SPINAL STENOSIS, A CAUSE OF CAUDA EQUINA COMPRESSION*

JOSEPH SCHATZKER and GEORGE F. PENNAL, TORONTO, ONTARIO, CANADA

In 1934 Mixter and Barr described the syndrome of the ruptured intervertebral disc in the lumbar region. Since then the herniated intervertebral disc has been considered to be the dominant cause of low back pain and sciatica. In 1953 Schlesinger and Tavers pointed out that in the presence of a narrow spinal canal patients with herniated intervertebral discs may present an atypical picture of multiple root or cauda equina compression. Verbiest in 1954 made the significant observation that structural narrowing of the spinal canal alone could, if it resulted in an incongruity between the capacity and the contents of the lumbar spinal canal, give rise to compression of the roots of the cauda equina in the absence of disc herniation. Yet, in spite of these observations, the spinal canal has received little attention. **Definition**—Spinal stenosis is a localised narrowing of the spinal canal due to a structural abnormality. Despite the many causes that it may have, if it gives rise to cauda equina compression, such narrowing results in a specific symptom complex, has a common pathogenesis of cauda equina compression, and demands the same treatment. It is for these reasons that we consider it prudent to regard spinal stenosis as a syndrome rather than to deal with each cause of stenosis as a separate entity.

**THE SYNDROME OF SPINAL STENOSIS**

**Symptoms**—Patients with this syndrome present symptoms of cauda equina "claudication" (Blau and Logue 1961) or of unremitting back pain with bizarre radicular radiation.

Cauda equina "claudication" is characterised by pain, weakness and numbness in the legs which comes on with walking and is relieved by rest. The symptoms so closely resemble those of vascular insufficiency that not infrequently arteriography is carried out before the true nature of the symptoms is appreciated. As the disease progresses some pain may be experienced at rest, and weakness on effort may become a prominent symptom. Unguarded movements such as coughing or sneezing rarely aggravate the pain. This is in contrast to the usual mechanical type of aggravation of symptoms in frank disc protrusion.

Some patients present symptoms and signs far more severe than can be explained on the basis of disc herniation. Back pain is often accompanied by bilateral asymmetrical radiation of pain, often in the femoral as well as in the sciatic nerve distribution. It is bizarre and unremitting and frequently not relieved by rest. Indeed, so severe and diffuse is this pain that clinically either a tumour of the cauda equina or gross functional overlay is diagnosed. Numbness and weakness occur, but are usually not the initial symptoms. Conservative measures invariably fail and the patient's disability progresses.

**Signs**—Spinal stenosis is not characterised by specific clinical abnormalities of either the musculo-skeletal or neurological systems. Indeed, symptoms may be present in the absence of any signs. If signs are present they consist in varying degrees of paravertebral spasm and limitation of lumbar movement and straight leg raising. Motor weakness is most evident peripherally. Impairment of deep tendon reflexes occurs in most patients, the ankle reflex being the most severely affected. Sensory changes, if present, are predominant in the fifth lumbar and first sacral dermatomes.

**Pathological findings**—All forms of spinal stenosis share two abnormalities which are responsible for the nerve root compression, namely, shallowness of the lateral recess and a

decrease in the dorso-ventral diameter of the spinal canal (Fig. 1). The lateral recess is that space bounded by the medial portion of the superior articular facet and the lamina above, by the pedicle laterally, and by the vertebral body, its superior lip and the adjacent disc below. The lateral recess contains the nerve root. It is in this limited space that the nerve root is most vulnerable to compression. Shallowness of the lateral recesses and the decrease in the dorso-ventral diameter of the spinal canal thus result in a decrease of its capacity. If a critical point is reached at which the canal can no longer accommodate its contents, then compression of the cauda equina results. The compression becomes manifest first in the lateral recesses, but as the dorso-ventral diameter of the canal becomes further reduced, the stenosis becomes more marked and the more medially lying roots come under compression. The compression is always posterior and postero-lateral. As the changes responsible for the compression are usually irreversible, once compression has occurred it increases in severity.

**Diagnosis**—The diagnosis of spinal stenosis is usually suggested by the patient's symptoms but it can be made before operation only by myelography. Although there are certain variations in the myelographic appearance of the different forms of spinal stenosis, there is one feature common to all, namely the postero-lateral and posterior defects in the column of opaque fluid which correspond anatomically to the areas of compression of the roots (Fig. 2). In order to demonstrate these defects it is essential that an adequate amount of opaque fluid be employed and that the radiographs be taken with the patient upright to ensure the greatest possible filling of the subarachnoid space. Ten millilitres of Ethiodan,* whether injected by the lumbar or cisternal routes, have been found to give the required filling to demonstrate any posterior or postero-lateral encroachment. If a complete block is encountered it is also important to visualise myelographically the segment above or below the block in order to evaluate the extent of the stenosis. Thus, two punctures may be necessary.

* British Drug House.

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**Fig. 1**

Diagrams to show the pathomorphology of spinal stenosis. Note the shallowness of the lateral recess and the decrease in the dorso-ventral diameter of the spinal canal. On the right the striations show the extent of the lateral decompression that is necessary to unroof the lateral recess.

**Fig. 2**

Oblique and lateral myelographs to show the features of spinal stenosis. Note the postero-lateral and posterior defects in the column of radiopaque fluid.
TABLE I

**"Developmental" Spinal Stenosis**

<table>
<thead>
<tr>
<th>Case number</th>
<th>Age in years</th>
<th>Occupation</th>
<th>Duration of symptoms</th>
<th>Myelographic findings</th>
<th>Extent of laminectomy</th>
<th>Length of follow-up</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>71</td>
<td>Pensioner</td>
<td>1 year</td>
<td>Complete block at L.3/4</td>
<td>Laminectomy and partial facetectomy</td>
<td>2 years</td>
<td>Complete relief of symptoms</td>
</tr>
<tr>
<td>2</td>
<td>63</td>
<td>Machinist</td>
<td>1 year</td>
<td>Narrowing of the canal from L.2 to L.5</td>
<td>Laminectomy and partial facetectomy</td>
<td>1 1/2 years</td>
<td>Complete relief of symptoms with return to work</td>
</tr>
<tr>
<td>3</td>
<td>59</td>
<td>Stock-broker</td>
<td>1 year</td>
<td>Complete block at L.4/5 with narrowing of the canal L.1 to L.5</td>
<td>Laminectomy and partial facetectomy L.1 to L.5 inclusive</td>
<td>1 year</td>
<td>Complete relief of symptoms with return to work</td>
</tr>
<tr>
<td>4</td>
<td>76</td>
<td>Pensioner</td>
<td>2 years</td>
<td>Narrowing of the canal at L.2/3 with a complete block at L.4/5</td>
<td>Central laminectomy L.3, L.4 and L.5</td>
<td>1 1/2 years</td>
<td>Failure. See discussion</td>
</tr>
</tbody>
</table>

**Mode of presentation: unremitting back pain and sciatica**

<table>
<thead>
<tr>
<th>Case number</th>
<th>Occupation</th>
<th>Duration of symptoms</th>
<th>Myelographic findings</th>
<th>Extent of laminectomy</th>
<th>Length of follow-up</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>Carpenter</td>
<td>10 years increasing over last 2 years</td>
<td>Narrowing of canal at L.3/4, L.4/5 and L.5/S.1. Most severe at L.4/5</td>
<td>Laminectomy and complete facetectomy L.3 to L.5 inclusive</td>
<td>4 months</td>
<td>Complete relief of symptoms with return to light work</td>
</tr>
<tr>
<td>6</td>
<td>Pensioner</td>
<td>27 years increasing over last 11 years. Complete incapacity</td>
<td>Poor myelograph with narrowing at L.4/5</td>
<td>Laminectomy and complete facetectomy L.3 to L.5 inclusive</td>
<td>6 months</td>
<td>Recurrence after initial relief. See discussion</td>
</tr>
<tr>
<td>7</td>
<td>Housewife</td>
<td>Chronic low back pain with gradual onset of severe sciatica</td>
<td>Narrowing at L.3/4, L.4/5 and L.5/S.1</td>
<td>Laminectomy and complete facetectomy L.3 to L.5 with resection of facets at L.5/S.1</td>
<td>7 months</td>
<td>Relief of sciatica. Back pain improved but not relieved</td>
</tr>
<tr>
<td>8</td>
<td>Student</td>
<td>8 months</td>
<td>Narrowing at L.1/2 and L.4/5 most severe but column also flattened in between</td>
<td>Central laminectomy and discotomy L.4/5</td>
<td>6 months</td>
<td>Recurrence after initial relief. See discussion</td>
</tr>
</tbody>
</table>

**Treatment**—The only form of successful treatment is surgical and it consists of a decompression which must be sufficient both longitudinally and laterally to relieve completely the stenosis. A midline laminectomy never completely relieves the compression in spinal stenosis. This is the most important and fundamental fact to appreciate in the treatment. The compression of the roots occurs in the lateral recesses. It is only when sufficient resection of the superior articular facets is carried out to unroof the whole of the lateral recesses that the roots are adequately decompressed. The facetectomies should be carried out in the vertical plane. As much as possible of both the superior and inferior articular facets should be preserved to safeguard against possible instability. If, however, a complete facetectomy becomes necessary to unroof the lateral recess, then this must be done.
CAUSES OF SPINAL STENOSIS

The causes of spinal stenosis may be summarised as follows. 1) Developmental; 2) degenerative spondylolisthesis; 3) spondylolytic spondylolisthesis; 4) iatrogenic; 5) traumatic; 6) diseases of the skeletal system, especially achondroplasia and Paget's disease.

CLINICAL MATERIAL

"DEVELOPMENTAL" SPINAL STENOSIS

Verbiest (1954, 1955) and Epstein, Epstein and Lavine (1962) described nerve root compression associated with "developmental" narrowing of the lumbar spinal canal. We propose to denote this form of narrowing as "developmental" spinal stenosis. Eight patients were found to have this form of narrowing (Table 1). Four presented symptoms of cauda equina claudication and four of unremitting back pain with bizarre sciatica. A suggestion of spinal stenosis was gained from the radiological appearance of the spine (Fig. 3). These changes consisted in narrowing of the interlaminar space, approximation of the articular facets to the midline, the normal interpedicular spacing and the plane of the zygoapophysial joints.

![FIG. 3](image)

"Developmental" spinal stenosis. Note in the antero-posterior radiograph the narrowing of the interlaminar space, the approximation of the articular facets to the midline, the normal interpedicular spacing and the plane of the zygoapophysial joints.

Although it demonstrates all the morbid anatomical changes of "developmental" spinal stenosis, no radiographic abnormalities could be discerned.
A definite diagnosis of spinal stenosis could be made only by myelography. Partial or complete blocks of the canal were found at one or two levels, usually at the third or fourth lumbar interspace (Fig. 4). Often there were bilateral waist-like defects in the oil column above and below the block. It was the lateral projection, however, that was diagnostic because it revealed the pathognomonic posterior and postero-lateral defects and the occasional more extensive dorsal flattening of the oil column (Fig. 5).

Operative findings—All patients were operated upon and the pathology carefully studied. No disc herniations were found. The narrowing of the spinal canal was caused by an abnormal configuration of the neural arch and its elements. The laminae of the involved segments often exceeded ten millimetres in thickness. They were foreshortened, overlapped, ended in large bulbous articular facets and slanted markedly in a dorso-ventral plane, thus jutting into the spinal canal and further diminishing its height. The interlaminar space was virtually obliterated.
by the overlap of the laminae and by the approximation of the large bulbous articular facets to the midline (Figs. 6 to 9). Laminectomy under these circumstances was difficult. The dural sac was under pressure, it bulged into the laminectomy hole, it lacked its usual epidural fat and it failed to pulsate. The ligamentum flavum was thickened and in two instances plaques of calcium were found in its substance. In the midline the dural sac was compressed by both the laminae and the thickened ligamentum flavum. A midline laminectomy, however, did not decompress the canal. It was only after adequate lateral decompression, which necessitated resection of variable portions of the superior articular facets, that the postero-lateral compression of the dural sac was relieved and that the roots, trapped in the lateral recesses, were decompressed (Fig. 1).

SPINAL STENOSIS FROM DEGENERATIVE SPONDYLOLISTHESIS

Macnab (1950) and Newman (1963) recognised that spondylolisthesis with an intact neural arch—degenerative spondylolisthesis—has the highest incidence of nerve root compression. Two patients with the syndrome of spinal stenosis were found to have degenerative
spondylolisthesis. One had cauda equina claudication, and the other had severe unremitting back pain and sciatica. Both were women in their forties, and both had a grade one slip of lumbar four on five. In the first, myelography revealed posterior and postero-lateral indentations in the fluid column (Figs. 10 and 11); in the second, myelography revealed a complete block at the level of the slip (Fig. 12) caused by posterior and postero-lateral encroachment, a finding diagnostic of spinal stenosis.

**Surgical findings**—Complete laminectomy of the forward slipping vertebra did little to decompress the roots. It was only after the superior articular facets of the fifth lumbar vertebra were excised that decompression was achieved of both the dural sac and of the fifth lumbar roots which were tightly compressed in the lateral recesses beneath the superior articular facets. To stabilise the segment an intertransverse fusion between the fourth and fifth lumbar vertebrae was done.

As the forward displacement in degenerative spondylolisthesis occurs, much osteoarthritic change develops at the zygoapophysial articulations (Newman 1963). The superior facets of the caudal vertebra develop large osteophytic outgrowths which are directed forwards and medially as if to form a shelf for the inferior facets of the forward slipping cephalad vertebra.

These osteophytic outgrowths result in marked narrowing of the lateral recesses and of the spinal canal more centrally and are responsible for the nerve root compression (Fig. 13).
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SPINAL STENOSIS FROM SPONDYLOLYTIC SPONDYLOLISTHESIS

A sixty-four-year-old Chinese cook presented an eight months' history of totally disabling pain, thought by the physicians to be characteristic of intermittent claudication. Aortography and arteriography were, however, normal. In the course of investigation a grade one lumbar four on lumbar five spondylolisthesis with a defect in the pars interarticularis was discovered (Fig. 14). Although the patient gave no history of previous back pain or sciatica and had no clinical findings of nerve root compression, myelography was done. At the level of the slip a sharp angulation was present in the fluid column, and oblique views disclosed postero-lateral and posterior encroachments (Fig. 15).

Surgical findings—At surgery the dural sac was found to be compressed postero-laterally by large masses of osteocartilaginous tissue which pouted from the defects in the pars interarticularis. Decompression was achieved by removal of the loose lamina of lumbar four, wide excision of the osteocartilaginous tissue from the pars interarticularis and by a laminectomy of lumbar three and lumbar five.

On the third post-operative day the patient was able to walk the length of the hospital corridor without difficulty. He has since returned to work, and one year later continues to be free of symptoms.

IATROGENIC SPINAL STENOSIS

A myelograph, done some time after a laminectomy and fusion, is difficult to interpret because of the inevitable scarring from the operation. If pain and new neurological signs are present, search is often made for new disc herniations. If none is found and the fusion appears solid, the patient is frequently subjected to varying periods of bed rest, physiotherapy, anti-inflammatory drugs, epidural cortisone, and finally a limited exploration may be performed.

The operative note often reads: "... the roots were quite tight and bound down in scar tissue, but no disc herniation was found." Experience with two patients leads the authors to conclude that spinal stenosis may be produced iatrogenically, and that such patients, instead of a keyhole laminectomy, require a liberal decompression.
Case 12—A forty-three-year-old man had had two laminectomies and attempted fusions done five years apart for continuing back pain and sciatica. Despite operations his symptoms progressed and he became more and more disabled. He had tenderness with much spasm and restriction of lumbar movement. The findings indicated a fifth root compression on the right, with limitation of straight leg raising, a positive sciatic nerve stretch test, and appropriate motor and sensory deficits. Radiographs showed massive bone formation posteriorly with the presence of a double pseudarthrosis (Fig. 16). The myelograph revealed a defect between the fourth and fifth lumbar vertebrae. Even more striking was the narrowing of the opaque column (Fig. 17). At operation, exposure of the dural sac was extremely difficult because his laminae had become incorporated into very thick blocks of bone which tightly enveloped and compressed the dural sac and the fifth lumbar and first sacral roots. A very generous decompression was carried out. No stabilisation was attempted. The patient was immediately relieved of all pain and had remained so when seen eleven months after operation.

Case 13—A thirty-five-year-old man had had a laminectomy and fusion from the fourth lumbar vertebra to the sacrum. He developed a painful pseudarthrosis between the fourth and fifth lumbar

FIG. 16

Case 12—Figure 16—Note in the antero-posterior projection the marked density of the fourth and fifth lumbar vertebrae. In the lateral projection there is massive bone formation posteriorly with pseudarthroses at L.4-5 and at L.5-S.1. Figure 17—Antero-posterior myelograph showing a defect in the column of fluid on the right at the level of L.4-5. There is also marked narrowing of the fluid column distally.

FIG. 17

Case 13—Antero-posterior, lateral and oblique myelographs. Note the defects in the dye column (arrows).
vertebrae which was repaired by means of two dowel grafts inserted into the pseudarthrosis. Almost immediately the patient’s symptoms became more intense, and in addition to his back pain he developed increasingly severe right leg pain, markedly aggravated by effort, and signs of fifth lumbar and first sacral root compression. A myelograph done five months later showed a large posterior defect in the dye column corresponding in level to the two dowel grafts (Fig. 18). At surgery the right half of the fusion mass was excised (Fig. 19). The roots were found to be tightly compressed from behind by a thick bar of bone (Fig. 20). A complete decompression was not carried out because all his symptoms were on the right side. The patient had immediate relief of his back and leg symptoms and remained well for almost a year. He then developed gradually increasing bilateral sciatica which was now much more severe on the left side. A myelograph done two years following his last decompression revealed the persistence of his spinal stenosis with more marked compression on the left side. The patient refused further surgery and the findings could not be confirmed. It seems likely, however, that further growth of the remaining fusion mass occurred with further narrowing of the spinal canal.

**DISCUSSION**

**Developmental spinal stenosis**—Patients with developmental spinal stenosis have a diminished capacity of their spinal canal. Any intrusion into the spinal canal will further diminish its size. Whether the intrusion will give rise to symptoms is determined by the severity of their developmental defect, that is the severity of their structural narrowing. How critical this relationship can be has already been pointed out by Schlesinger and Taveras (1953) who emphasised that a disc herniation into a spinal canal narrow at one level can give rise to signs and symptoms of a much larger space-occupying lesion.

The structural abnormality in developmental spinal stenosis is such that not only are the lateral recesses very shallow but the posterior articular facets are much closer to the midline than normally. In this way intrusions from the floor of the lateral recess, such as a posterior osteophyte or disc herniation, or from its roof from an anterior osteophyte on the zygoapophysial articulation, can diminish the capacity of the spinal canal. As no disc herniations were found in our series it appears that degenerative changes play a significant role in diminishing the capacity of the spinal canal in spinal stenosis. The age of onset of symptoms is thus determined by both the severity of the developmental stenosis and by the severity and age of onset of the degenerative changes.

The three failures in our series of “developmental” spinal stenosis illustrate important principles in the management of such cases.

In the first, Case 4, despite definite myelographic evidence of stenosis between the third and fourth lumbar vertebrae (Fig. 21) the surgeon satisfied himself with doing a central
laminectomy of the third, fourth and fifth lumbar vertebrae to relieve a complete block at the lumbar four/five level. Lateral decompression was not done (Fig. 22) because pulsation of the dural sac returned. Not only did the surgeon fail to decompress the lateral recesses but he also disregarded the higher stenotic area.

In the second patient, Case 6, great difficulty was encountered in carrying out the myelograph. Difficulty with myelography in "developmental" spinal stenosis is frequent because not only is the interlaminar space very narrow with consequent difficulty in introducing the needle, but also the subarachnoid space is tight because of external compression. Despite a successful tap and free flow of spinal fluid, not uncommonly both a subarachnoid and subdural injection of the contrast medium results. Such a myelograph must not be accepted for diagnostic purposes. If lumbar myelography fails then a cisternal puncture must be done.

Although in Case 6 the myelograph was not of a good diagnostic quality it did show posterior encroachment at the lumbar four/five level. Because of the difficulties encountered the myelograph was not repeated and a cisternal puncture was not done.

At operation, exposure of the dural sac was extremely difficult and typical "developmental" spinal stenosis was encountered. A complete decompression from lumbar three to the sacrum was done. The patient was initially improved, but six months later pain and leg weakness on effort returned. A second myelograph failed. Three hours later the patient became paraplegic; at an immediate exploration the spinal stenosis was found to extend to the thoraco-lumbar junction. A very thin epidural clot was the cause of his paraplegia which regressed following decompression. The complication of paraplegia makes it impossible to give an accurate clinical evaluation of the patient following his second decompression. The pain he had before operation was, however, relieved.
In the third patient, Case 8—despite evident stenosis at lumbar one/two and the more marked myelographic changes at lumbar four/five—the patient's symptoms and signs pointed to the lumbar four/five segment and his extreme youth prompted the surgeon to do a limited decompression of the fourth and fifth lumbar vertebrae. The disc was opened but no herniation was encountered. The patient was initially improved, but within six months his symptoms returned with the addition of pain in the femoral nerve distribution. A second—and more radical—decompression relieved him of all symptoms.

These cases clearly indicate that the only certain method of evaluating the extent of the stenosis is by myelography, and that relief of symptoms can only be expected if an adequate lateral and longitudinal decompression is carried out. In this series patients whose stenosis was relieved at the time of their first decompression have remained free of symptoms. The three recurrences were in those in whom the decompression was not adequate both laterally and longitudinally. These cases further emphasise the fact that return of dural pulsation is not an indication of an adequate decompression.

Although partial and complete facetectomy has been practised in patients with "developmental" spinal stenosis without stabilisation of the spine, spondylolisthesis has not developed, nor have any patients complained of pain suggestive of mechanical vertebral instability. All patients were fitted with a lumbo-sacral brace and were encouraged to use it permanently.

**Spinal stenosis from degenerative spondylolisthesis**—Patients with degenerative spondylolisthesis, with evidence of root compression, in whom posterior and postero-lateral encroachments in the myelograph are seen, but with no disc herniation, must be considered to have spinal stenosis. In such cases wide decompression of the canal is necessary, even if it requires complete facetectomy. Such a segment should then be stabilised by an intertransverse fusion to prevent further forward displacement.

**Spinal stenosis from spondylolytic spondylolisthesis**—Patients with spondylolisthesis with a defect in the pars interarticularis, who present symptoms and signs of root compression, and in whom posterior and postero-lateral indentations, with no disc herniation, are seen in the myelograph, have spinal stenosis. In these, in addition to a central laminectomy, wide lateral removal of the osteocartilaginous tissue from the pars interarticularis and division of any fibrous bands or adhesions is essential. Whether stabilisation of the spine is done depends on the patient's age. In the older patient with spondylolytic spondylolisthesis progression of the forward slip is unlikely. Furthermore, the lateral decompression does not influence the stability of the segment. We are of the opinion that, if there is no indication for fusion because of the spondylolisthesis itself, then, in the older patient, stabilisation is not necessary. It must also be remembered that these patients may present the syndrome of spinal stenosis in the absence of any overt neurological signs.

**Iatrogenic spinal stenosis**—Iatrogenic spinal stenosis may be produced at operation, as the patient with the two dowel grafts illustrates, or it may develop in association with a pseudarthrosis. The progression of the stenosis in both cases suggested that it was caused by new bone formation along the front of the laminae. This new bone formation was confirmed at the time of surgery. This raises a further possibility. The capacity of the spinal canal determines the severity of any compression of the cauda equina, and the smallest intrusion into a tight canal may result in very severe root compression. If, at the time of laminectomy and fusion, spinal stenosis is not recognised and a conventional posterior interlaminar fusion is done, then progression of the spinal stenosis may result. As spinal stenosis is rare, this probable complication of spinal fusion would undoubtedly be very rare, but it would explain the progression of root compression in some patients who have had such a fusion carried out, and in whom no new cause such as a disc herniation is found to explain the progression of their symptoms and signs.
The authors have dealt with "developmental" spinal stenosis, spinal stenosis associated with spondylolisthesis and without a defect in the pars interarticularis, and with iatrogenic spinal stenosis. Of these, the "developmental" form appears to be the most common. Teng and Papatheodorou (1963) described spondylosis as a cause of cauda equina compression, but a review of their article leads the authors to the conclusion that their cases represent "developmental" spinal stenosis with degenerative changes of ageing. If spondylosis alone could give rise to cauda equina compression, then this should be a much more frequently encountered entity. Achondroplasia (Epstein et al. 1962), Paget's disease (Hartman and Dohn 1966) and trauma (Brish, Lerner and Braham 1964) are much rarer forms of structural narrowing of the spinal canal.

SUMMARY

1. The syndrome of spinal stenosis is due to compression of the cauda equina from structural narrowing of the lumbar spinal canal.
2. Patients with this syndrome present symptoms of cauda equina claudication or of unremitting bizarre back pain and sciatica.
3. The compression of the cauda equina is always posterior and postero-lateral and is caused by narrowing of the lateral recesses and of the dorso-ventral diameter of the spinal canal.
4. The diagnosis can be made only by myelography. The only form of successful relief of the nerve root compression in spinal stenosis is adequate lateral and longitudinal decompression.

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