Acta orthop. scand. 45, 683–692, 1974

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SPINAL INTERMITTENT DYSBASIA

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Accepted 25.iv.74

The symptom of intermittent claudication or dysbasia is common and well known as an indication of occlusive arterial disease, with pain in the lower extremities being produced by muscular ischaemia during exercise and relieved when exercise is interrupted. In this connection the term "dysbasia" is nowadays often preferred to "claudication", as pain during walking and inability to continue walking rather than limping is the characteristic feature of this group of disorders. A number of other conditions can produce symptoms almost identical with those of arterial intermittent dysbasia and are often referred to as "pseudoclaudication". This report will deal with the clinically well-defined condition where a spinal stenosis, causing compression of the cauda equina, is the origin of the disorder. A review of the literature on this subject will be given. The condition is rather uncommon and during the period 1970 through 1973 only six patients have been operated upon with this diagnosis at the Department of Orthopaedic Surgery II, University of Gothenburg. Four of these cases will be described in detail as they illustrate the diagnostic and therapeutic difficulties of this condition.

CASE REPORTS

Four patients with typical histories of spinal intermittent dysbasia and with clinical and radiological characteristics of this syndrome will be reviewed. The pre-operative investigations in all cases include studies of the arterial blood flow by means of plethysmography and iscillometry, in no case revealing any abnormality.

Case 1—E.R.

A 55-year-old factory worker, first seen at our clinic in September 1970, who had for one year had an increasingly disabling low back pain radiating into both legs brought on by walking 100–200 metres. Pain was predominant in the right leg and

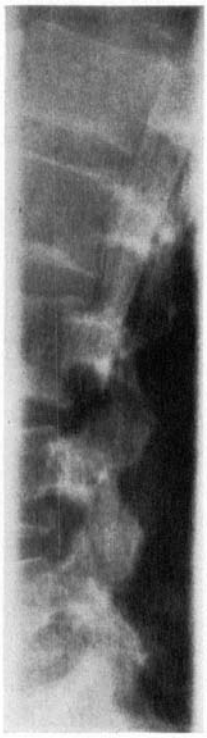

Figure 1. Case 1. Myelography with water-soluble contrast medium. Almost total block at the level of the L.II–L.III disc.

accompanied by paraesthesias and weakness; the muscles of the legs "felt like jelly". The pain and weakness disappeared after sitting down for a few minutes but paraesthesias could persist also during rest. On examination a diminished Achilles tendon reflex and slightly weaker triceps surae muscle on the right side were the only positive findings. Peripheral pulses were normal. Myelography with contrast injection between L.I and L.II showed an almost total block at the level of the L.II–L.III disc (Figure 1). At operation in October 1970 a slight protrusion of this disc was noted but the dural sac pulsated normally and the nerve root was freely movable. At the next lower level the pulsations were extinguished but occurred normally after the right half of the L.III lamina had been removed. A postoperative myelography was made to exclude the presence of an intradural expansive process below the explored levels. The filling of the subarachnoidal space was now complete, but with deep impressions both anteriorly and posteriorly on a level with the three lowest lumbar discs (Figure 2). No further intervention was made at this stage as the patient became symptom-free after the operation except for a slight persisting discomfort in his left leg, and also could go back to work. He was well for 2 years but from December 1970 symptoms of the same character and severity as before successively returned. Decompressive laminectomy was performed in April 1973. Especially at the L.IV–L.V level a marked reduction of the space of the spinal canal was present with failure of the dural sac to pulsate below the level. In addition to a reduced distance between the bulging discs and the

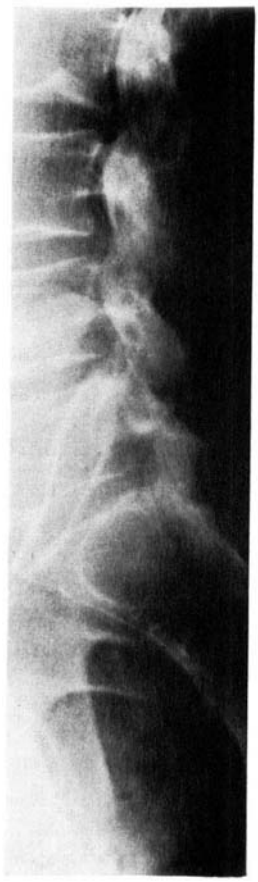

Figure 2. Case 1. Myelography after removal of the right half of the L.III lamina shows filling of the subarachnoidal space with deep impressions both anteriorly and posteriorly on a level with the lowest discs.

abnormally thick laminae, a reduced interpeduncular distance was noted. The laminae of L.III, L.IV and L.V were removed together with sufficient parts of the facets of the intervertebral joints to unroof the lateral recesses and expose the nerve roots. After operation the symptoms gradually subsided and the patient returned to his rather heavy work after 6 months.

Case 2—G.B.

A 46-year-old truck driver was operated upon in 1963 with extirpation of a sequestrum from the L.IV–L.V disc after a 6 months' history of right-sided sciatica with clinical and myelographical signs of a disc herniation. The patient was free from pain for 3 months after the operation but then low back pain re-appeared radiating into both legs, most pronounced in the left leg. A myelography, done 8 months after the operation, showed defective filling of the subarachnoidal space at the L.IV–L.V disc level, interpreted as an effect of postoperative adhesions. The patient was recommended rest and treated with analgesics without relief. He had

not been able to work during the period from 1963 to December 1970, when he was again admitted to our clinic. He was then unable to walk more than 100 metres because of weakness and numbness in his left leg and pain in the lumbosacral region radiating into both legs. All symptoms subsided at rest. Tendon reflexes were normal and no motor weakness was found. Sensation was slightly impaired in the lateral aspect of his left foot and the straight leg-raising test was positive at about 60 degrees bilaterally. Myelography showed a marked anterior impression with incomplete filling of the subarachnoidal space at the L.IV–L.V disc level. A herniated disc was suspected and a partial left-sided hemilaminectomy was carried out in March 1971. A bulging disc and osteophytes on the margins of the vertebral bodies were the only pathological findings. In spite of the limited decompression the patient was completely relieved of his symptoms and able to return to work and he has remained symptomless since then.

Case 3—A.A.

A 67-year-old pensioner who had for 7 years complained of pain in the sacral region and in both legs, most prominent in the calves, with numbness and tingling sensations in the feet and ankle regions. The symptoms were evoked by walking for 3–4 minutes and disappeared completely after a few minutes' rest. Peripheral pulses were normal and there were no signs of circulatory disturbance. No neurological abnormalities were found. Radiographs of the lumbar spine showed advanced spondylarthritis with large osteophytes surrounding the greatly reduced three lowest discs and also affecting the intervertebral joints. Myelography showed deep impressions in the subarachnoidal space on a level with the three lowest discs with medial dislocation of the corresponding nerve roots. In November 1972 decompressive laminectomy of L.III, L.IV and L.V was performed. Beginning distally, we noted a much reduced sagittal diameter of the spinal canal, from which the dural sac protruded into the laminectomy opening. The normal pulsations of the dura synchronus with respiration were absent at this stage but appeared after completion of the laminectomies. The decompressive procedure was not extended laterally to unroof the lateral recesses. Two weeks after operation the patient could walk for 20 minutes without the distress that would before have forced him to rest after 4 minutes. One year after operation he is completely symptom-free in his right leg, but experiences a slight aching and tiredness in his left leg after about 30 minutes' walk, causing him to rest for a short while.

Case 4—B.M.

A 43-year-old labourer who had for 3 years suffered from low back pain radiating into both legs and diffuse numbness in both legs after walking for 10–15 minutes. He was able to work only for short periods. Complete relief occurred during rest and the patient noted that he could ride a bicycle without distress. At this stage decompressive laminectomy was planned. In December 1972 acute aggravation occurred with pain from the left buttock along the dorsal aspect of the left leg to the heel. This pain was continuous, not disappearing during rest, regardless of position. When the patient was seen at our clinic in February 1973 his left

Figure 3. Myelography showing total block at the level of the L.IV–L.V disc and impressions in the subarachnoidal space at the levels of the two next cranial discs.

Achilles tendon reflex was absent but no motor or sensory disturbances were noted. The straight leg-raising test provoked pain in the back at 45 degrees bilaterally. Peripheral pulses were normal. A walk-way test was interrupted after 290 metres because of increasing, cramp-like pain in both legs. Ergometry showed excellent physical capacity and no symptoms developed. Myelography revealed a total block at the level of the L.IV–L.V disc and deep impressions in the subarachnoidal space at the levels of the two next cranial discs (Figure 3). In April 1973 decompressive laminectomy of L.IV and L.V was performed and the dural sac and its contents were found to be compressed in the abnormally narrow spinal canal. No cerebrospinal fluid leaked through an accidental opening in the dura. The sagittal diameter of the spinal canal was much reduced by abnormally thick and horizontally placed laminae and by hypertrophic ligamenta flava. In addition, a disc sequestrum was found, herniating from the ruptured L.V–S.I disc, and compressing the left S.I root. The patient was relieved from his symptoms after operation except for a slight and occasional aching in his left ankle region and he returned to full work 5 months after operation.

DISCUSSION

Verbiest in 1954 was the first to associate the clinical picture of radiating pain, sensory disturbance and impairment of motor function in the lower limbs, precipitated by walking or standing and relieved by rest, with a developmental narrowing of the lumbar canal. Since then several authors have described this clinical entity and thrown light upon its pathogenesis (Epstein et al. 1962, Teng & Papatheodorou 1963, Joffe et al. 1966, Spanos & Andrew 1966, Jones & Thomson 1968, Schatzker & Pennal 1968, Kavanaugh et al. 1968, Ehni et al. 1969, Weiser 1971, Weber & deKlerk 1973, Nelson 1973).

The significance of a congenital narrow lumbar spinal canal with shortened interpeduncular distance, short, broad peduncles and abnormally thick and horisontally placed laminae as the basic disorder is pointed out in many reports (Jones & Thomson 1968, Schatzker & Pennal 1968, Ehni et al. 1969, Weber & deKlerk 1973). This narrowing of the spinal canal is in itself symptomless, but when degenerative changes affecting intervertebral discs, vertebral bodies and intervertebral joints further encroach on the space in the spinal canal, the function of the cauda equina is interfered with and symptoms are produced. Several explanations have been given regarding the mechanism of this interference. Kavanaugh et al. (1968) suggested that increased pressure of the cerebrospinal fluid below the level of the block causes collapse of veins with resultant anoxia of the nerve trunks. Blau & Rushworth (1958) showed that exercising a hindlimb in the mouse produced local increased vascularity in the corresponding segment of the spinal cord and dilatation of the veins of the spinal nerve roots. Blau & Logue (1961) suggested that the symptoms of spinal intermittent dysbasia were produced by increase in pressure on abnormally confined nerve roots due to vasodilatation during exercise. If the space occupied by the nerve root is so diminished that dilatation of the vessels corresponding to the functional demands is made impossible, anoxia of the nerve tissue will also impair its function. Many authors stress the mechanical factors, noting the correlation between posture and symptoms. Breig (1960) in cadaver studies showed that extension of the lumbar spine caused a shortening of the spinal canal, slackening of the ligamenta flava with increase in their cross-sectional area, narrowing of the intervertebral foramina and slight posterior protrusion of the discs, all factors contributing to a narrowing of the spinal canal.

Clinical characteristics

Symptoms. The clinical history of pain, paraesthesia and weakness in the legs brought on by exercise and relieved by rest is strongly suggestive of arterial occlusive disease affecting the arterial supply to the lower limbs. Close questioning, however, will reveal details that differentiate arterial from spinal intermittent dysbasia. The patient with occlusive disease describes an aching, cramp-like pain in the calves and thighs brought on by walking a fairly constant distance and also by other activities such as cycling. The pain is promptly relieved when the patient stands still for a few minutes. Walking up stairs or an incline will precipitate the pain sooner, whereas walking at a slower pace will delay the onset of pain. In patients with arterial occlusive disease paraesthesias and weakness are usually not prominent symptoms.

In spinal intermittent dysbasia the pain is often vaguely described as located in the low back extending to the buttocks and legs, as a rule without a radicular pattern. The distress is often dominated not by pain but by paraesthesias and weakness, described as numbness, tingling, heaviness and unsteadiness. The symptoms can be precipitated not only by walking but also by certain positions such as standing and even lying with the back straightened and often are not relieved unless the patient sits down or bends forward. In some cases symptoms develop when the patients walk down stairs but not when they climb stairs, and a patient who can walk only for a few minutes may be able to ride a bicycle long distances without distress.

Signs. The presence of normal peripheral pulses both at rest and after exercise in a patient with a history of intermittent dysbasia should make the examiner suspect a spinal origin of the disorder. There is no specific clinical abnormality associated with spinal intermittent dysbasia and this discrepancy between symptoms and signs is often striking. Movements of the lumbar column are often almost or totally unrestricted and painless and the straight leg-raising test may be normal. Sensory disturbances are sometimes found, usually in the L.V. and S.I. dermatomes, but are often absent. Muscular atrophy and weakness may occur but are seldom pronounced. Reflex asymmetry is a common finding, the Achilles tendon reflex being the most frequently affected. A repeat examination after the patient has provoked symptoms by walking an appropriate distance will often reveal neurological signs of great diagnostic importance. One or more tendon reflexes may

have disappeared and motor and sensory disturbances, as a rule without a distinct radicular pattern, may be demonstrated.

Radiological findings. Radiographs of the lumbar spine usually show spondylosis with narrowing of disc spaces and osteophyte formation. If advanced, these changes alone might be responsible for the narrowing of the spinal canal and compression of the cauda equina that produces the symptoms. Developmental abnormalities of the neutral arches resulting in reduced sagittal and transverse diameters of the spinal canal, however, have been shown by many authors to be of fundamental importance (Jones & Thomson 1968, Weber & deKlerk 1973, Nelson 1973). Shallowness of the lateral recesses containing the nerve roots is an important part of the anomaly and must be considered at operation (Schatzker & Pennal 1968, Nelson 1973). Several attempts have been made to produce guides for measuring the size of the spinal canal on plain radiographs (Jones & Thomson 1968, Weber & deKlerk 1973). To obtain a definite diagnosis, myelography should be performed. The most common finding is an hourglass-like deformation of the subarachnoidal space at one or more levels, with the encroachments as a rule most marked from the dorsal and ventral aspects. Often a complete block is present or the nerve roots may be so tightly packed in the compressed dural sac that a myelographic picture suggesting epidural injection is produced.

Treatment

Although spontaneous regression of symptoms has been reported (Kavanaugh et al. 1968), surgical treatment as a rule is necessary. The decompressive laminectomy must be sufficient both longitudinally and laterally to relieve completely the stenosis at all engaged levels. A partial, or even total, facetectomy may be necessary to decompress adequately the nerve roots in the lateral recesses.

The return of symptoms in our case 1 was regarded as a consequence of inadequately extended decompression, both longitudinally and laterally, at the first operation. The persistance of some symptoms in case 3 may also be interpreted as a result of inadequate lateral decompression. In case 2, however, permanent relief was obtained after partial hemilaminectomies. As shown by myelography, the stenosis in this case was limited to one level. In case 4 a history of acute debuting sciatica with the clinical characteristic of lumbar disc herniation was superimposed upon the long-standing symptoms suggestive of spinal

intermittent dysbasia and at operation both diagnoses were verified. Several similar cases are reported in the literature and it may be valuable to keep the possibility of such coincidence in mind when planning disc surgery.

SUMMARY

Spinal abnormalities causing a reduction of the space within the spinal canal and a compression of its contents have for the last two decades been reported by several authors as a clinical entity with symptoms strongly suggestive of the "intermittent claudication" or "intermittent dysbasia" well known as a symptom of arterial occlusive disease compromising the blood supply to the lower limbs. The term "spinal intermittent dysbasia" is proposed for this clinical entity.

Some clinical characteristics that provide valuable clues in distinguishing a spinal from a vascular disorder are presented and four cases known to the authors are reviewed.

Myelography should be performed to verify the diagnosis and give information necessary in planning the operation. The general opinion that treatment consists of laminectomies of adequate extent both longitudinally and laterally to decompress the dural sac as well as the nerve roots is consentient with our experience.

REFERENCES

Blau, J. N. & Rushworth, G. (1958) Observations on the blood vessels of the spinal cord and their responses to motor activity. *Brain* **81**, 354.

Blau, J. N. & Logue, V. (1961) Intermittent claudication of the cauda equina. An unusual syndrome resulting from central protrusion of a lumbar intervertebral disc. *Lancet* **i**, 1081.

Breig, A. (1960) *Biomechanics of the central nervous system.* Almqvist & Wiksell, Stockholm.

Ehni, G., Clark, K., Wilson, C. B. & Alexander, E. (1969) Significance of the small lumbar spinal canal: Cauda equina compression due to spondylosis. *J. Neurosurg.* **31**, 490.

Epstein, J. A., Epstein, B. S. & Lavine, L. (1962) Nerve root compression associated with narrowing of the lumbar spinal canal. *J. Neurol. Neurosurg. Psychiat.* **25**, 165.

Joffe, R., Appleby, A. & Arjona, V. (1966) "Intermittent ischemia" of the cauda equina due to stenosis of the lumbar canal. *J. Neurol. Neurosurg. Psychiat.* **29**, 315.

Jones, R. A. C. & Thomson, J. L. G. (1968) The narrow lumbar canal. *J. Bone Jt Surg.* **50**-B, 595.

Kavanaugh, G. J., Svien, H. J., Holman, C. B. & Johnson, R. M. (1968) "Pseudoclaudication" syndrome produced by compression of the cauda equina. *J. Amer. med. Ass.* **206**, 2477.

Nelson, M. A. (1973) Lumbar spinal stenosis. *J. Bone Jt Surg.* **55**-B, 506.

Schatzker, J. & Pennal, G. F. (1968) Spinal stenosis, a cause of cauda equina compression. *J. Bone Jt Surg.* **50**-B, 606.

Spanos, N. C. & Andrew, J. (1966) Intermittent claudication and lateral lumbar disc protrusion. *J. Neurol. Neurosurg. Psychiat.* **29**, 273.

Teng, P. & Papatheodorou, C. (1963) Lumbar spondylosis with compression of the cauda equina. *Arch. Neurol. (Chic.)* **8**, 221.

Weber, F. A. & deKlerk, D. J. J. (1973) Spinal stenosis. *S. Afr. med. J.* **47**, 207.

Weiser, H. I. (1971) Neurogene claudication intermittens. *Z. Orthop.* **108**, 689.

Verbiest, H. (1954) A radicular syndrome from developmental narrowing of the lumbar vertebral canal. *J. Bone Jt Surg.* **36**-B, 230.

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