abilities quickly returned to their previous level, he continued to need cuing to initiate his self care. The patient was discharged home under 24 hour supervision of relatives.

The neuropsychological testing completed after the operation revealed average intellectual skills but slow performance on the timed visual perceptual tasks. Although his intellectual performance was significantly better than the initial testing, intelligence level was slightly below that expected given his educational history and age. The patient's slow processing on the timed visual perceptual tasks probably resulted in a decline in his intelligence score. On a problem solving task his performance was mildly perseverative and, most likely because of his memory disturbance, the patient had difficulty maintaining a strategy over time.

The neuropsychological testing also revealed a long-term memory dysfunction (figure). Although immediate recall of verbal material was more accurate than before the operation, the patient's ability to recall material after a significant delay was severely impaired. When a list of seven words was presented verbally, he was able to repeat six (immediate or working memory). When asked to recall the words after a delay, no words were recalled spontaneously, semantic clues were not helpful, and recognition memory was also found to be severely impaired.

A brief assessment took place two months after the operation. Again, the patient was not able to provide the date, his own age, or recall recent significant events. Formal testing of verbal memory again revealed a severely impaired long-term memory.

Our patient has many symptoms consistent with a diagnosis of an amnestic syndrome. He was unable to acquire new visual or verbal information efficiently and was only able to learn procedures with much repetition and numerous cues. Memory difficulties influenced his ability to perform efficiently on a problem solving task. Neuropsychological testing revealed particularly severe deficits in encoding new information and retrieval after a delay or distraction.

Testing completed before operation showed adequate long-term memory functioning, so it is unlikely that alcohol abuse itself caused pre-existing deficits. The patient's drinking history, however, may have compromised diencephalic structures so that no alternative limbic memory pathways were available after surgical intervention on fornices compressed by the tumour. This would explain the apparent contradiction between our results and Apuzzo's suggestion that the fornix is not required for normal memory functioning.[1]

This is the second published case of surgery in which a transcallosal approach was associated with the development of a non-transient amnestic syndrome as Berti *et al* also presented a patient who demonstrated amnesia after similar surgery.[2] The results from our patient indicate possible interactions between alcohol abuse and outcome from particular neurosurgical approaches.

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Isolated aneurysm of a spinal radicular artery presenting as spinal subarachnoid haemorrhage

Isolated aneurysms within the vertebral canal are rare, with 11 patients reported in the literature.[1] We present a patient with symptoms and signs of spinal subarachnoid haemorrhage in whom MRI was used to localise the intradural haematoma to restrict selective spinal angiography and in whom an isolated intradural aneurysm of a spinal radicular artery was clipped successfully.

A 59 year old white woman was admitted with acute backache. She was well until the day before admission when she experienced the sudden onset of stabbing frontal headaches that subsided after five minutes and were followed by low back pain radiating up to the midthoracic level. The pain was worse with body movements. On admission she had mild meningism and an incomplete level at T7-T8 with loss of pinprick sensation spanning about three sensible dermatomes. One week later, a girdle-like pain appeared that originated in the back at the thoracolumbar level and radiated into the right upper abdomen. The girdle-like pain and the diffuse backache resolved over the next two weeks together with the meningism and signs of incomplete transection.

CT revealed intracranial subarachnoid haemorrhage but four-vessel angiography failed to show any intracranial bleeding source. A lumbar puncture confirmed the diagnosis of spinal subarachnoid haemorrhage. Spinal MRI showed a subdural haematoma at the lower thoracic levels (figs 1 and 2). Selective spinal angiography at these levels depicted a saccular aneurysm originating from the right radicular artery at T8 (fig 3).

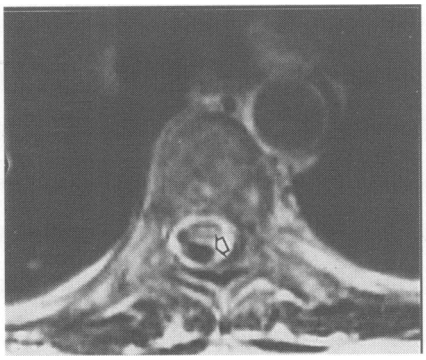

Figure 1 T1 weighted horizontal section at lower thoracic levels showing subdural accumulation of blood (arrow).

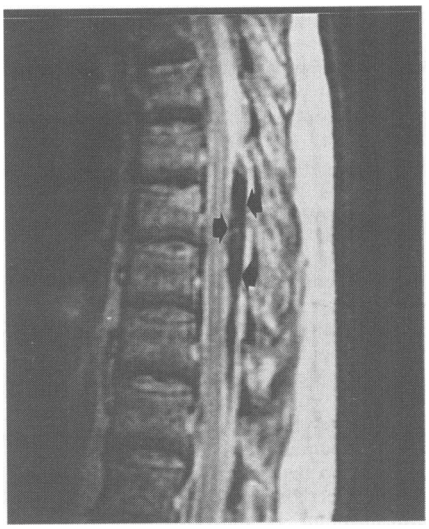

Figure 2 T1 weighted sagittal section through the spine. Note subdural haematoma (arrows).

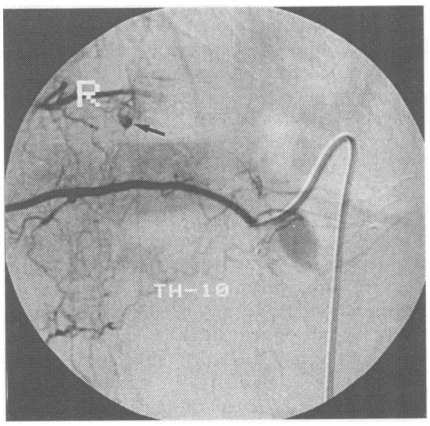

Figure 3 Angiographic demonstration of the aneurysm (arrow).

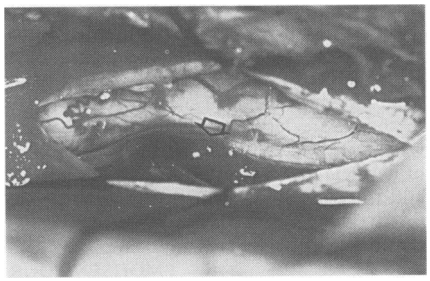

Figure 4 Intraoperative situation before evacuation of haematoma and clipping of the aneurysm (arrow).

On day 32 after spinal subarachnoid haemorrhage (13 April 1992) hemilaminectomy was performed at T8 and T9. After opening the dura, the dorsal radicular fila of nerve root T8 and the denticulate ligament appeared displaced by a saccular aneurysm of the related radicular artery measuring 5 mm in diameter (fig 4). After removal of the surrounding blood clot the spinal cord at the aneurysm site showed a yellowish discolouration. The aneurysmal sac was clipped and resected. Histological examination confirmed the diagnosis of a true saccular aneurysm with a thin fibrotic wall and organised blood adhering to the inner surface.

The patient made an uneventful recovery with complete resolution of neurological

symptoms and signs (after 13 months).

Most spinal aneurysms occur in the presence of a spinal arteriovenous malformation. Isolated aneurysms of spinal arteries visualised during the initial arterial phase of angiography with direct connection to the artery are extremely rare. Most of the 12 patients reported were adults, with women affected more often. All reported isolated aneurysms of spinal arteries occurred at the cervical or thoracic level, mostly arising from the anterior spinal artery; a spinal radicular artery was less often involved.

Spinal aneurysms may present as spinal subarachnoid haemorrhage or cord compression. Ten of the 12 patients had aneurysm rupture and eight of the 12 patients developed symptoms and signs of spinal subarachnoid haemorrhage. The differentiation between subarachnoid haemorrhage of cerebral and of spinal origin is a crucial point in diagnosis. According to Prieto and Cantu,[2] the following features suggest a spinal rather than an intracranial origin: radicular or lumbar pain more severe than headache, rapid disappearance of headache and cerebral symptoms and persistence or aggravation of spinal symptoms, a normal level of consciousness, and the intensity of the radicular pain. Some of these features were present in our patient and prompted MRI of the spine and selective spinal angiography after negative cerebral panangiography. The other major presenting feature of patients with isolated aneurysm of the spinal arteries are cord compression syndromes. This less frequent presentation mode has been reported four times. In our patient the signs of incomplete transection may be attributable to the space occupying intradural extramedullary haematoma at lower thoracic cord levels rather than to the aneurysm itself.

Surgical exclusion of the aneurysm sac is the treatment of choice. Two out of three patients reported in whom surgery was not feasible, died.[3 4] Our patient underwent surgery after the decline of all major clinical symptoms and signs. We do not know to what extent vasospasm or rebleeding occur in these rare lesions. Although the history was negative, the yellowish discolouration of nervous tissue surrounding the aneurysm and the episode of girdle-like pain after the first week of illness may indicate that recurrent bleeding episodes had taken place.

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MATTERS ARISING

Delirium and quantitative EEG

We appreciate the comments about our paper[1] from Dr Primavera and colleagues at the University of Genoa.[2] We are familiar with the confusion assessment method developed by Inoyue *et al*,[3] and agree that it represents an improvement over DSM-III-R criteria in the hands of non-psychiatrist evaluators.

In our laboratory, quantitative EEG (QEEG) studies are always done in conjunction with conventional EEG studies for the very reason that Primavera *et al* note: some electrophysiological abnormalities that may be associated with certain aetiologies of delirium periodic lateralised epileptiform discharges, triphasic waves, spikes and sharp waves) may be detected only on conventional EEG. We would like to raise the point, however, that once the initial EEG/QEEG study of a delirious patient has been analysed, it might be possible (and less costly) to monitor the course of treatment/resolution with follow-up "quick looks" by QEEG alone. We would emphasise that a careful study of this application would be needed before this practice could be recommended.

We have also performed a study of serial EEG/QEEG in delirium,[4] and found similarly that electrophysiological measures remained abnormal after clinical delirium by DSM-II-R criteria had resolved. We recognise that EEG/QEEG measures are the more sensitive index, and have found that mini-mental state examination is in the same range of sensitivity. We would be interested to hear of any other objective measures of delirium that are as sensitive, particularly those that assess aspects of delirium other than cognitive.

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Management of subarachnoid haemorrhage

The very long review on the management of subarachnoid haemorrhage by Kopitnik and Samson[1] is full of statements that are at least controversial. For a discussion of all of these, we would need more space than is available here. We therefore list the controversial statements in italics with our refutation and reference to the appropriate literature beneath it. Finally, we fully agree with the last sentence of the review by Kopitnik and Samson which says that physicians who diagnose and manage patients with subarachnoid haemorrhage would be well advised to keep up with the ever changing developments in the management of this ubiquitous and catastrophic condition.

"At least one third of patients with aneurysmal subarachnoid haemorrhage will have a minor leak."

All studies on these so-called warning leaks are retrospective and hospital based.

"Brief loss of consciousness occurs in most patients [with subarachnoid haemorrhage] . . ."

Half the patients with subarachnoid haemorrhage do not lose consciousness at the ictus. In the other half, the loss of consciousness can last a few seconds but may also be never-ending.[2]

"The Fisher grading system is used to relate the amount of subarachnoid blood on a CT scan to the probability of developing delayed [cerebral] ischemia . . ."

This method is difficult to apply outside the centre of origin.[3]

"Visual examination of CSF obtained by lumbar puncture can confirm the diagnosis of [subarachnoid haemorrhage] . . ."

Visual examination of CSF is an unreliable method to confirm subarachnoid haemorrhage.[4]

"Xanthochromia . . . is usually undetected at three weeks."

At three weeks the probability of detecting xanthochromia is over 70%.[4]

"If a traumatic lumbar puncture is suspected, partial . . . clearing of the CSF may occur . . ."

This partial clearing may also occur in patients with subarachnoid haemorrhage.[5]

"One of the more universally accepted grading scales . . . is that of Hunt and Hess . . ."

Problems in applying grading systems such as these were shown by two studies.[6 7]

". . . the angiogram is repeated [in vasospasm] if a portion of the cerebral vasculature is not adequately visualised on the initial study, or in patients who have a large amount of subarachnoid blood visualised on CT scanning."

The pattern of haemorrhage on CT is a crucial factor in assessing the need for follow up angiograms in patients with an initially negative study.[8]

". . . 80% of patients with [subarachnoid haemorrhage] of undetermined aetiology will have a good outcome . . ."

All patients with perimesencephalic haemorrhage have a good outcome, whereas 25% of patients with an aneurysmal pattern of haemorrhage on CT and a negative angiogram die or are left disabled.[8]

"We have frequently observed that the interpeduncular or perimesencephalic cisterns often demonstrate focal blood collection when [subarachnoid haemorrhage] of unknown aetiology occurs."