Short report

Pseudoclaudication syndrome caused by a tumour of the cauda equina

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SUMMARY A typical case of pseudoclaudication syndrome (neurogenic intermittent claudication), where the cause of the lumbar spinal canal narrowing was an intraspinal neoplasm, is reported. A neoplasm as the cause of this syndrome seems to be very rare, possibly because of the progressive nature of the space-occupying lesion and hence the rapid development of other predominant symptoms.

The pseudoclaudication syndrome (Kavanaugh et al., 1968) is now a well-defined clinical complex. It is also called neurogenic intermittent claudication (Evans, 1964; Weiser, 1971), spinal intermittent dysbasia (Olsson and Sodemann, 1974), intermittent claudication of the cauda equina (Blau and Logue, 1961), and intermittent ischaemia of the cauda equina (Joffe et al., 1966).

Its symptoms resemble intermittent claudication caused by arterial insufficiency, but thorough questioning of the patient reveals certain differentiating features. The most common symptom is leg pain brought on by walking, but there may instead be other types of sensory symptoms such as paraesthesiae, numbness, or sometimes more bizarre sensations. Motor symptoms such as weakness, drop foot, staggering gait, or ataxia may also occur either alone or together with sensory disturbances. Intermittent bladder paresis, as described by Fagius and Westerberg (1976), may occur in exceptional cases.

It is characteristic of the syndrome that in most cases symptoms are also elicited by standing upright for a while and that, when symptoms have been brought on by walking, standing still is not enough to ameliorate them. The patient has to break the upright posture by sitting down, squatting down, or bending forward. On clinical examination at rest either nothing abnormal at all is found or signs of affection of the cauda equina. No spinal cord symptoms occur.

The cause of the syndrome is entrapment of the cauda equina by a localised narrowing of the spinal canal. In most cases myelography will reveal a total or almost total occlusion of the subarachnoid space at the level of the third to fifth lumbar vertebra. Pathogenetically an intermittent pressure on the cauda equina fibres seems to be essential, and different explanations for this are suggested. Blau and Logue (1961) and Evans (1964) stress the possibility of relative or absolute ischaemia of the cauda equina caused by the increased flow of nerve impulses on muscular exercise. Direct pressure by disc protrusion combined with nerve root thickening during standing and walking is considered by van Gelderen (1948), Verbiest (1954, 1955), and Brish et al. (1964). Kavanaugh et al. (1968) suggest the cause to be pressure on the nerve fibres by venous overfilling below the subarachnoid block.

The narrowing of the spinal canal may be any space-occupying process, but thickening of the ligamentum flavum (Kavanaugh et al., 1968), bony hypertrophy, and spondylosis (Joffe et al., 1966; Schatzker and Pennal, 1968; Weiser, 1971; Olsson and Sodemann, 1974) or congenital narrowing (Verbiest, 1954, 1955; Hancock, 1967) seem to be most usual. Weiser (1971) reported compression fracture of a vertebra, and Silver et al. (1969) a medially situated disc protrusion as the cause of the syndrome. A few cases of similar disturbances but without symptoms on standing, caused by a laterally situated disc protrusion, are described by Spanos and Andrew (1966) and Silver et al. (1969).

We have recently seen a patient with a pseudoclaudication syndrome caused by a tumour.

Case report

A 60 year old woman had been in excellent health until early summer 1973, when lumbar back pain developed insidiously. After enduring the pain for
one year she finally sought medical advice. A radiograph of the lumbar spine in June 1974 revealed an area of osteolytic destruction within the body of the fourth lumbar vertebra. A metastatic deposit was suspected, and a search for a supposed primary tumour was instituted. Radiographs of the lungs, breasts, kidneys, and gastrointestinal tract, a scintigram of the thyroid, and gynaecological examinations were all normal. Several attempts to obtain a histopathological diagnosis by means of needle biopsy from the diseased vertebra failed. Radiotherapy was given in September 1974 with partial relief of the pain.

The patient was referred to us in February 1975 for neurological evaluation. She gave the following history. In addition to her constant backache, since July 1973 she had had position-dependent pain in both legs, radiating down the ventral and lateral aspects of the thighs, the medial aspect of the legs below the knees and sometimes but not always reaching the soles of the feet. The pain was accompanied by topographically ill-defined paraesthesiae in the form of pins and needles in both legs. The pain and the paraesthesiae were never present in the supine or sitting position, but were regularly brought on by standing still for a few minutes or by walking 200 metres. The pain and paraesthesiae vanished almost immediately when she sat down. The lumbar back pain was not much altered by changes of the body position. There was no disturbance of micturition. On neurological examination there were no signs of weakness, muscular atrophy, or sensory disturbance. The plantar responses were flexor. The only abnormal finding was a questionable diminution of the left patellar reflex, all other tendon reflexes being normal.

A new radiograph of the lumbar spine showed progression of the osteolytic changes, particularly in the posterior part of the vertebral body. In addition, anteriorly situated sclerotic changes had now appeared. On positive contrast myelography with metrizamide (Amipaque) a ventrally located impression at the level of the fourth lumbar vertebra was seen to reduce the subarachnoid space to about half of its normal sagittal width.

At operation an epidural tumour with a glassy, greyish appearance was found. As the tumour bled very easily, the surgeon thought it unwise to try to achieve effective decompression, and only a small biopsy sample was taken. Consequently her symptoms remained unchanged. The histopathological diagnosis was poorly differentiated carcinoma, probably adenocarcinoma. No conclusion regarding the probable primary site of the tumour could be drawn.

Discussion
As already mentioned, the anatomical basis for the pseudoclaudication syndrome, the marked narrowing of the subarachnoid space, might theoretically be of any kind. A tumour would seem a highly conceivable cause—and yet this seems to be very rare. Wilson (1969), in a review, says that "compression of cauda equina roots from other causes (... neoplasms ...) rarely produces an intermittent claudication syndrome." In this statement he refers to Jennett (1956), but the latter reports on cauda equina compression caused solely by prolapsed intervertebral discs, and furthermore he does not describe any pseudoclaudication symptoms. Bradley (1974, 1975) mentions that the syndrome may arise from "tumours like ependymomata", but his references (Verbiest, 1954, 1955; Joffe et al., 1966) do not describe cauda equina tumour as the cause of the syndrome.

In a very recent paper Fearnside and Adams (1978) present a review of no fewer than 70 consecutive patients with a cauda equina tumour (admitted over a 40 year period), and among these none had symptoms of the pseudoclaudication syndrome. It is interesting to note that 35 of their patients (50%) experienced back pain with or without sciatica in recumbency, most often nocturnally, compelling the patients to get up and walk around for relief. The dependence of pain on body position in these patients is in a way the reverse of what is the rule in the pseudoclaudication syndrome. Allen (1930; cited by King, 1975) also states that back pain in recumbency is a prominent symptom of cauda equina tumours.

We have found no case reports describing patients with a pseudoclaudication syndrome caused by a tumour affecting the cauda equina. A possible explanation for this may be the progressive nature of a tumour—intermittent disturbances may occur for a short time and be followed by persistent and perhaps more alarming symptoms. The short period of intermittent troubles may thus be forgotten by the patient at the time of admission to hospital.

References
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