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."