A 65 year old ex-policeman was admitted with a three day history of falling to his right whenever he attempted to sit, stand, or walk. He was normal until a day before the onset of this neurological event when he had complained of a mild generalised throbbing headache. The next morning he noticed difficulty in getting out of bed and needed support to even sit erect. He was unable to stand or walk without support. Whenever he attempted to do so he leaned heavily to his right and fell over. He had never experienced such an event in the past. There was no history of drugs that could produce extrapyramidal syndrome or ataxia. He gave no history of head trauma. He was not a diabetic and was not hypertensive.

On admission his blood pressure was 140/80 mmHg and his heart rate was 90 beats/min. The cardiovascular system was normal. There were no external injuries. He was conscious and well orientated to his surroundings. Speech and memory were normal. His pupils were normal in size and reacted equally well to light. He had a mild drift of the outstretched right arm. Power of the other limbs was normal. Muscle tone was normal in all limbs. The deep tendon reflexes were hyperactive on the left side. The Babinski’s reflex was flexor on both sides.

His tendency to fall to his right was obvious and striking. He was unable to sit, stand, or walk without support. When asked to rise from a recumbent posture he would grapple at the cot railings with his left hand and struggle to do so. When supported he could sit erect for a few seconds but gradually leaned to his right and diagonally backwards. When helped to stand erect, he would fall in the same direction. Supported walking was possible for only a few steps and was terminated by the falling attack. Another striking feature was that he made no postural adjustments to overcome such falls and barely expressed concern about them (figure; left).

He had no evidence of hemianesthesia or visual field cut. Bedside tests for sensory neglect were negative. His right arm was underused in motor tasks. This was out of proportion to the mild weakness of that limb, indicating the presence of motor neglect as well as the pyramidal lesion. He had no features of cerebellar, vestibular, or peripheral nerve disorder.

A clinical diagnosis of “ease of falling” syndrome was made. A plain and contrast enhanced CT of the head surprisingly showed a large subdural haematoma in the left frontoparietal region (figure; right). The haematoma was isodense with the cortex and compressed the ipsilateral subcortical structures and lateral ventricle and produced a shift of the midline structures to the opposite side. There was no evidence of damage to the underlying brain. Chest radiograph, ECG, and carotid Doppler studies were normal. Blood chemistry was normal. Serological tests for syphilis were negative.

A burrhole was made on the left side of the skull and 250 ml of altered blood was evacuated under local anaesthesia. The result was dramatic. The patient could sit erect without support in the immediate postoperative hours. Detailed evaluation was carried out over the next 24 hours and photographically documented. He could sit erect, stand, and walk by himself without any tendency to fall. The outstretched right arm showed no drift. The pyramidal signs and motor neglect disappeared.

The “ease of falling” syndrome has become well characterised through the studies of Masdeu and Gorelick,1 Awerbuch et al,2 and Labadie et al.3 Isolated cases with similar features had already been reported by Fisher and Cole6 in 1965 and by Fisher in 1979 and 1982.23 As noted by all these authors the falls are a contralateral slow tilting motion either laterally or diagonally backwards. The patient shows lack of awareness and does not make postural adjustments to avoid the fall. Criteria require that the patient should exhibit such falling events in the absence of significant hemiparesis, hemianesthesia, cerebellar ataxia, vestibular dysfunction, proprioceptive loss, and peripheral nerve disorder. Our patient qualifies for the diagnosis of this syndrome.

All previously reported cases had an intracerebral lesion affecting either the putamen, pallidum, or the thalamus. Lacunar infarcts and haemorrhages are the only lesions that have produced this acute condition.
painless spinal cord compression after subarachnoid haemorrhage. This has not to our knowledge been previously described.

A 70 year old woman was admitted to the Wessex Neurological Centre in September 1991 with difficulty walking and poor balance. Ten months earlier the patient had collapsed at home with severe headache and a diagnosis of subarachnoid haemorrhage was confirmed on lumbar puncture, which showed uniformly heavy blood staining. A cranial CT showed no evidence of haemorrhage and bilateral carotid angiography was normal. The patient had no abnormal neurological signs and recovered with conservative measures. After discharge she remained well for three weeks and then experienced sudden weakness in her left leg for which she did not seek any medical attention. This resolved after three days and was not associated with any sensory disturbance.

In June 1991 the patient developed a progressive weakness of her right leg followed by increasing left leg weakness. She noticed an ascending numbness involving both legs and by the time of her admission this had reached her umbilicus. She had no sphincteric disturbance or back pain.

On admission the patient was ataxic and was unable to walk unaided. Clinical examination showed a spastic paraparesis with grade 3 power, the weakness being slightly more pronounced on the right. Both knee jerks were brisk and the planters extended; the ankle jerks were absent. There was a sensory level at T8; light touch pinprick and temperature were all reduced below this level and joint position sense was absent in both legs. The gait was ataxic and Romberg's test was positive.

Routine blood tests and plain spinal radiographs were reported as normal. MRI of the dorsal spine showed a multiloculated intradural cystic lesion from T4 to T8 having the same signal intensity as CSF on the T1 and T2 weighted images with evidence of spinal cord compression (figure A, B).

Two days after admission a thoracic laminectomy from T4 to T8 was performed. There was an extensive cyst lying mainly on the right side. Using a Valsalva manoeuvre the cyst was shown to gradually fill over a period of time. The cyst was removed throughout the length of the exposure although a portion of the cyst below T8 was left.

Pathological examination of the specimen showed a meningeal cyst with a thickened fibrocollagenous outer wall containing areas of calcification with no inner arachnoid membrane. There was no evidence of previous haemorrhage. Postoperatively there was an initial deterioration of the patient's paraesthesia that has since improved although the patient has remained ataxic due to poor joint position sense.

Previous classification of meningeal cysts has been histologically misleading with intradural cysts being designated arachnoid cysts or arachnoid diverticula. A recent classification by Nabours et al, however, suggested that diagnosis and classification should be based on radiological, histological, and operative findings. They suggested dividing meningeal cysts into extradural and intradural and recommended MRI as a preoperative assessment to define accurately the anatomy and tissue structure of the cyst.

Further reports with MRI of sacral meningeal cysts have shown flow sensitive sequences useful in differentiating communicating from non-communicating cysts.4 We consider that it is better to regard a communicating cyst as in our case as a pouch rather than a true cyst.

Intradural cysts have been described more commonly than intradural cysts and whereas intradural cysts are usually asymptomatic,1 presentation with back pain is much more common than cord compression. This is by contrast with our case where the initial symptomatology of was of sudden but transient left leg weakness possibly due to filling and then emptying of the cyst or as a result of a vascular event. This was followed later by painless progressive spinal cord compression.

We believe that it is likely that the patient's initial symptoms were related to a change in the CSF dynamics after subarachnoid haemorrhage. This caused a rise in the overall CSF pressure and allowed its

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1 Fisher CM, Cole M. Holomodal ataxia and crural paresis; a vascular syndrome. 7 Neurou 1965;28:48-55.

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Spinal cord compression secondary to a thoracic meningeal cyst after subarachnoid haemorrhage: a case report

Spinal meningeal cysts are rare and their aetiology uncertain, although an association with congenital abnormalities and familial tendency favours a congenital origin.1 Spinal meningeal cysts are very rare, although intermittent symptoms of back pain and fluctuating spinal cord compression can occur and may be explained by emptying and filling of the cyst.1 We report the case of a thoracic intradural cyst causing