated lamellae and extend outward toward the periphery.[53] These radiating clefts in the annulus weaken its resistance to nuclear herniation. If they are

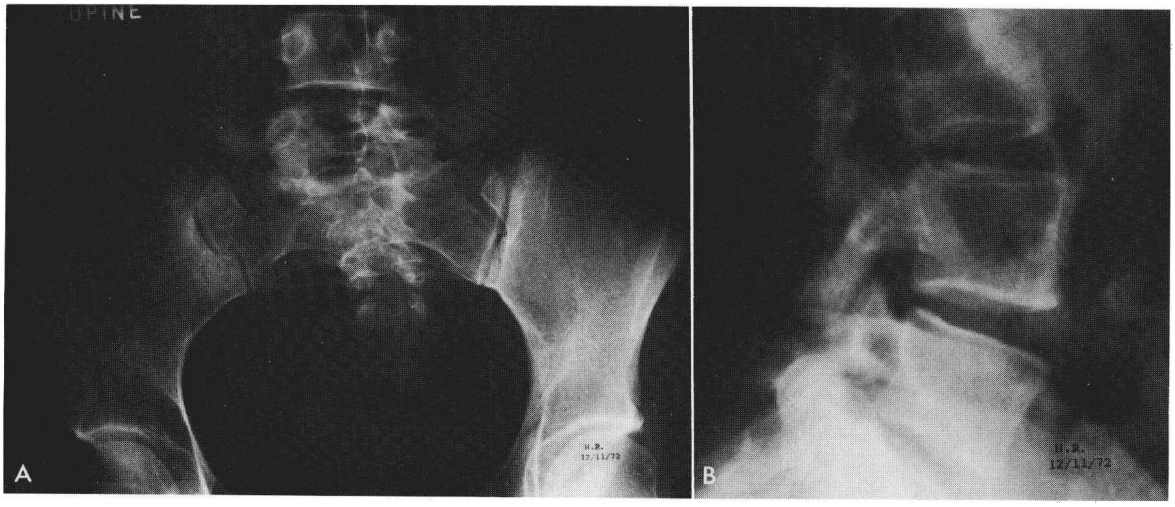


**Figure 9–3.** *A*, Anterior posterior X-ray of the lumbar spine reveals a transitional fifth lumbar vertebra. *B*, Lateral view of the same patient reveals loss of height of the disc space between the transitional vertebra and the sacrum. This is not due to disc degeneration but rather to the congenital variation of the transitional vertebra.

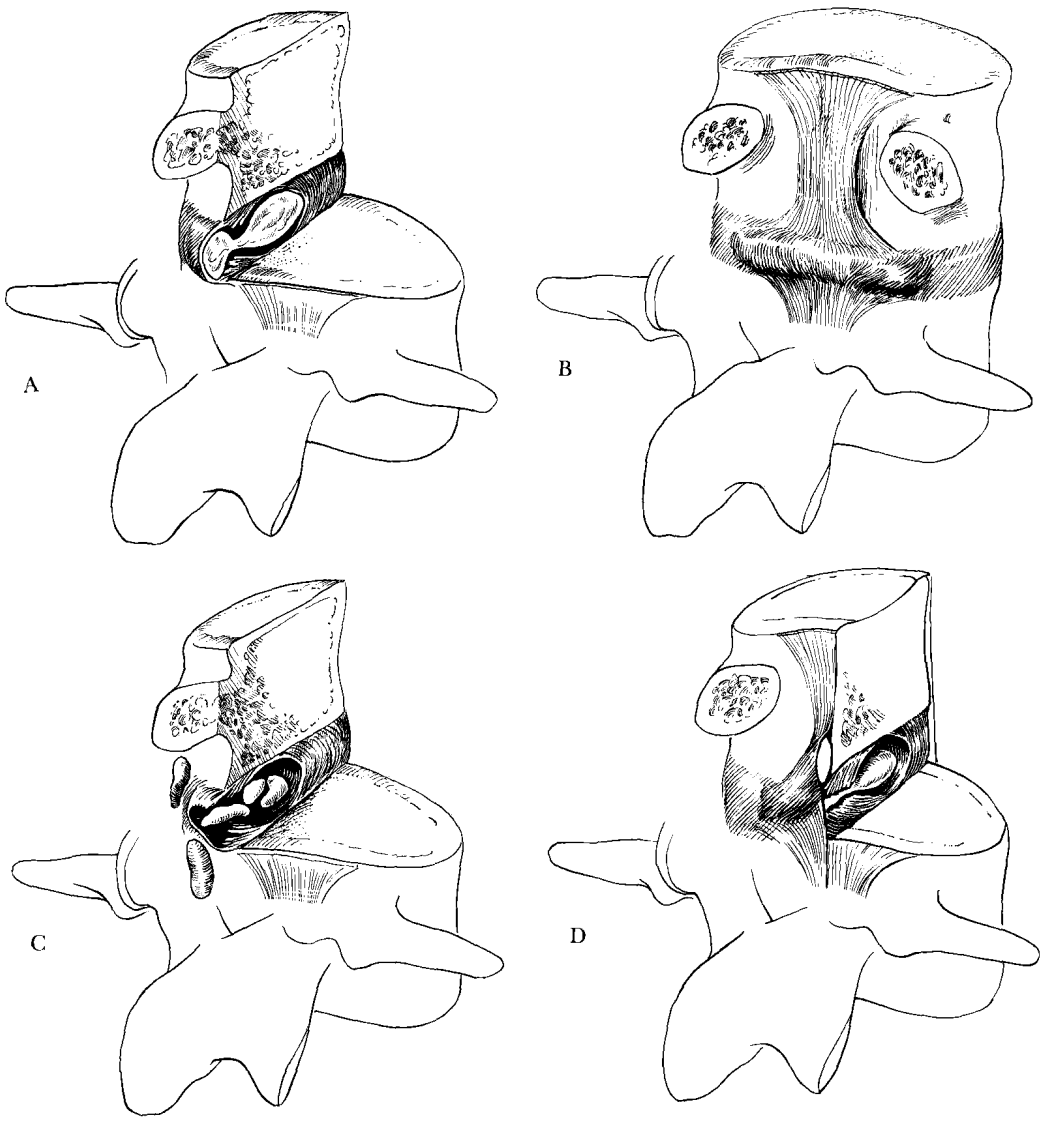


**Figure 9–4.** *A,* Herniation of a lumbar disc beneath the posterior longitudinal ligament in the common lateral position. *B,* Herniation of a lumbar disc in the less frequent central position beneath the strong portion of the posterior longitudinal ligament. *C,* Complete herniation of a lumbar disc through a rupture in the annulus and the posterior longitudinal ligament, with free fragments in the neural canal. These fragments may migrate cranially, caudally or into the intervertebral foramen. *D,* Herniation of a lumbar disc, with upward migration of the disc fragments, beneath the posterior longitudinal ligaments. These fragments may go unnoticed unless specific exploration in these areas is undertaken. (From DePalma, A. F., and Rothman, R. H.: The Intervertebral Disc. Philadelphia, W. B. Saunders Co., 1970.)

subject to persistent mechanical pressure by a turgid nucleus, herniation may ensue. Herniation is a much greater threat in the younger individual, between the ages of 30 and 50, having good turgor in the nucleus, than in the elderly, in whom the nucleus is desiccated and fibrotic. In the older individual the degenerated, thinned disc will often develop without any of the signs and symptoms of acute nerve root compression. This may explain the predominance of acute disc syndromes in the middle-aged population and their rarity in the elderly.

Posterior displacement of the nucleus pulposus may occur in a variety of ways (Fig. 9–4). In an extreme circumstance, there may be a massive nuclear retropulsion, in which a large volume of disc material is suddenly thrust into the spinal canal, producing a profound neurologic catastrophe. More com-

monly, the extrusion is a gradual and intermittent process. The nucleus progressively bulges through the rent in the annulus, being retained in position by the posterior longitudinal ligament. This ligament, which may be stretched and detached by the herniating nuclear material as it forces its way relentlessly backward to the spinal canal, may rupture, with the formation of a free sequestrum into the spinal canal. This disc fragment may then migrate cephalad or caudad or laterally into the intervertebral foramen. It is not only the size of the nuclear herniation which determines its clinical significance but also the direction in which this herniation takes place. In addition, the shape of the spinal canal is shown to be of great importance. Failure to recognize the variety of types of disc pathology having significant spatial relationships will lead to inadequate surgical treatment of these problems. It should also be pointed out that posterior protrusion of disc material may often occur at some site other than that of an obviously thin disc.[16]

Subsequent to the loss of the integrity of the annulus fibrosus, there may be invasion of the disc by granulation material, either through rents in the annulus or through cracks in the cartilaginous end plate. The role of this granulation tissue in regard to the production of pain is as yet unclear.

It should be emphasized that, as the process of disc herniation occurs, biochemical as well as mechanical irritation of the nerve root must be considered. The clinical symptoms which are produced are in all likelihood closely related to biochemical irritation of the nearby nerve root by degradation products of the degenerating disc. It has been dramatically demonstrated by Falconer that myelographic defects are unchanged after successful conservative treatment of sciatica.[32] Thus, mechanical factors alone are insufficient explanations for nerve root symptomatology.

## Variations in the Shape of the Spinal Canal

Verbiest has highlighted the importance of the narrow spinal canal in the production of nerve root compression in the cauda equina.[119, 120] It is not only the absolute dimensions of the spinal canal which are of importance but also the configuration (Fig. 9–5). The trefoil canal, with narrow lateral recesses, presents a configuration in which the nerve roots are particularly vulnerable to compression.

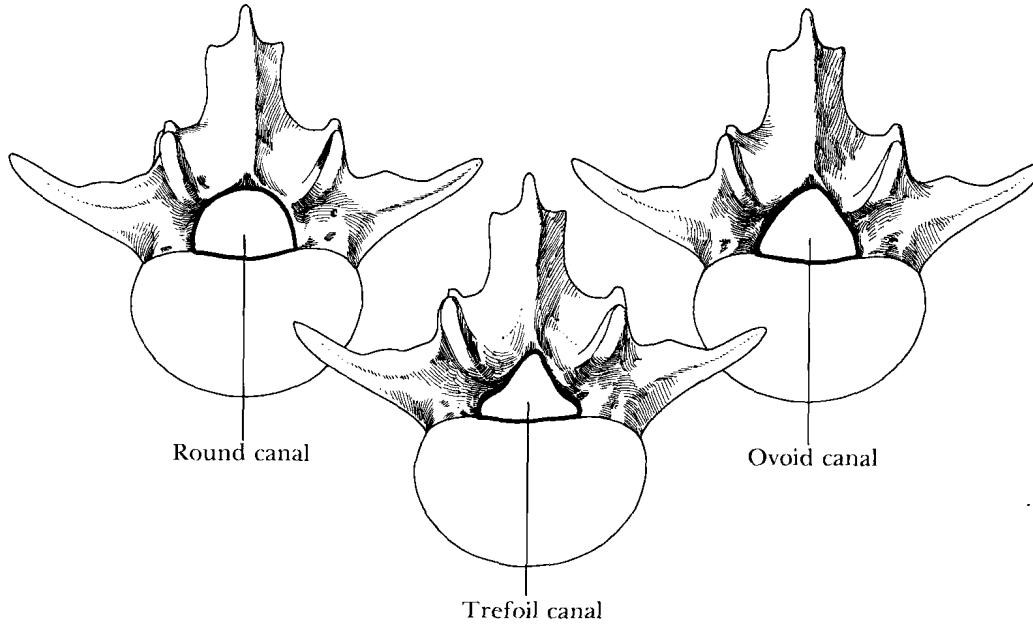


**Figure 9–5.** The three variations of the spinal canal: round, ovoid and trefoil. The lateral recesses of the trefoil canal render the lumbar roots particularly vulnerable to compression by extruded disc material. (From DePalma, A. F., and Rothman, R. H.: The Intervertebral Disc. Philadelphia, W. B. Saunders Co., 1970.)

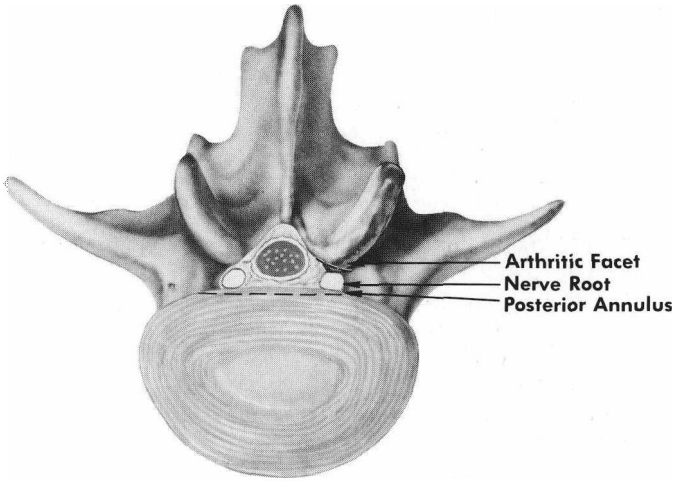


**Figure 9–6.** This drawing illustrates entrapment of a lumbar nerve root in a lateral narrow recess secondary to degenerative changes. Note how posterior buckling of the annulus and arthritic changes in the facet joints can compromise the nerve root in the anterior-posterior direction. Failure to decompress this recess will result in persistent sciatica after surgery.

The anterior-posterior diameter of the canal is the most critical dimension and can be measured precisely only during surgery. Myelography will suggest this diagnostic entity when there is anterior-posterior compromise of the dye column. Plain radiograms will only imply this diagnosis. A "canal to body ratio" developed by Jones is felt to be helpful in substantiating this diagnosis.[64]

It is of interest that this problem is seen most often in males and most frequently at the L4 level. This is, of course, the most narrow area in the lumbar canal.

Patients with congenitally small lumbar spinal canals, and particularly those with a narrow lateral recess, will be susceptible to degenerative changes of the intervertebral disc in terms of nerve root compression. Often a small nuclear herniation of only one to two millimeters in height will cause marked nerve root compression under these circumstances. If the canal were round in configuration this type of herniation would undoubtedly cause little difficulty. In addition to nuclear herniations, posterior osteophytes and/or degenerative changes in the apophyseal joints can produce nerve root compression in this lateral recess. If this type of pathology is unrecognized, failure to adequately decompress these recesses may result in failure to relieve symptoms after back surgery (Fig. 9–6).

A great variety of clinical manifestations may be produced by these structural abnormalities. Unilateral or bilateral sciatica may result, with both motor and sensory disturbance. So-called "cauda equina claudication" can also occur, with bilateral leg pain accentuated by walking and relieved by rest. It may be that the symptoms are produced by relative ischemia of the cauda equina during exercise.

### Foraminal Encroachment

It should be recalled that the lumbar nerve root rests well up at the most cranial part of the intervertebral foramen, a relatively protected position. Soft disc protrusions will only infrequently compress nerve root exiting at the same level. However, as disc degeneration progresses the dimensions of the intervertebral foramen may decrease owing to loss of height of the disc space, formation of posterior-lateral osteophytes and osteoarthritis of the apophyseal joints. As the disc space narrows the superior articular facet of the inferior vertebra will tend to sublux in a superior direction and may impinge the nerve root. These considerations make it mandatory that in all nerve root decompression, the root be followed well out beyond the foramen, and the surgeon must be completely satisfied that there is no compression in this area (Fig. 9–7).

### Pedicle Migration

It has been lucidly shown by MacNab that as disc degeneration occurs, with subsequent loss of height of the disc space, the pedicles will migrate in a caudal direction relative to the nerve root and may tether the root during its descent.[72, 73] This may produce an intractable radiculitis and, again, if unrecog-

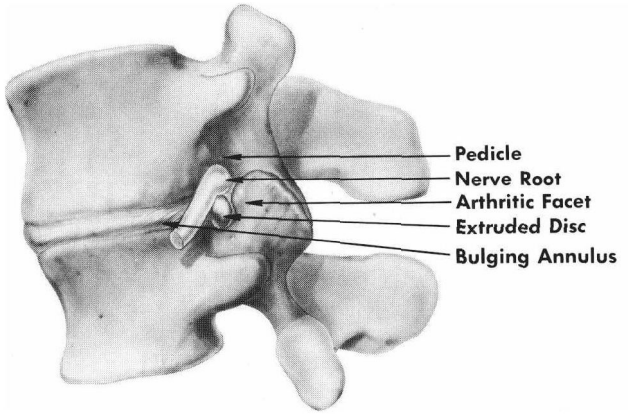


**Figure 9–7.** Foraminal encroachment secondary to chronic disc degeneration. The nerve root exiting the foramen will be compressed by the arthritic facet joint posteriorly, the relative descent of the pedicle superiorly and the posterior bulge of the annulus or disc extrusion anteriorly. The surgeon must satisfy himself that the nerve root is entirely free throughout the entire course of the foramen when the nerve root is decompressed.

nized, will lead to failure of nerve root decompression. This entity is most often seen in the elderly individual (Fig. 9–8).

## CLINICAL SYNDROME OF LUMBAR DISC DISEASE

Lumbar disc disease represents the most common cause of back and leg pain. It is a multifaceted syndrome and must be recognized as such if a diagnosis is to be correct and the treatment effective. One sees with disturbing regularity missed diagnoses of herniated lumbar discs that present in an atypical fashion, unfamiliar to the practitioner. It is equally precarious, however, to polarize one's thinking at the opposite extreme and attribute all cases of back and leg pain to abnormalities of the intervertebral disc. A wide variety of vascular, infectious and space-occupying lesions can mimic the herniated lumbar disc. An attempt will be made in this section to outline the classic picture of the lumbar disc syndrome as well as the more common variants.

### History

#### BACK PAIN

The majority of patients with degenerative disc disease in the lumbar spine have low back pain as the earliest symptom. The onset is most often in the third decade of life and is insidious in nature. The patient recalls that after periods of prolonged physical activity or working in a position that stresses the spine, pain appears in the lumbosacral area. These early episodes of low back pain are usually not accompanied by sciatica. The pain may last a few days and usually subsides with limitation of activity and bedrest. The pain pattern at this time is mechanical in nature, in the sense

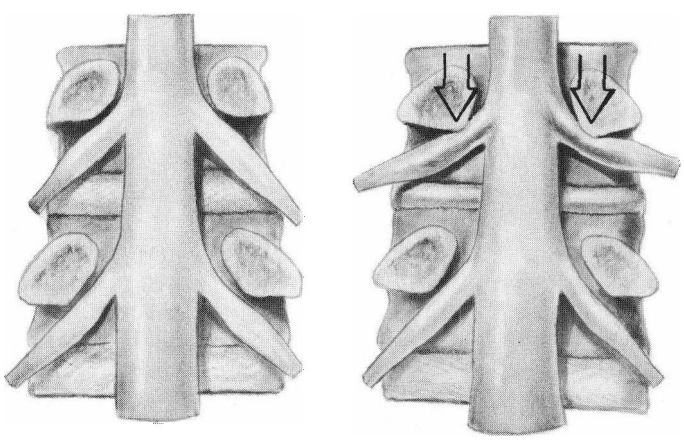

**Figure 9–8.** These drawings illustrate the relative caudal migration of the pedicles during disc degeneration which may tether the nerve roots. During exploration of a nerve root it must be ascertained that the root is free not only of compression but also of tension as well. If the root is tethered about the pedicle, excision of the offending pedicle must be undertaken.