Occult lumbar spinal stenosis

A. R. CHOUDHURY AND J. C. TAYLOR

From the Regional Department of Neurosurgery, Derbyshire Royal Infirmary, Derby

SUMMARY Twenty-eight patients presenting with low back pain, associated with sciatic or femoral neuropathy, were found to have lateral recess stenosis occurring as a result of hypertrophy of the facet joints, with preservation within normal limits of the sagittal AP diameter of the lumbar canal. Pathology was believed to be traumatic in origin, and the variable nature of the adhesions suggested recurrent inflammation; the hypertrophy of the facet joints may have been the result of traumatic inflammatory hyperaemia. Radiological investigations were unhelpful. The diagnosis of the condition was made at the time of surgical exploration by the findings of alteration of the facet joints, adhesions and fixity of the nerve roots, normal sagittal AP diameter of the canal, and absence of other significant lesions. Gratifying results were obtained with decompression by wide laminectomy with excision of overhanging facet joints and release of adhesions.

The concept of stenosis of the cervical spinal canal has been widely recognised for many years (Pallis *et al.*, 1954; Payne and Spillane, 1957; Hinck *et al.*, 1964; Hinck and Sachdev, 1966). What we feel to be an equally important state of stenosis of the lumbar spinal canal has, however, only relatively recently achieved recognition (Verbiest, 1954; Epstein *et al.*, 1962; Teng and Papatheodorou, 1963; Jones and Thomson, 1968; Schatzker and Pennal, 1968; Ehni, 1969a,b; Nelson, 1973; Roberson *et al.*, 1973). Most of the reports have so far been concerned with a congenitally narrow spinal canal in which there is insufficient space to accommodate comfortably nerve roots, meningeal tissues, and extradural supporting structures. Although this state is well tolerated in early life, onset of degenerative changes with spur formation and hypertrophic spondylosis provoke symptoms of nerve root compression. Myelography is positive in these cases, showing narrowing of the oil column varying in degree to complete obstruction.

In this paper we describe a group of 28 patients who presented with low back pain associated with unilateral or bilateral sciatic or femoral neuropathy. Plain radiographs of the lumbar spine were unhelpful, and myelography was either negative or showed only minimal changes because of a normal antero-posterior diameter of the canal and lack of filling of the lateral recesses. In all cases surgical exploration of the lumbar canal was carried out on clinical

indications, by means of a full laminectomy. In all cases nerve roots were found to be incarcerated in the stenosed lateral recesses, because of hypertrophy of the facet joints. Decompression of the roots by unroofing the lateral recesses and excision of the overhanging facet joints has relieved the symptoms. This condition we have named 'occult spinal stenosis'.

The purpose of this paper is to emphasise the role of facet joint hypertrophy in causing lateral recess stenosis and entrapment neuropathy, and to describe its treatment by wide laminectomy. We believe this mechanism to be of great importance in the causation of severe disability in a large number of patients suffering from low back pain with radiation to the lower limb.

Patients and methods

Between June 1974 and May 1976, 28 of 37 patients with lumbar spinal stenosis, were shown to have the occult type (Table 1). These patients were all in the

Table 1 *Categorisation of lumbar spinal stenosis in 37 cases*

Category	Number of cases	
Occult stenosis	19	28
stenosis only	5	
with bulging disc	4	
with small HNP		
Overt stenosis	7	9
narrow canal	1	
Paget's disease	1	
spondylosis and angioma		

Address for correspondence and reprint requests: A. R. Choudhury, FRCS, Department of Neurosurgery, Derbyshire Royal Infirmary, Derby, England.

Accepted 24 November 1976

care of one neurosurgeon (JCT) so that uniformity of the criteria for selection of patients for surgery was maintained and a similar surgical procedure was applied in all cases. There were 19 males and nine females. The age span was from 17 to 61 years with the majority (20) being between 20 and 50 years (Table 2). The symptoms and signs were elucidated (Tables 3–5). Plain radiographs of the spine were normal except for alteration in disc spaces, minor spondylolisthesis, and evidence of spondylosis in some of the older patients. The spinal fluid dynamics (when measured), CSF cell count and protein level were normal. Myelography was done in 24 cases, and found negative in 18. Six showed minimal or questionable findings with shallow indentation or asymmetry of the lateral margins of the column of opaque medium being the only significant changes. Rescreen myelography was carried out in selected cases during follow-up.

Table 2 Age and sex in 28 cases of occult lumbar spinal stenosis

Age (yr)	Male	Female	Total
17–20	1	1	2
21–30	6	2	8
31–40	3	2	5
41–50	5	2	7
51–60	3	2	5
61	1	—	1
Total	19	9	28

Table 3 Symptoms in 28 cases of occult lumbar spinal stenosis

Symptoms	Number of cases
Pain	
Low back pain	28
Low back pain with unilateral sciatica	22
Low back pain with bilateral sciatica	2
Low back pain with anterior thigh pain	4
Paraesthesia	21
Severe muscle weakness: foot drop	3
Disturbance of vesical control	3

Table 4 Spinal and tension signs in 28 cases of occult lumbar spinal stenosis

Signs	Number of cases
Spinal	
Reduced mobility	24
Deformity	
diminished lumbar lordosis	12
lumbar scoliosis (convex to the side of pain)	2
local tenderness	10
Tension	
positive SLRT*	20
positive FNST†	4
positive SLRT and FNST	2

*SLRT = straight leg raising test.

†FNST = femoral nerve stretching test.

Table 5 Neurological signs in 28 cases of occult lumbar spinal stenosis

Signs	Number of cases
Motor	
slight atrophy or hypotonia in territory of affected nerve roots	4
slight weakness of extensors and flexors of digits	6
severe weakness: foot drop	3
Sensory	
hypoalgesia and hypoaesthesia	10
anaesthesia	2
hyperaesthesia	—
Reflexes	
absent ankle jerk: unilateral	6
bilateral	2
reduced ankle jerk	2
reduced knee jerk	2

SELECTION OF PATIENTS FOR SURGERY

Although myelography is a useful tool in preoperative assessment of patients with persisting root irritation, the decision to operate does not depend upon the myelographic findings alone, but on clinical evaluation of the patient. The following criteria were used as a guideline.

1. Severe sciatica or femoral pain persisting or increasing despite all conservative measures.
2. Recurrent incapacitating episodes of low back pain, sciatic or femoral pain with evidence of impairment of nerve root conduction.
3. Evidence of increasing impairment of nerve root conduction.
4. Presence of motor weakness.
5. Impairment of bladder function.

SURGICAL PROCEDURE

All cases were operated upon under general anaesthesia, with endotracheal intubation and controlled respiration. The technique has been uniform throughout the series, and the prone position was used. A midline longitudinal incision was made and minimal bleeding ensured by subperiosteal elevation of the muscles from the spinous processes and laminae. The muscles were retracted laterally to expose the facet joints. The extent of the operation was determined on the findings at exploration. Excision of the lamina was extended laterally to include part or most of the facet joint to unroof the lateral recess and decompress the nerve roots. The anteroposterior (AP) diameter and the interfacet joint distance were measured.

The AP diameter varied from 18 mm to 24 mm, and interfacet joint distance varied from 1 cm to 3 cm. Exploration of intervertebral disc spaces showed a bulging disc in only five cases and a small herniated disc in another four. Nerve roots, which were usually discoloured, were found tightly wedged laterally, and bound down and fixed by scarring and adhesions.

After the adhesions were released, the roots could be retracted easily and the nerve sheaths filled with spinal fluid to resume a normal appearance.

After careful haemostasis, the dural sac and the nerve roots were lined with gelfoam, and the wounds were closed in several layers, to avoid a dead space. Wound drainage was not used. Although free movements of the limbs were encouraged in bed, ambulation was not allowed until the sixth to seventh day after the operation, by which time the wounds were soundly healed and the patients free from pain.

FOLLOW-UP

All patients were followed up for from two months to two years. Clinical, and in some cases radiological, evaluation was made to assess the operative results which were classified as follows:

- Excellent=Pain free; unrestricted activity. Good=Occasional backache or pain in the limb but patient allowed to live and work normally and comfortably.
- Fair=Improved functional capacity but with reduced activity. Poor=No improvement.

Results

CLINICAL STUDIES

The results of the operation were assessed on the basis of subjective and objective improvement, capacity to work and residual disability (Table 6).

Pain disappeared completely in 24 cases, and paraesthesia and disturbance of bladder function when present, recovered completely postoperatively. Of the

spinal signs, deformity disappeared in most cases, except for diminution of lordosis which persisted in four cases. Local tenderness also disappeared, and movement of full range was usually restored. The tension signs disappeared in all cases: straight leg raising and femoral nerve stretching caused only a sense of tightness in the posterior muscles or the quadriceps, in some cases. Motor and sensory disturbances disappeared early, and reduced ankle jerk persisted in two cases.

In terms of complete absence of symptoms and signs and resumption of unrestricted activity, an excellent result was obtained in 24 of 28 cases. 'Much improved' implies that the patient was well satisfied with the result and could resume normal activity but had occasional backache or pain in the lower limb (two cases). 'Improved' implies recurring attacks of pain and resumption of reduced activity (two cases).

COMPLICATIONS OF OPERATION (TABLE 7)

Retention of urine was infrequent, and catheterisation was required in only six cases for 12 to 48 hours. One patient developed deep venous thrombosis soon after discharge and was readmitted and treated successfully. This patient had been bed-bound preoperatively for four weeks with severe pain. Another patient developed a CSF collection in the wound which subsided after a few aspirations. He was a young man who had had an orthopaedic laminectomy three years previously for removal of a prolapsed disc, but was pain free only for a short period. There were such dense adhesions at the site of the previous operation that it needed blunt and sharp dissection for release, resulting in a small dural tear. No patient in our series had a wound haematoma or infection. There was no mortality.

Discussion

The historic paper by Mixter and Barr in 1934, has led to the universal acceptance of the concept of the intervertebral disc herniation as the major cause of lumbar nerve root involvement and sciatic pain. In our view, preoccupation with the intervertebral disc lesion and its surgical access *via* the very limited interlaminar

Table 6 Summary of results of operation in 28 cases of occult lumbar spinal stenosis

Features	Number of cases
Symptoms	
Low back pain: absent	24
mild (occasional)	2
recurring	2
Lower limb pain: absent	24
mild (occasional)	2
recurring	2
Paraesthesia: absent	21
present	—
Disturbance of vesical function—full recovery	3 (all)
Physical Signs	
Spinal signs: absent	26 (all)
Tension signs: absent	26 (all)
Neurological Signs	
Motor disturbances: full recovery	13 (all)
Sensory disturbances: full recovery	12 (all)
Reduced ankle jerk: persistent	2
Capacity to Work	
Full	26
Reduced	2
Incapacitated	—
Subjective Result	
Cured	24
Much improved	2
Improved	2

Table 7 Postoperative complications in 28 cases of occult lumbar spinal stenosis

Complications	Number of cases
Retention of urine (12–48 hours)	6
Deep venous thrombosis	1
CSF collection	1
Wound haematoma	—
Wound infection	—
Mortality	—

approach, has been responsible for the neglect or overlooking of other causes of nerve root entrapment. Most surgeons would surely agree that progress in the understanding and management of the lumbosacral syndrome has not been conspicuous in the last 25 years. Root entrapment commonly occurs in lateral recess stenosis, which may be responsible for a syndrome which closely mimics a disc herniation (MacNab, 1971).

In an articulated spine, the lumbar canal is formed anteriorly by the posterior surface of the vertebrae and the intervertebral discs, laterally by the pedicles, and posterolaterally by the facet joints and the laminae with the spinous processes. The interlaminar space is filled up by ligamentum flavum. Normally the canal widens in the direction of the sacrum. It is somewhat pentagonal in shape, and its anteroposterior diameter varies normally from 22 mm to 25 mm with some even larger. Certain individuals, however, have canals as shallow as 15 mm or less (Ehni, 1969a,b).

In developmentally small canals, the anteroposterior diameter is shallow even in the sagittal plane. Hence encroachment on the canal by pathological processes leads to earlier, severe, and commonly bilateral symptoms.

In the normal sized canal the lateral recess is shallow. For this reason even though the hypertrophic changes are generalised, facet joint hypertrophy leads to underlying nerve root compression and the symptoms are usually unilateral.

The posterior surface of the lumbar vertebra forms a characteristic four sided figure constructed by joining the four articular processes. In the first and second vertebrae, the figure is a trapezium with base narrower than the top, while in the third it makes a parallelogram with long vertical sides. The fourth figure is practically a square, and the fifth a parallelogram elongated laterally. Alteration in these characteristics occurs in facet joint hypertrophy.

Trauma may act as a predisposing factor in the pathogenesis of lumbar spinal stenosis as this was present in all cases. The appearance of symptoms in these cases may relate to inflammatory thickening of the investing tissues secondary to acute or chronic trauma. Thinning of the intervertebral disc leads to facet joint subluxation. This may lead to abnormal sliding movements with reactive hyperaemia and hypertrophy (Epstein *et al.*, 1973). Encroachment on the available space ultimately results in root compression. Chronic irritation produces perineural fibrotic changes and adhesions (Epstein *et al.*, 1972).

A long history of low back pain, at first intermittent, but later continuous, was the most constant symptom in all our cases. Radiation of pain in the lower limb occurred months or years later. There was

no conspicuous physical sign except restriction of back mobility in most cases (24 cases). Abnormalities in neurological examination were minimal and, when present, were not adequate to provide accurate interspace localisation. Interestingly, two patients with marked foot drop had full back mobility and no tension signs.

Radiographs of the lumbar spine were not helpful and spinal tomography was not done. Myelography was done in 24 cases, and this was negative or equivocal because of normal ventro-dorsal diameter of the spinal canal or lack of filling of the lateral recess. Discrepancies in myelography are well known (Scoville *et al.*, 1948) for anteriorly placed disc herniation. Facet joint hypertrophy with consequent lateral recess stenosis causes nerve root compression but myelography is generally negative in a spinal canal of normal size because the stenotic lateral recess is away from the theca.

Surgical exploration at several levels was required. Lateral recess stenosis was suspected by the presence of shingling of the laminae or alteration of the characteristic four sided figure formed by articular processes. Observations at the time of operative exposure were the principal guides to the extent of laminectomy.

A wide laminectomy, including the excision of the overhanging hypertrophied facet, is required to expose the nerve roots in the extreme lateral recess. The adhesions were released to free the nerve roots. In only four cases was a disc prolapse found at a distal level, and these were removed.

It is significant that the four patients who underwent previous orthopaedic laminectomy for removal of a prolapsed disc, showed evidence of spinal stenosis but no recurrent disc prolapse. Wide laminectomy with release of adhesions to decompress the nerve roots relieved the symptoms. The recurrence of symptoms after initial successful excision of a prolapsed disc is commonly due to spinal stenosis secondary to degenerative changes (Crock, 1976). This is why we remove the prolapsed disc through a full laminectomy in the first instance in anticipation of later degenerative changes. Such a policy has been advocated by others (Paine and Haung, 1972; Shenkin and Hash, 1976).

Conclusions

1. Lumbar spinal stenosis is a relatively common condition. There is nothing in the onset or quality of low back pain and lower limb pain to differentiate it from that seen with a disc prolapse.
2. Myelography is negative in a great majority of cases of the occult type of stenosis.

3. Diagnosis is based on probability and confirmed only at exploration.
4. Treatment is by wide laminectomy.
5. Results of operation are gratifying.

We are grateful to Dr R. Whitaker and Dr B. Worthington, consultant neuroradiologists, for their help in radiological investigations. We would like to express our gratitude to Miss Barbara Tustin Smith for much secretarial assistance.

References

- Crock, H. V. (1976). Observations on the management of failed spinal operations. *Journal of Bone and Joint Surgery*, **58**, 193–199.
- Ehni, G. (1969a). Significance of the small lumbar canal; cauda equina compression syndromes due to spondylosis. Part 1: Introduction. *Journal of Neurosurgery*, **31**, 490–494.
- Ehni, G. (1969b). Significance of small lumbar canal; cauda equina compression syndromes due to spondylosis. Part 4: Acute compression artificially induced during operation. *Journal of Neurosurgery*, **31**, 507–512.
- Epstein, J. A., Epstein, B. S., and Lavine, L. (1962). Nerve root compression associated with narrowing of the lumbar spinal canal. *Journal of Neurology, Neurosurgery, and Psychiatry*, **25**, 165–176.
- Epstein, J. A., Epstein, B. S., Rosenthal, A. D., Carras, R., and Lavine, L. S. (1972). Sciatica caused by nerve root entrapment in the lateral recess: the superior facet syndrome. *Journal of Neurosurgery*, **36**, 584–589.
- Epstein, J. A., Epstein, B. S., Lavine, L. S., Carras, R., Rosenthal, A. D., and Sumnar, P. (1973). Lumbar nerve root compression at the intervertebral foramina caused by arthritis of the posterior facets. *Journal of Neurosurgery*, **39**, 362–369.
- Hinck, V. C., Gordy, P. D., and Storino, H. E. (1964). Developmental stenosis of the cervical spinal canal. *Neurology (Minneapolis)*, **14**, 864–868.
- Hinck, V. C., and Sachdev, N. S. (1966). Developmental stenosis of the cervical spinal canal. *Brain*, **89**, 27–36.
- Jones, R. A. C., and Thomson, J. L. G. (1968). The narrow lumbar canal. A clinical and radiological review. *Journal of Bone and Joint Surgery*, **50**, 595–605.
- MacNab, I. (1971). Negative disc exploration. An analysis of the causes of nerve root involvement in sixty-eight patients. *Journal of Bone and Joint Surgery*, **53**, 891–903.
- Mixer, W. J., and Barr, J. S. (1934). Rupture of the intervertebral disc with involvement of the spinal canal. *New England Journal of Medicine*, **211**, 210–215.
- Nelson, M. A. (1973). Lumbar spinal stenosis. *Journal of Bone and Joint Surgery*, **55**, 506–512.
- Paine, K. W. E., and Haung, P. W. H. (1972). Lumbar disc syndrome. *Journal of Neurosurgery*, **37**, 75–82.
- Pallis, C., Jones, A. M., and Spillane, J. D. (1954). Cervical spondylosis: Incidence and implications. *Brain*, **77**, 274–289.
- Payne, E. E., and Spillane, J. D. (1957). The cervical spine: An anatomico-pathological study of 70 specimens (using a special technique) with particular reference to the problem of cervical spondylosis. *Brain*, **80**, 571–596.
- Roberson, G. H., Liewellyn, H. J., and Taveras, J. M. (1973). The narrow lumbar canal syndrome. *Neuroradiology*, **107**, 89–97.
- Schatzker, J., and Pennal, G. F. (1968). Spinal stenosis, a cause of cauda equina compression. *Journal of Bone and Joint Surgery*, **50**, 606–618.
- Scoville, W. B., Moretz, W. H., and Hankins, W. D. (1948). Discrepancies in myelography. Statistical survey of 200 operative cases undergoing pantopaque myelography. *Surgery, Gynecology, and Obstetrics*, **86**, 559–564.
- Shenkin, H. A., and Hash, C. J. (1976). A new approach to the surgical treatment of lumbar spondylosis. *Journal of Neurosurgery*, **44**, 148–155.
- Teng, P., and Papatheodorou, C. (1963). Lumbar spondylosis with compression of cauda equina. *Archives of Neurology (Chicago)*, **8**, 221–229.
- Verbiest, H. (1954). A radicular syndrome from developmental narrowing of the lumbar vertebral canal. *Journal of Bone and Joint Surgery*, **36**, 230–237.