migraine, Raynaud’s disease, papilloedema, ophthalmic artery aneurysm and osteoma. To our knowledge, no case has been ascribed to protein C deficiency.

Protein C is a vitamin K dependent plasma glycoprotein. When activated, it inhibits coagulation by inactivating factors V and VIII. It facilitates fibrinolysis by elevating plasminogen activator levels. Deficiency is inherited as an autosomal dominant trait. Since 1981, a number of families have been reported to have protein C deficiency and recurring thrombo-embolic events. Like antithrombin deficiency, protein C deficiency is typically associated with venous thrombosis and pulmonary emboli. The problem has generally not been associated with arterial disease or neurological symptoms. Recent reports indicate, however, that deficiency of either protein C or serum antithrombin can cause stroke by venous thrombosis.6-8 Paradoxical embolism from a venous source might also produce neurological symptoms in this setting. A causal relationship between protein C deficiency and amaurosis fugax cannot be proved in our patient, but we submit that the connection is plausible.

Protein C deficiency may go unrecognised for many years and does not inevitably present thrombotic complications. Some patients with amaurosis fugax have no clear explanation for their symptoms. Some of them may have hypercoagulable states characterised by deficiencies of clotting inhibitors. If so, appropriate laboratory screening may lead to a more definitive diagnosis and therapy for these individuals.

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Exercise provoked faecal incontinence in spinal stenosis

Sir: Lumbar canal stenosis, first delineated by Verbiest in the 1950s,1 has been linked for many years with intermittent neurological symptoms in the lower limb. Blau and Logue, in 19612 introduced the term “claudication of the cauda equina”, because of similarities with symptoms caused by peripheral vascular disease in the legs.

Symptoms classically associated with lumbar canal stenosis include pain, paraesthiesiae and/or weakness in the legs, occurring on walking or sometimes with a change in posture, which are relieved by rest, and are associated with minor sensorimotor asymmetry but normal pedal pulses. We report a case of faecal incontinence provoked by exercise in a patient with lumbar canal stenosis, whose symptoms remitted following lumbar decompression. Such an association has not previously been described.

Mr H.H. first presented in April 1979 aged 62 years, with a three year history of dragging of the right leg occurring after he had walked several miles. In March 1978 he became suddenly impotent, and had noticed increased leg weakness since that time. He had had low back pain for 10 years but was otherwise asymptomatic, and had suffered no bladder or bowel disturbance.

Examination revealed reduced muscle bulk in the right thigh, and increased right knee and ankle reflexes, with normal sensation and intact cremasteric and anal reflexes. CSF studies and visual evoked responses were unhelpful, and metrizamide myelography showed a small arachnoid cyst posteriorly in the mid-dorsal region, with minor disc prolapse at the L 4/5 level. He was followed up in outpatients.

He re-presented in early 1985 with a two year history of faecal incontinence provoked by walking. Initially this only occurred if he walked several miles, but the distance had gradually decreased to 400 yards and he was forced to wear incontinence pads throughout the day. He was fully continent at rest.

His right leg felt stiffer than before, but he had no urinary or other symptoms. Examination revealed decreased muscle bulk in the right thigh as before, but there were no new sensory or motor signs, and anal sphincter tone was normal at rest. The distressing nature of his symptoms precluded provocation testing.

A cerebral CT scan was unremarkable. A 50% reduction in the sagittal diameter of the subarachnoid space at L 4/5, associated with disc degeneration (see fig). Lesser narrowing of the spinal canal due to disc disease was also present at L 3/4. There was no significant abnormality in the dorsal and cervical regions, and the dorsal arachnoid cyst was unchanged. A lumbar decompression was performed, with removal of the spinous processes and laminae of L 4/5 and the upper part of S 1. At operation gross osteoarthropathy and hypertrophy of the ligamentum flavum was noted. The postoperative period was uneventful. One month after decompression the patient was able to walk more quickly than he had for three years. He had not been faecally incontinent for several weeks, and was confident enough to have discarded his incontinence pads. Three months later he had had no further episodes of incontinence, and his only residual symptom was slight stiffness of the right leg.

While exercise provoked symptoms in some legs related to stenosis of the lumbar canal are well described, the association of incontinence with the syndrome is less certain. Sharr, Garfeld and Jenkins4 found radiological evidence of lumbar stenosis and neurogenic symptoms of “neurogenic claudication” in some women with urinary incontinence aggravated by exercise and posture. However, Hawkes and Roberts4 found a 50% incidence of urological symptoms both in men and women with “neurogenic claudication” and equally in a control group with peripheral vascular disease.

Faecal incontinence has not previously been reported in this context, and its occurrence in our patient requires explanation. Maintenance of faecal continence is now thought to depend chiefly on the puborectalis muscle, which acts as a sling round the anal and rectal divisions. Although the internal smooth muscle sphincter makes the largest contribution to intraluminal rectal pressure, its division causes little functional disability.5 Both the puborectalis and the external sphincter muscles are supplied by S 2–4 nerve roots and show tonic activity which ceases during defaecation.6,7 La Fuente, Andrew and Joy8 have shown in a cadaver...
model that lumbar disc prolapse causes less stretch in the sacral roots than in their lumbar counterparts. The lower tension in the sacral roots may cause relative sparing from the exercise induced ischaemic neuritis which Blau and Logue2 thought caused “neurogenic claudication”, and this explains the rarity of sphincter movement in this condition.

Most causes of faecal incontinence are “idiopathic” and are not associated with more generalised neurological disturbance. Electromyography and histological studies suggest that injury to the distal nerve supply, perhaps due to stretch injury during straining at stool or childbirth, causes partial denervation of the muscles of the pelvic floor.9 It may be that in our patient an already compromised sphincter mechanism was rendered incompetent by an exercise induced alteration in sacral outflow.

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