'Intermittent ischaemia' of the cauda equina due to stenosis of the lumbar canal

R. JOFFE, A. APPLEBY, AND V. ARJONA

From the Regional Neurological Centre, General Hospital, Newcastle upon Tyne

'Intermittent claudication' of the spinal cord was first described by Dejerine in 1911. In 1961, Blau and Logue described an unusual syndrome resulting from central protrusion of a lumbar intervertebral disc which gave rise to symptoms suggesting 'intermittent claudication of the cauda equina'. Since this report, a further series of cases has been described by Evans (1964).

We have recently observed five cases of cauda equina compression in which the symptoms and signs were evoked or accentuated by exertion. Three patients were shown to have central disc protrusions in the lumbar region as described by Blau and Logue (1961). The purpose of this report is to describe two further patients with intermittent claudication of the cauda equina who presented with similar clinical features but in whom the radiological and operative findings were unusual.

CASE HISTORIES

CASE 1 Mr. H.M., aged 55 years, a steelworker, was admitted to Newcastle General Hospital on 24 August 1965. His main complaint on admission was pain in the back which passed down the back of both thighs and laterally to both knees into the calves. The pain was always related to exertion and was not present at rest. He could walk about 100 yards before the onset of pain. After resting for about two to three minutes he would be able to resume walking but then would have to stop. Cold weather made no difference to his symptoms.

Two years previously, in 1963, while working as a furnaceman he suddenly developed, while bending, severe burning pain in the right calf and in the lateral part of the right foot. The pain resolved after a few weeks of rest, but he was left with numbness in the foot which persisted up to the time of the present admission. On examination he was hypertensive (blood pressure 210/110 mm. Hg). All peripheral vessels were easily palpable; other clinical abnormalities were restricted to the nervous system and it was found that he had weakness of eversion of the foot on the right side with sensory loss over the S1 dermatome. The ankle jerks were absent bilaterally. The plantar response on the right side was equivocal. There were no bladder symptoms. In the ward it was possible to provoke his symptoms by causing him to walk along a flat surface after about five minutes. Straight leg raising was

90° bilaterally. A lumbar puncture showed no block with a cerebrospinal fluid protein level of 70 mg./100 ml.

Radiology Straight films of the lumbar spine showed dense bone surrounding the posterior articulations and this was ascribed to degenerative changes in the joints.

Myelographic examination The myelogram (Fig. 1) revealed narrowing of the lumbar spinal canal at the level of the posterior articulations throughout the lumbar region; this was produced by posteriorly situated oval masses of dense bone. The effective canal at the level of the disc spaces seemed to measure only 4 or 5 mm. There was complete obstruction to the flow of Myodil at the level of the L4-5 disc space and the column was displaced posteriorly a distance of about 3 mm. at this level.

The findings thus suggested a stenosis of the lumbar canal with a mild disc protrusion at L4-5 causing a complete block. The degree of disc protrusion was such that one would not have expected such a dramatic obstruction had the canal been of normal calibre.

Operative findings A subperiosteal laminectomy of L4 was performed. The lamina was extremely thick antero-posteriorly and it was difficult to remove as the bone appeared to be much harder than normal. When the canal was reached, after removing a normal looking ligamentum subflavum, the dural sac was seen to be non-pulsatile and it was obviously constricted by the heavy lamina. At the level of the upper border of L4 the canal widened and the dura pulsated normally. The L5 lamina was also very thick and the canal was narrow at this point. Laminectomy of L5 was therefore done extending laterally to remove the medial part of the articular processes.

After removing both laminae the dural sac filled with cerebrospinal fluid and started to pulsate. The L5 and S1 roots were visualized bilaterally and were lying free in the canal. The dural sac was then retracted laterally to explore the anterior surface of the canal. The L4-5 disc was soft and protruding slightly, but not enough to cause any degree of compression.

Post-operatively At follow-up examination on 6 January 1966 the patient was walking very much better but still had absent ankle reflexes. The plantar responses were flexor. He was able to walk any distance without recurrence of pain, but still had some sense of stiffness in the legs.

CASE 2 Mr. G.C., aged 58 years, a security officer, was admitted to Newcastle General Hospital on September 20, 1965. His main complaint was numbness of the left foot

FIG. 1. Complete obstruction to the downward flow of Myodil at the upper border of the L4/L5 disc space in case 1. The arrows point to the dense mass of bone which is narrowing the canal from its posterior aspect opposite L4. The dense bone at L5 is not clearly seen because its anterior limit is not delineated by Myodil.

and leg for two years. He was well until two years before admission when he developed pain and numbness of the left foot and calf which was brought on by standing for long periods or by walking. It never occurred at rest. He found that if he did not rest immediately the pain would come on, and he would then develop pain in the buttock on the left side. Further exercise brought on similar dysaesthesiae below the knee on the right. He found that as time went on he was becoming more and more incapacitated and now could barely walk 50 yards without having to rest. He was seen first by a vascular surgeon as a possible case of vascular intermittent claudication but after numerous tests, including studies of radioactive sodium exchange during exercise, it was felt that the vessels were not diseased and that the pain was not due to true intermittent claudication.

On examination he was overweight and well built. Blood pressure was 150/90 mm. Hg. All peripheral limb vessels were easily palpable. In the central nervous system, apart from ankle reflexes which were only just elicitable, there were no significant abnormalities. There

was no sensory loss in the lower extremities. Walking in the ward precipitated aching and numbness in the left foot and leg. There was no sphincter disturbance. Lumbar puncture was attempted but failed and myelography was performed by the cisternal route.

Radiology The findings in this case were very similar to those in case 1. There was an almost complete hold-up of Myodil at the upper border of L3. The lumbar canal was narrowed in an antero-posterior direction by dense masses of bone seen posteriorly in the region of the articular processes and there did not appear to be any significant degree of disc protrusion (Fig. 2).

Re-examination after an interval showed that most of the Myodil had reached the terminal theca and was now held up completely below the L4-5 disc space (Fig. 3).

Operative findings Laminectomy of L4 was performed. The lamina was very thick and the bone hard and difficult to remove. The canal was very narrow and the dural sac was constricted by the thickened lamina and not pulsating. The narrow canal extended up to the upper border of L3 and down to the lower border of L5. The L3 and

FIG. 2

FIG. 2. The arrows point to the posterior masses of dense bone which narrow the lumbar canal in case 2.

FIG. 3. The same patient as in Fig. 2 after some days, showing that most of the Myodil had gradually passed the obstruction.

L5 laminae were removed, and these too were extremely thickened.

After this decompression the dural sac filled with cerebrospinal fluid and pulsated normally. The anterior part of the canal was explored and there was no evidence of disc protrusion.

Post-operatively At follow-up examination on January 6, 1966 the patient was well and able to walk as well as ever; he was symptom-free.

DISCUSSION

Whereas in the past narrowing of the bony vertebral canal with cord compression has been described in association with diseases of bone like achondroplasia (chondrodystrophia foetalis) (Summita, 1910;

Spillane, 1952) and with bony deformities like spina bifida (Sarpyener, 1947), it was Verbiest (1954) who first described narrowing of the vertebral canal without any developmental anomaly or bone disease being present. He described seven cases of narrow vertebral canal presenting with symptoms of 'claudication of the cauda equina'. In 1955 he described a further six cases and maintained that the narrowing was developmental and not due to bone disease. Subsequently, Epstein, Epstein, and Lavine (1962) described their clinical, radiological, and operative findings in 29 such cases.

Two of the cases described by Blau and Logue (1961) had narrow lumbar canals at operation and in one of these there was only a minor degree of disc

protrusion. The pathophysiology of the syndrome of 'intermittent claudication' of the cauda equina has been fully discussed by Blau and Logue (1961); these authors postulate that an ischaemic neuropathy could account for the symptoms and that the ischaemia is produced by compression of nerve roots during exercise as a result of disc protrusion. It is apparent, however, from the cases of Verbiest (1954, 1955) and from our own two cases that ischaemia of the cauda equina can be produced by stenosis of the lumbar canal without disc protrusion or with only a very slight degree of disc protrusion which would not be significant in a lumbar canal of normal width.

Highman (1965), in a review of patients with complete myelographic block in lumbar degenerative disease, reported two patients with stenosis of the lumbar canal, one of whom had 'intermittent claudication' of the cauda equina. Teng and Papatheodorou (1963) described 30 cases showing constriction of the spinal canal by hypertrophic laminal arches, pedicles, and articular facets with or without disc herniation. They ascribed the hypertrophic articular facets to spondylosis but whatever the aetiology of the condition it is clear that the bony hypertrophy does bulge into the spinal canal and narrows its antero-posterior diameter.

Although, in severe cases, the hypertrophic bone may be detected by careful scrutiny of plain radiographs, it is extremely difficult to measure the antero-posterior diameter of the lumbar spinal canal on such films. Myelography, however, shows the hypertrophic bone more strikingly because the indentations in the posterior border of the Myodil column are clearly seen. Further, myelography will show the site of maximum obstruction and also permits fairly accurate measurement of the antero-posterior diameter of the canal. It seems likely that the hypertrophic bone is a result of degenerative disease and that the effects are much more profound in those patients who already have a spinal canal which, in diameter, approaches the lower limit of normal variation.

While intermittent ischaemia of the cauda equina due to prolapsed intervertebral disc is well known, the syndrome of a narrow vertebral canal, due to hypertrophic bone but without actual bony disease, which may present with intermittent ischaemia of the

cauda equina, is not nearly so well documented in the neurological literature.

Accurate pre-operative assessment is important since this combination of circumstances requires a more extensive bony decompression than is necessary in those cases which are caused by disc protrusion.

SUMMARY

Two further cases of so-called 'intermittent claudication' of the cauda equina are described, due to stenosis of the lumbar spinal canal.

It is suggested that in a patient with a rather narrow spinal canal the formation of sclerotic bone posteriorly, probably as a result of degenerative disease, can cause sufficient obstruction to give rise to intermittent ischaemia of the cauda equina even when no marked disc protrusion is present. Laminectomy of the affected vertebrae with decompression of the theca gives immediate relief of symptoms.

We are very grateful to Dr. J. N. Walton for allowing us to publish these cases and for his constant advice and encouragement, and we are also grateful to Mr. J. Hankinson for permission to publish the findings at operation.

REFERENCES

- Blau, J. N., and Logue, V. (1961). Intermittent claudication of the cauda equina. *Lancet*, **1**, 1081-1086.
- Dejerine (1911). La claudication intermittente de la moelle épinière. *Presse méd.*, **19**, 981-984.
- Epstein, J. A., Epstein, B. S., and Lavine, L. (1962). Nerve root compression associated with narrowing of the lumbar spinal canal. *J. Neurol. Psychiat.*, **25**, 165-176.
- Evans, J. G. (1964). Neurogenic intermittent claudication. *Brit. med. J.*, **2**, 985-987.
- Highman, J. H. (1965). Complete myelographic block in lumbar degenerative disease. *Clin. Radiol.*, **16**, 106-111.
- Sarpyener, M. A. (1947). Spina bifida aperta and congenital stricture of the spinal canal. *J. Bone Jt Surg.*, **29**, 817-821.
- Spillane, J. D. (1952). Three cases of achondroplasia with neurological complications. *J. Neurol. Neurosurg. Psychiat.*, **15**, 246-252.
- Sumita, M. (1910). Beiträge zur Lehre von der Chondrodystrophia foetalis (Kaufmann) und Osteogenesis imperfecta (Vrolik), mit besonderer Berücksichtigung der anatomischen und klinischen Differentialdiagnose. *Dtsch. Z. Chir.*, **107**, 1-110.
- Teng, P., and Papatheodorou, C. (1963). Myelographic findings in spondylosis of the lumbar spine. *Brit. J. Radiol.*, **36**, 122-128.
- Verbiest, H. (1954). A radicular syndrome from developmental narrowing of the lumbar vertebral canal. *J. Bone Jt Surg.*, **36B**, 230-237.
- (1955). Further experience on the pathological influence of a developmental narrowness of the bony lumbar vertebral canal. *Ibid.*, **37B**, 576-583.