Pathology and Pathogenesis of Lumbar Spondylosis and Stenosis

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Study of autopsy specimens of the lumbar spine makes it possible to construct a spectrum of pathologic change. Progressive degenerative changes in the posterior joints lead to marked destruction and instability. Similar changes in the disc result in herniation, internal disruption, and resorption. Combined changes in posterior joint and disc sometimes produce entrapment of a spinal nerve in the lateral recess, central stenosis at one level, or both of these conditions. Changes at one level often lead, over a period of years, to multilevel spondylosis and/or stenosis. Developmental stenosis is an enhancing factor in the presence of a small herniation or moderate degenerative stenosis. Lesions such as major trauma, spondylolisthesis, those following spinal fusion, Paget's disease, and fluorosis, on occasion act directly to produce central or lateral stenosis. [Key words: low-back pain, degenerative disease, apophyseal joints, intervertebral disc, spinal stenosis, lateral nerve entrapment]

THIS PRESENTATION is based on observations made in the dissection of 50 lumbar spines obtained at autopsy, supplemented by those made during the course of laminectomies in 161 patients. The authors saw not only the progressive degenerative changes that take place in the posterior joints and discs, but also the ways in which spinal nerves may be entrapped as a result of these changes.

The interpretation of these observations in formulating a theory which explains the pathogenesis of spondylosis and stenosis may be open to question. But such a theory is of assistance in that it enables the surgeon to see the picture as a comprehensive whole. It helps him to reach a diagnosis and to formulate a rational type of treatment.

It is evident to the authors that the changes to be described sometimes lead to narrowing of the central spinal canal and lateral nerve canals. Thus, spinal stenosis is seen, not as a separate entity, but as part and parcel of the degenerative process.

While this study demonstrates where and how the cauda equina and lumbar spinal nerves may become entrapped by narrowing of the canals in which these structures run, it does not explain what happens to nerves that are entrapped, nor why sometimes such entrapment produces symptoms and sometimes does not. Thus study of pathologic anatomy should stimulate the surgeon to further study of pathophysiology.

PATHOGENESIS

The whole spectrum of degenerative change is shown in Figure 1. Starting with repeated minor trauma, the degenerative process continues over many years until gross spondylosis is observed. At intervals over such a period of time, complications such as herniation of the nucleus pulposus, lateral stenosis, central stenosis at one level, and multilevel stenosis may be encountered.

Farfan and co-workers have drawn attention to three important facts: 1) At each level in the lumbar spine the

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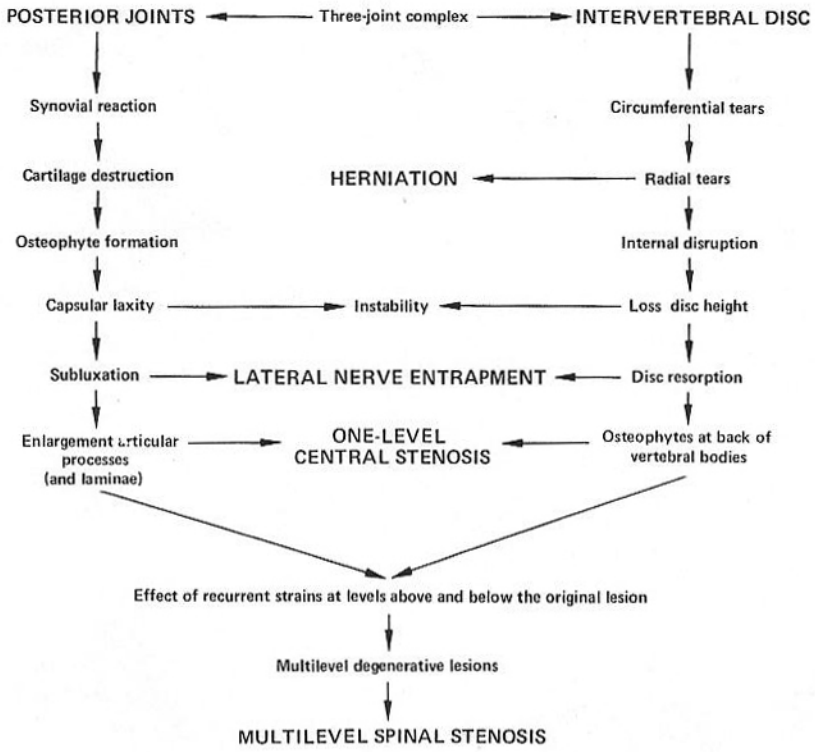


Fig 1. The spectrum of degenerative change that leads from minor strains to marked spondylosis and stenosis.

two posterior joints and the disc form the "three-joint complex." Lesions affecting the posterior joints also affect the disc and vice versa.[4] 2) At the lower two levels the posterior joints are aligned obliquely. This puts these joints at greater risk of recurrent rotational strain.[5] 3) At the same two levels the discs are wedge shaped, greater in height anteriorly than posteriorly. This also places these joints at risk.[5]

The same authors postulate two types of injury as being responsible for initiating degenerative changes in the three-joint complex. 1) Recurrent rotational strain which can quite rapidly lead to degeneration of posterior joints and disc, and 2) minor compression injuries, sometimes with rupture of a cartilage plate, which lead more slowly to degenerative changes in the disc and later in the posterior joints. Changes starting at one level, usually L4–5, later in life place the level above and the level below at risk to strain, and in this way the process spreads. The initial lesion becomes more severe and the degenerative changes become generalized.

Remembering the concept of the three-joint complex, it is convenient to study first the changes seen in the posterior joints, then those in the disc, and finally, the interaction of the two.

Posterior Joint Changes

These diarthrodial joints with articular cartilage and a synovial membrane have a posterior capsule of collagen. The medial and anterior capsule, formed by lateral extensions of the ligamentum flavum, is 80% elastin and 20% collagen.[15] We do not know whether the elasticity of the medial capsule makes the joint stronger, or weaker and thus more liable to strain.

The changes to be seen include the following:[6] 1) a synovial reaction, 2) fibrillation of articular cartilage, 3) gross degeneration and irregularity of articular cartilage, 4) the formation of osteophytes, 5) fracture of an articular process, 6) loose bodies in the joint, and 7) laxity of the joint capsule, resulting in instability.

Figure 2A is of a posterior joint with the posterior capsule removed. Note the presence of smooth osteophytes on both sides of the joint. In Figure 2B, the same joint has been strained to open up the space. In this way recurrent strains, with laxity of the ligaments, can result in minor degrees of subluxation of the joint.

Figure 3A is a pathologic specimen that demonstrates marked degenerative change in the posterior joint. The medial capsule has been removed. The arrow points to a large synovial tag. Figure 3B is of the same joint. The synovial tag has been removed. The two articular surfaces are markedly irregular. Most of the articular cartilage has been eroded. Small osteophytic outgrowths can be seen.

Changes in the Intervertebral Disc

Aging Changes

The nucleus pulposus is gelatinous in nature only during childhood and adolescence. In adult life it becomes increasingly fibrous in nature with advancing years.[1,7,8] Figure 4A, a specimen from a 6-year-old boy, is a longitudinal section that demonstrates clearly the

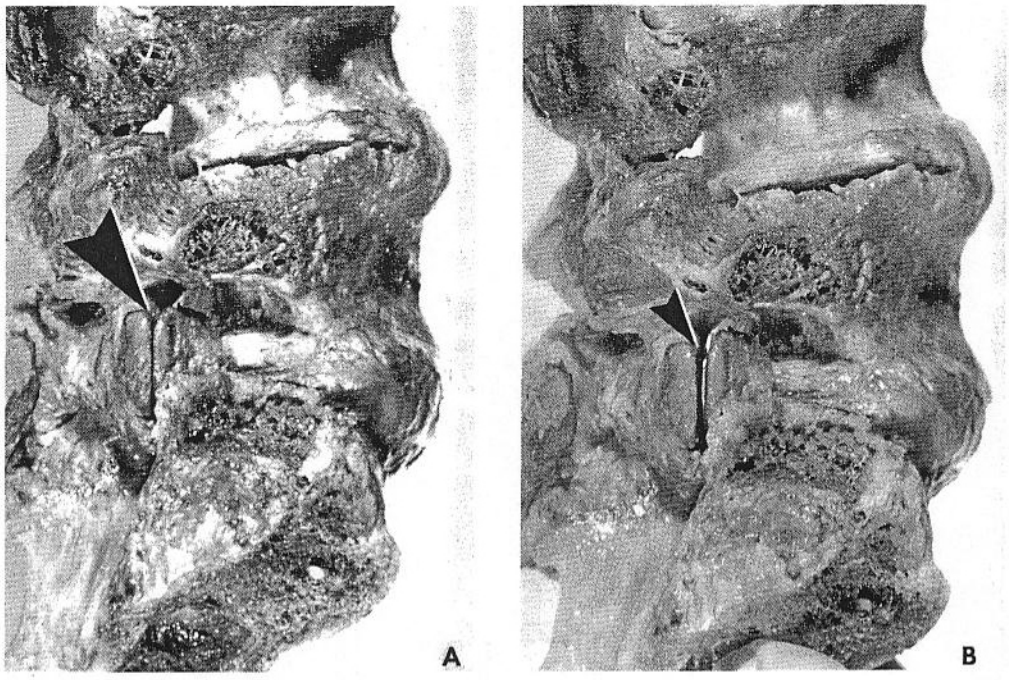


Fig 2. Pathologic specimen showing degenerative posterior joints. **A.** The posterior capsule has been removed. Smooth protuberant osteophytes are seen on both sides of the joint **(arrow). B.** The same specimen with the posterior joints strained and opened up **(arrow).**

presence of well-demarcated, gelatinous nucleus pulposus at several levels. Figure 4B, a specimen from a 17-year-old male patient, shows in cross section a disc with the well-defined nucleus pulposus. Figure 4C, a specimen from a patient aged 55 years, is a cross section of a nucleus pulposus that has become increasingly fibrous in nature. The circumferential collagen fibers of the annulus fibrosus extend far toward the center of the disc. Figure 4D, from a patient aged 75 years, another cross-sectional view, shows marked fibrosis of the whole of the disc which is internally disrupted. It is, of course, impossible to assess to what extent these changes are due to aging or to repeated minor trauma, or to both of these.

Annular Tears

The earliest sign of rotational injury is the formation of circumferential tears in the annulus fibrosus. Most frequently these are seen in the posterolateral part of the annulus.[12] Figure 5A, a cross-sectional view, demonstrates the presence of two such tears, one on either side. With repeated minor trauma such circumferential tears enlarge and coalesce to form one or more radial tears,

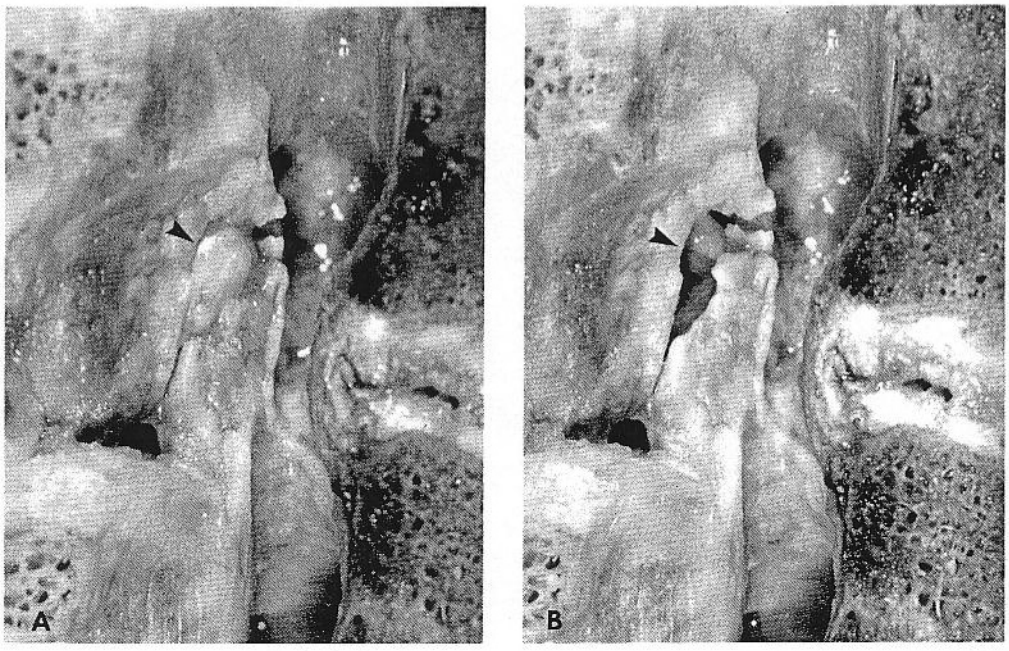
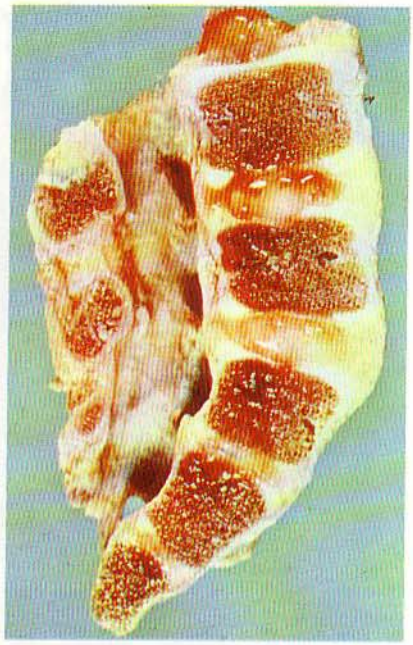


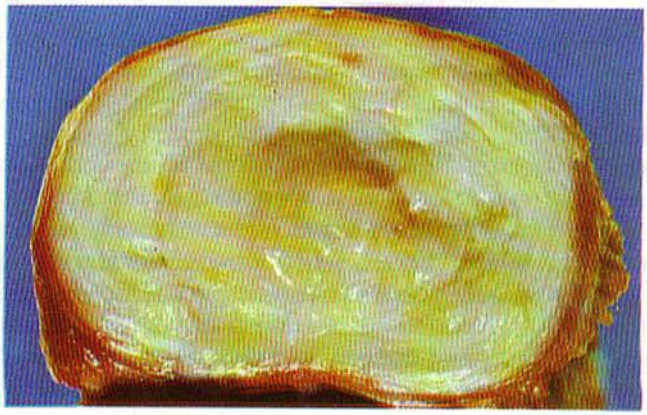
Fig 3. Longitudinal section of specimen demonstrating marked degenerative changes in a posterior joint. **A.** A large synovial tag is present that fills the joint space **(arrow). B.** The same specimen with the synovial tag removed. On both sides of the joint the articular cartilage is eroded and the surfaces are irregular **(arrow).**



4A

4D

5A



4B

4C

5B

Fig 4. The changes that take place in the intervertebral disc with aging. **A.** A longitudinal section of a specimen from a 6-year-old subject. The nucleus pulposus at all levels is gelatinous and well defined. **B.** A cross-sectional specimen from a 17-year-old subject. A well-defined central nucleus pulposus can be seen. **C.** A cross-sectional specimen from a 55-year-old subject. The fibers of the annulus fibrosus occupy more of the disc. The nucleus pulposus is ill defined and has undergone slight central internal disruption. **D.** A cross-section specimen from a 75-year-old subject. The ill-defined nucleus pulposus has undergone very marked internal disruption which has also involved the annulus. **Fig 5.** Tears in the annulus fibrosus. **A.** Cross-sectional view of two circumferential posterolateral annular tears, one on each side. **B.** A cross-sectional view showing several radial tears. These are formed by the enlargement, spread, and coalescence of circumferential tears.

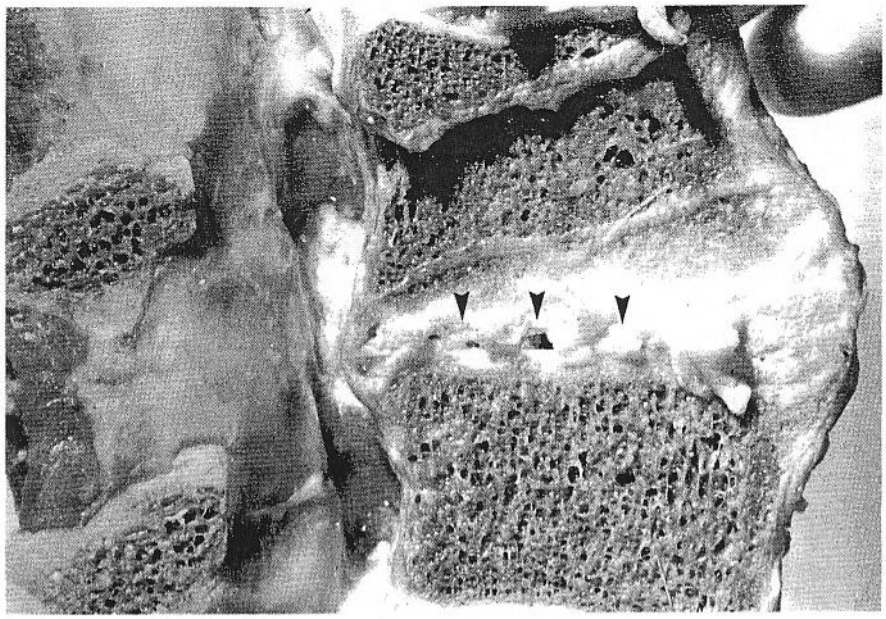


Fig 6. Longitudinal section showing internal disruption of the L4–5 disc. There is loss of disc height and crevices can be seen running from front to back of the disc **(arrows).** The posterior joint is covered by the lateral extension of the ligamentum flavum.

shown in Figure 5B. It appears likely that most disc herniations occur at this stage.

Internal Disruption

From this point on, further minor mechanical trauma, possibly enhanced by biochemical and immunologic factors, leads to internal disruption of the disc. This is depicted in Figure 6. There is a complete internal tear from side to side and from back to front of the disc, without herniation. The disc height is markedly reduced. The annulus bulges right around the circumference of the disc. This results in marked instability of the affected segment. Discography shows a marked correlation between loss of disc height, the presence of traction spurs,[9] and disruption of the disc. Repeated abnormal rotational movement leads also to further degeneration of the posterior joints. At this stage, in fact, the disc resembles a deflated football. Herniation is unlikely because the intradiscal pressure is so much reduced.

Disc Resorption

Further loss of the contents of the disc results in resorption of the disc.[2] The changes to be seen are shown in Figure 7. The disc contents have been almost completely resorbed. The narrow space between the vertebral bodies is occupied by a small amount of fibrous tissue. Vertebral body bone on either side of the disc is sclerotic. As seen in Figure 8, osteophytes may form which divide the intervertebral foramen in two. It is easy to imagine how in this situation there may be entrapment of the spinal nerve at this level. The final stage of this process of resorption, seen only rarely, is depicted in Figure 9. Here there is spontaneous fusion of the body of L5 to the sacrum.

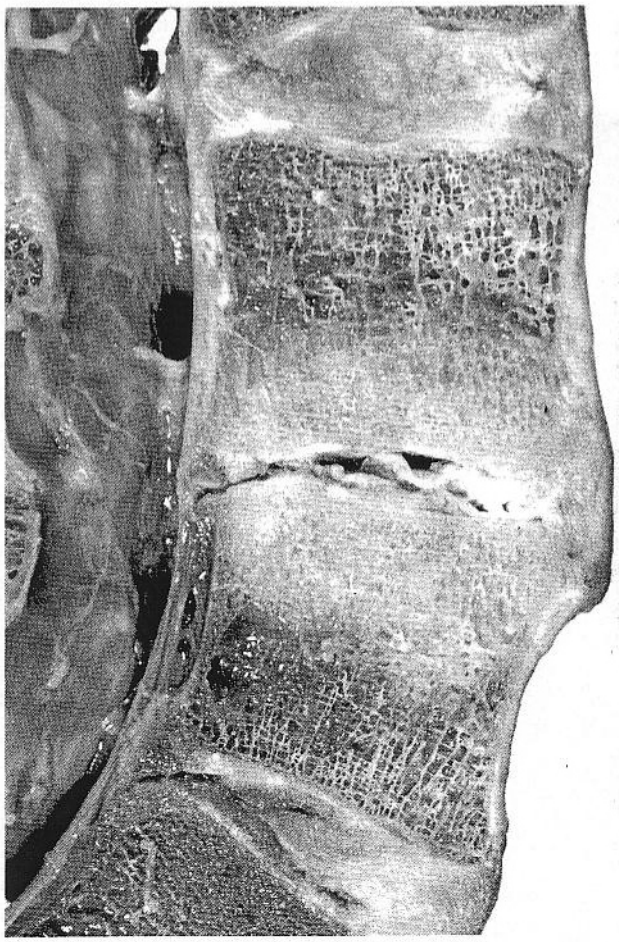


Fig 7. Longitudinal section showing marked resorption of the L4–5 disc. Vertebral body bone on either side of this disc is markedly sclerotic.

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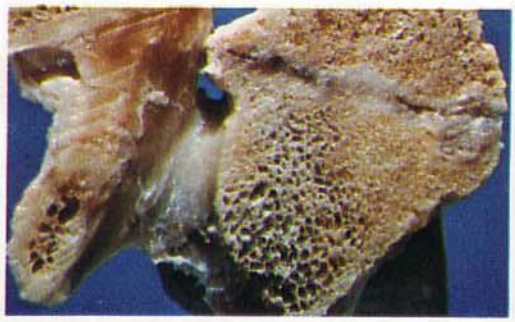

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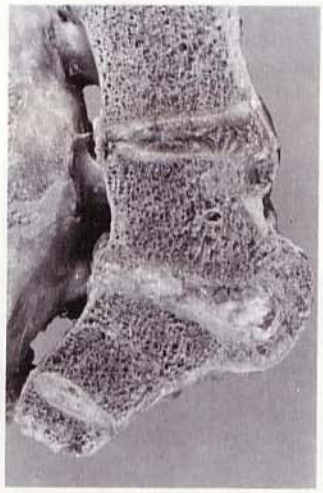

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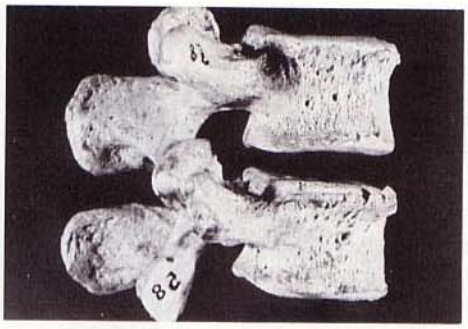
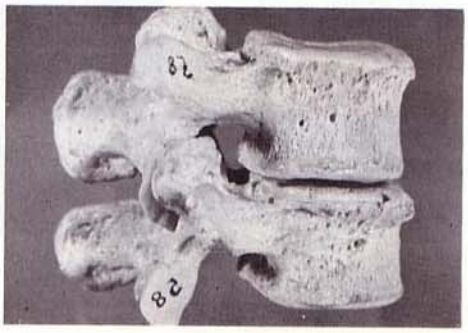


11A



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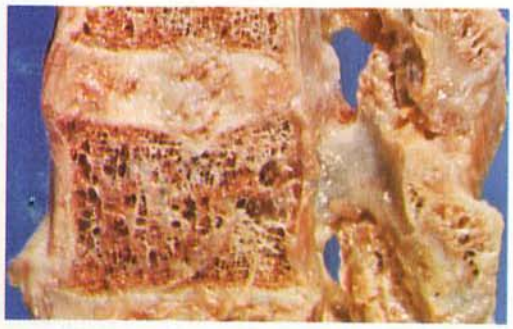

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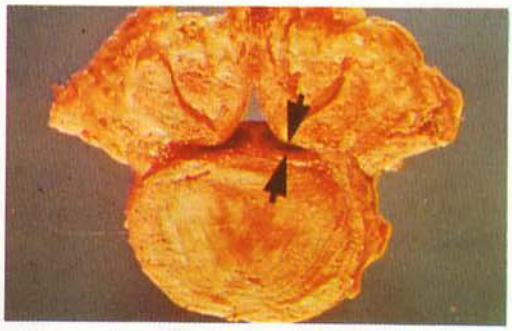

13A

13B

Fig 8. Longitudinal section demonstrating marked resorption of the L5–S1 disc. Osteophytes from the back of the vertebral bodies have divided the foramen into two parts. **Fig 9.** Longitudinal section showing the final stage of disc resorption, not commonly seen, spontaneous anterior fusion by bone at L5–S1. **Fig 10.** Longitudinal section showing internal disruption leading to resorption of the disc. At the upper level, a fracture of the cartilage plate with herniation of the nucleus into the vertebral body can be seen. At the next two levels there is progressive internal disruption of the disc. At the lowest level (L5–S1) there is marked resorption of the disc. **Fig 11.** Effect of diminution of disc height. **A.** Two vertebrae in normal position. Note the height of the disc space and size of the foramen. **B.** The vertebrae have been approximated to simulate loss of disc height. The superior articular process of the lower vertebrae has moved anteriorly and the size of the foramen is reduced. **Fig 12.** Longitudinal section of specimen. Medial to the foramina, both lateral recesses are narrowed, the lower more than the upper. The superior articular processes are both subluxated and enlarged. **Fig 13.** Cross-sectional views of pathologic specimens showing central and lateral stenosis. **A.** The central spinal canal is narrowed by posterolateral enlargement of the inferior articular processes. The lateral nerve canals (lateral recesses) are narrowed by subluxation of the superior articular processes **(arrows). B.** The lateral recess on the left is slightly narrowed due to subluxation of the superior articular process. That on the right is markedly narrowed from both subluxation and osteophytic enlargement of the superior articular process.

Disruption leading to Resorption

Crock[2] has described internal disruption of the disc and isolated disc resorption as two distinct entities. It seems likely to us that the process of degeneration, starting with annular tears, progressing to internal disruption, leads finally to resorption of the disc. The whole spectrum of change in the disc is demonstrated in Figure 10, a longitudinal section of the lumbar spine. At the uppermost level there is rupture of a cartilage plate, a compression injury, with herniation of the nucleus pulposus into the body below. At the next level there is a large central tear in the disc passing from front to back, depicting early internal disruption. At one level lower than this, there is more marked loss of disc height together with a large internal tear, representing more marked internal disruption. At the lowest level, L5–S1, marked disc resorption can be seen. We have in this specimen a picture of the whole spectrum of degenerative change. It is likely that the earliest changes occurred at the L5–S1 level and the most recent changes in the upper lumbar spine.

Combined Changes

We turn now to consider the three-joint complex as a whole. Here it is simplest to think of the ways in which loss of disc height affects the complex.

Common causes of loss of disc height are the following: 1) internal disruption of the disc, 2) resorption of the disc, 3) chemonucleolysis, and 4) discectomy.

The sequelae of loss of disc height are the following: 1) subluxation of the posterior joints, 2) upward and forward displacement of the superior articular process, and 3) narrowing of the lateral recess, which sometimes results in entrapment of the spinal nerve just medial to the intervertebral foramen.

Figure 11A shows the normal disc space between two vertebrae, normal alignment of the posterior facets, and normal size of the foramen. In Figure 11B, the vertebral bodies are approximated to narrow the disc space. As a result, the superior articular process is displaced upward and forward on the inferior articular process and the foramen is narrowed. The most significant narrowing occurs just medial to the foramen, as seen in Figure 12, a midline longitudinal section of the lumbar spine. Upward and forward displacement of the superior articular process has markedly narrowed the lateral recess just medial to the intervertebral foramen. The same process is seen in cross-sectional view in Figure 13A. The lateral recesses are markedly narrowed due to upward and forward displacement of the superior articular processes. In this specimen the central spinal canal also is narrowed by enlargement of the inferior articular processes of the upper vertebra, which bulge posteromedially into the canal. Thus in one autopsy specimen there is demonstrated both central and lateral stenosis. In Figure 13B on the left, the lateral recess (lateral nerve canal) is somewhat narrowed by subluxation of the superior articular process; on the right the recess is very markedly narrowed by both subluxation and irregular enlargement of the superior articular process.

The picture revealed by a study of autopsy specimens can now be seen almost as clearly in computerized axial tomographic (CAT) scans. Figure 14 of a CAT scan demonstrates both central and lateral stenosis, resulting from a previous posterior spinal fusion. The type of nerve entrapment in the lateral recess that is clearly seen in these specimens was described by Williams in 1932,[14] 1 year before Mixter and Barr[10] published their classic paper on disc herniations. The importance and the frequency of this complication of loss of disc height are not yet fully appreciated.

Recurrent Rotational Deformity

From what has been written above in considering the degenerative changes that take place in the posterior joints and discs, it is clear that in the early stages, abnormal movement can produce low-back and referred leg pain, mediated through the medial branches of the posterior primary rami and sinuvertebral nerves.[3] It is more difficult to appreciate that later on, instability superimposed on structural changes can cause recurrent spinal nerve entrapment. For example, in degenerative lesions affecting the L4–5 segment, changes in the posterior joints lead to loss of articular cartilage, osteophytic enlargement of the articular processes, stretching of the capsule, and an abnormal degree of movement of these joints. At the same time, internal disruption of the disc results in approximation of the vertebral bodies, bulging

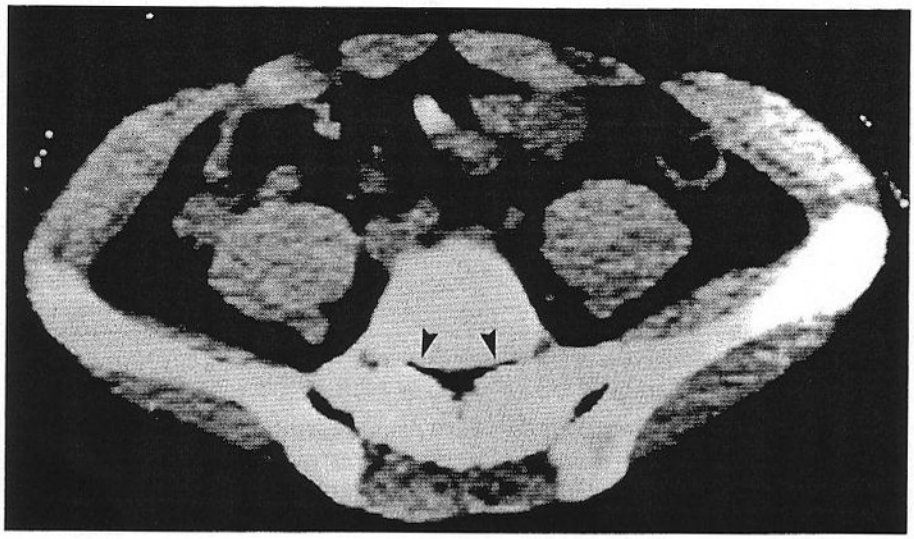

Fig 14. Computerized axial tomographic scan at the L5 level of a patient who had a discectomy 10 years previously and a posterior fusion 1 year later. A massive posterior fusion can be seen at the lower part of the figure which produced marked stenosis of the central spinal canal. There is also narrowing of the lateral recesses medial to the pedicles **(arrows).**

of the annulus fibrosus right around the periphery of the disc, and upward and forward subluxation of the superior articular processes. These changes produce some narrowing of the lateral recesses. If the L4 vertebra is rotated on L5, on one or other side depending on the direction of rotation, the posterior joint opens up, the superior articular process of L5 moves forward, and the lateral recess is further narrowed—a dynamic process. This process has been illustrated in color in a previous paper.[15]

Involvement of Several Levels

It is well documented that, whereas in the early stages lesions are confined to one level (most commonly L4–5 or L5–S1), later the process spreads to involve several levels in the lumbar spine. At each of these levels the process develops in the ways described previously. Experimental work supports the view that abnormal mobility and decreased mobility at one level predispose to strains at levels above and below.[13] This process is clearly demonstrated in Figure 15, a longitudinal section of an autopsy specimen obtained many years after a posterior spinal fusion from L3 to the sacrum. The discs below the fusion are almost normal, being well protected from recurrent strain by the fusion. At the L2–3 level, just above the top of the fusion, there is marked internal disruption of the disc. The posterior joints are enlarged and protrude posteromedially into the spinal canal, producing central stenosis at this level. In this instance there has been for years no movement of posterior joints or discs from L3 to the sacrum. Recurrent strain has centered on the L2–3 joint complex with resultant degeneration at this level. The final stage of this process, seen at the end of the spectrum, is severe degenerative disease of the whole lumbar spine, often accompanied by some degree of scoliosis, with a ro-

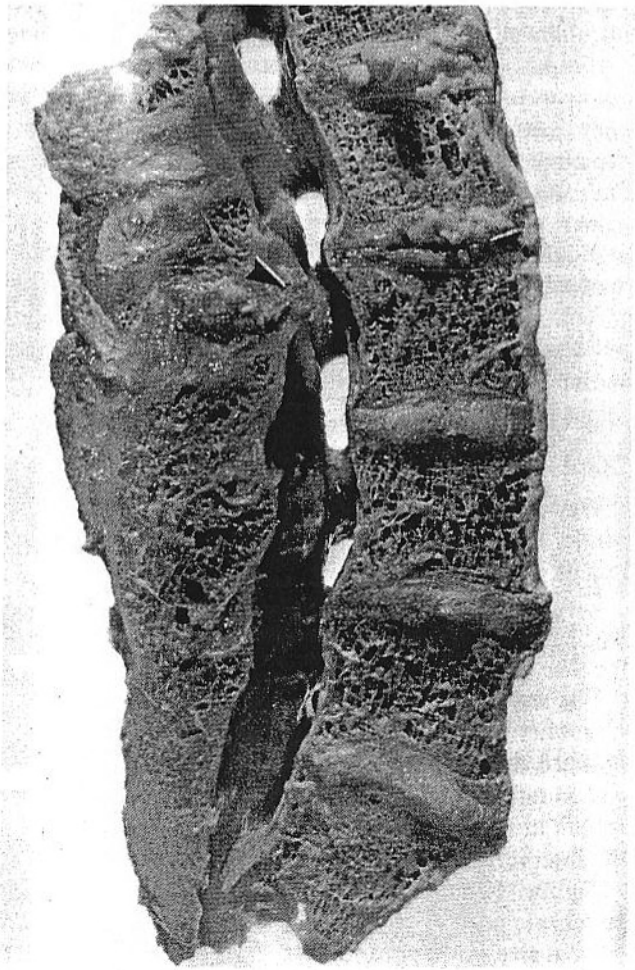

Fig 15. Longitudinal section of the lumbar spine. A massive posterior fusion can be seen extending from L3 to the sacrum. The discs opposite the fusion are remarkably well preserved. At the L2–3 level just above the fusion, the disc has undergone internal disruption **(small arrow).** At the same level the posterior articular processes are enlarged and protrude into the spinal canal to produce central stenosis **(large arrow).**

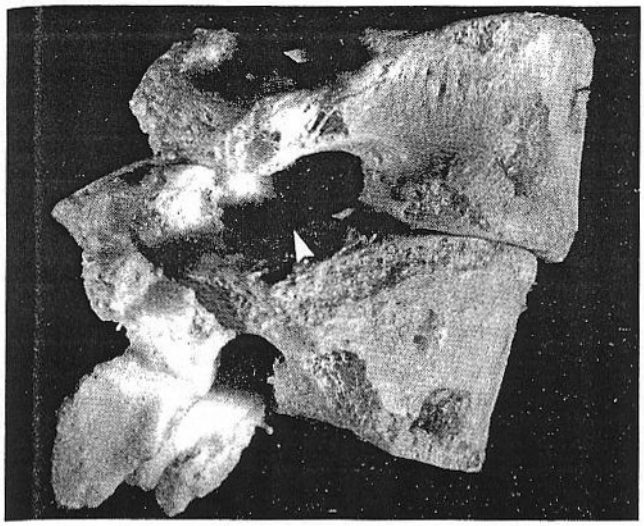

Fig 16. Two lumbar vertebrae, L4 and L5, from a patient with degenerative spondylolisthesis. L4 has slipped forward on L5. The inferior articular process of L4 almost touches the back of the vertebral body of L5 **(arrow).** This produces entrapment laterally of the L5 nerve. (Figure courtesy of JB Lippincott, Disorders of the Lumbar Spine, Edited by AJ Helfet and DM Gruebel Lee, Figure 4-19B, p 42, and of Dr. HF Farfan).

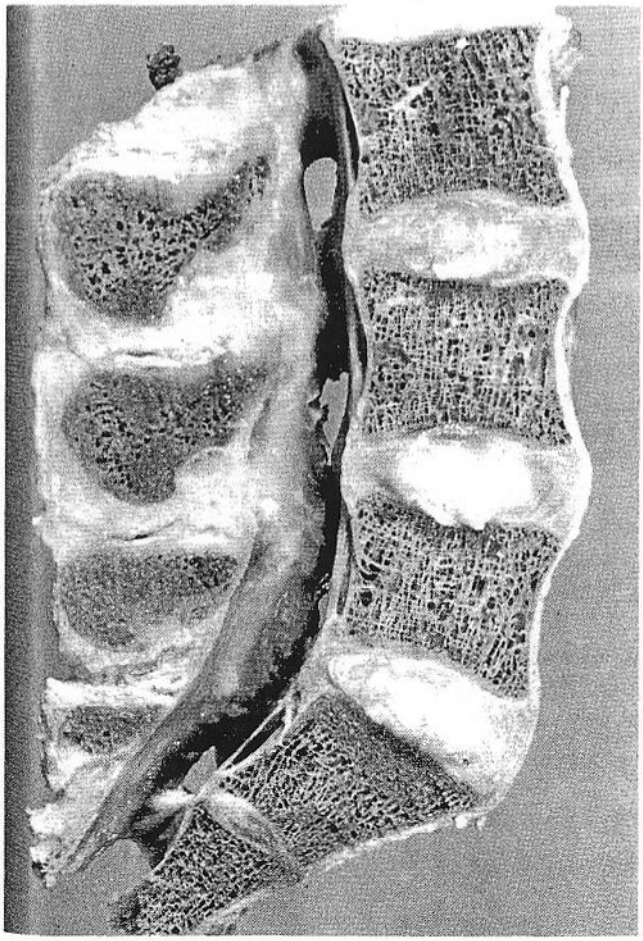

Fig 17. Longitudinal section showing developmental stenosis. The whole central lumbar spinal canal is narrow. The discs are normal. At each level the nucleus pulposus is well defined.

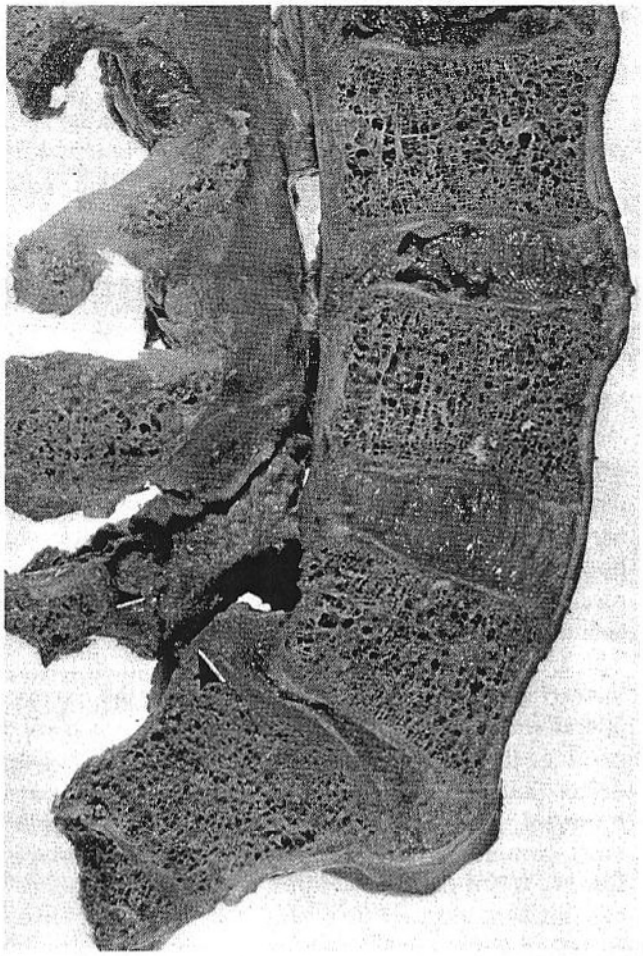

Fig 18. Longitudinal section of L5–S1 isthmic spondylolisthesis. On the right, the forward slip of the body of L5 on the sacrum can be clearly seen. On the left the defect in the pars interarticularis is demonstrated **(small arrow).** The upper part of the pars, just above the defect, has enlarged anteriorly to produce stenosis of the lateral part of the nerve canal **(large arrow).** It is likely that this has entrapped the S1 nerve. The L5–S1 foramen is narrowed with entrapment of the L5 nerve.

tatory element, and in some instances with both central and lateral stenosis, with nerve entrapment at several levels.

Degenerative Spondylolisthesis

Brief mention should be made of the curious type of lesion, seen more often in women than in men,[11] which is sometimes seen as an accompaniment of degenerative lesions. Figure 16, (courtesy Dr. HF Farfan), is of the L4 and L5 vertebrae in a case of degenerative spondylolisthesis. The body of L4 has slipped forward on that of L5. The main pathologic finding was very marked erosion of the superior articular processes of L5. This allowed the inferior articular processes of L4 to slip forward as the disc yielded. The anterior portion of the inferior articular processes of L4 is almost in contact with the posterior aspect of the vertebral body of L5. The L5 nerves were entrapped at this site between the articular processes and the vertebral body.

Other Factors

Developmental Stenosis

Figure 17, a longitudinal section of the lumbar spine, demonstrates a moderate degree of developmental narrowing of the lumbar spinal canal. We do not believe that this type of stenosis alone causes symptoms, but that a small disc herniation or a minor degree of degenerative stenosis is more likely to cause nerve entrapment when it is present. Thus, developmental stenosis may be regarded as an enhancing factor.

Lesions That Result in Direct Nerve Entrapment

Several different types of lesions can, in a direct way, produce nerve entrapment without developmental or degenerative narrowing of the main spinal canal or nerve canals. These are major fracture of a vertebral body, isthmic spondylolisthesis, postfusion stenosis, and rarely, Paget's disease and fluorosis. Figure 18 is a longitudinal section from a case of isthmic spondylolisthesis. The arrow on the left points to the defect in the pars interarticularis, and that on the right, to the narrowed lateral recess which lies opposite new bone and fibrocartilage just above the defect. It is this point above the defect that is responsible most often for the nerve entrapment. Figure 15 demonstrates postfusion stenosis, most commonly seen at the upper end of the spinal fusion. When in these lesions there is some degree of concomitant degenerative stenosis, the entrapment of spinal nerves is rendered even more likely.

CONCLUSION

This paper attempts to document, by the help of illustrations, the changes that occur in degenerative and allied lesions in the lumbar spine. In doing this it is convenient to synthesize them into a spectrum of change which shows something of the pathogenesis of this process. Such documentation, in our opinion, forms the basis for better understanding of the ways in which lumbar spinal nerves may be entrapped.

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