lance, psychomotor speed, intelligence) with sparing of memory (table). An EEG disclosed diffuse slow activity. Cerebral MRI showed circumscribed bilateral lucencies on the lentiform nuclei on T1 weighted images. On 10 July liver transplantation was performed, with a successful course and a rapid improvement of the neurological disturbances. Immunosuppressive treatment with cyclosporine did not induce neurological complications. One month after liver transplantation only a mild dysarthria persisted. An EEG was normal. A neuropsychological assessment 3 months after surgery showed a remarkable improvement in the cognitive performances, especially in information processing control tasks (table), whereas cerebral MRI was unchanged. Twelve months later, neurological examination was normal and cerebral MRI disclosed a reduction of basal ganglia lucencies. Neuropsychological testing documented a slight further improvement in control functions of information processing, with a slight decline in some memory performances (table). No other neurological problems emerged during subsequent months.

This patient had an AHD presenting with movement and cognitive disorders. The first consisted in disabling movement disorders, with severe bradykinesia and dysarthria. The cognitive impairment included both a decreased functioning of the frontal executive functions and single function deficits (expecially visuospatial abilities and language), conveying a picture of "hepatic dementia". Cerebral MRI documented the basal ganglia lesions usually seen in AHD.[2] Both the clinical and the neuroradiological abnormalities were reversed by liver transplantation. After surgery, the recovery from neurological impairment was prompt and complete, whereas neuroimaging improvement occurred later. This outcome resembles that previously seen in a patient with Wilson's disease.[3] Despite the different pathogenesis, the similarities between AHD and Wilson's disease are remarkable for pathological lesions and clinical and neuroradiological presentation.[1 2] Liver transplantation has been reported to reverse neurological manifestations in most patients with Wilson's disease.[3] Liver transplantation in AHD is confined to two cases. A cirrhotic patient with improved chronic cognitive and motor disorders after liver transplantation was described in 1970.[4] Twenty years later, Powell *et al*[5] reported a case of successful liver transplantation in AHD. Their patient had a significant improvement in intellectual functions and chronic neurological signs early after surgery. Our present finding confirms these positive results and also documents that neuroradiological abnormalities are reversible. It is conceivable that both Wilson's disease and AHD are characterised by an early stage neuropathological process mainly affecting the basal ganglia, where MRI detectable hepatocerebral degeneration is slowly reversible and liver transplantation can rapidly improve neurological symptoms. The duration of the disease does not seem to be a crucial factor, as patients with long standing encephalopathy may also recover after liver transplantation both in AHD[5] and in Wilson's disease.[3] This conclusion has pathogenetic and therapeutic implications: the presence of signs and symptoms of chronic hepatocerebral degeneration, both in Wilson's disease and in the acquired non-Wilsonian form, should not be considered a contraindication for liver transplantation and surgery may be the elective treatment for the neurological syndrome.

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CORRESPONDENCE

Unexpected sudden death after lateral medullary infarction

I read with interest the study of Fitzek *et al*,[1] which included 15 patients with lower brain stem infarction. One patient with a "complete Wallenberg's syndrome" (No 15) died during the period of observation. Details on that patient's death are not included in the paper.

Through personal communication with the authors I have learned that their patient No 15, a 69 year old man, died unexpectedly 14 days after an acute brain stem infarction. Because the family refused a necropsy, we do not know with certainty whether some other acute process was involved in the patient's death. However, an ECG and chest radiograph after presentation had been normal.

Recent reports[2–5] have described patients who experienced unexpected sudden cardiorespiratory arrest several days after lateral medullary infarction, at a time when they were convalescing well and were stable medically and neurologically after a stroke which caused minimal motor disability. The reports have speculated about mechanisms by which cardiorespiratory arrest occurred; cardiac arrhythmia is among these.[4]

Although I do not know many pertinent details surrounding the death of the 69 year old man described by Fitzek *et al*,[1] I speculate that his death may have resulted from cardiopulmonary arrest caused by an intermediate event in which the lateral medullary infarction and surrounding brain tissue disturbance (possibly ischaemic penumbra) influenced brain stem cardiac and respiratory centres together with autonomic pathways in a manner which at this time is not understood.

A recent neuropathological study[6] of five patients disclosed similar characteristic ischaemic lesions in the solitary tract nuclei of the medulla after subacute hypoperfusion of the brain during acute heart failure. It was speculated that these medullary lesions had in turn caused autonomic instability which precipitated death in each case. It is plausible that ischaemic lesions of the solitary tract nuclei result initially with some lateral medullary infarctions, and that such lesions may in turn precipitate some occurrences of cardiorespiratory arrest.

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Postictal psychosis related regional cerebral hyperfusion

I wish to comment on the postictal psychosis related regional cerebral hyperperfusion reported by Fong *et al*.[1] Based on the their findings of hyperperfusion on SPECT within the time frame of postictal psychosis, the authors argue against the hypothesis that postictal psychosis is a psychic manifestation of a Todd's phenomenon. Two previous studies have shown a focal increase in cerebral blood flow on brain imaging during traditional motor Todd's paresis.[2 3] An angiogram during a Todd's paresis may demonstrate a vascular "blush" perhaps representing loss of cerebrovascular autoregulation at the site of the epileptic focus.[2] Hence, hyperperfusion may signal hypofunction, and the findings of Fong *et al* are indeed consistent with postictal psychosis as a Todd's equivalent.

The strongest argument that postictal psychosis is not a Todd's equivalent is the delayed onset of psychosis compared with the decrescendo course of Todd's motor, cognitive, and visual phenomena.[4 5]

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Fong replies:
We thank Boylan very much for the interesting letter provoking a second thought on the pathogenesis of postictal psychosis. Our data showed a definite increase in regional cerebral blood flow (rCBF) in both patients with postictal psychosis. As pointed out by Boylan, postictal psychosis may or may not be secondary to Todd's paralysis. In fact, the clinical features of postictal psychosis point against the hypothesis of Todd's phenomenon being the underlying pathophysiology.

We think that the underlying mechanism of postictal psychosis is due to activation of a subcortical circuit. In our patients, the antiepileptic agents were restarted after a bout of secondary generalised tonic-clonic seizures. The re-institution of anticonvulsant drugs may cause a preferential suppression of abnormal cerebral cortical activities and hence normalise the surface EEG recording. In turn, it may result in a gradual build up of abnormal electrical activities propagating via subcortical neuronal networks which is shown by cerebral SPECT studies as areas of enhanced rCBF. This can explain the characteristic lucid interval of postictal psychosis[1] and the activation of subcortical circuits may cause clinical psychosis.[2]

To understand the pathophysiology of postictal psychosis, we wish to study the electrical activities of patients with postictal psychosis by intracranial electrodes and regional cerebral metabolism by cerebral PET.

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HTLV-I and HIV infections of the CNS in tropical areas

I read with interest the recent article by Cabre *et al*.[1] I make three comments on the section of the review pertaining to HIV disease. I disagree with the statement made on page 551 that chorea is pathognomonic of toxoplasmosis encephalitis in patients with AIDS. Chorea may also occur in patients with AIDS dementia complex (ADC).[2] Secondly, there are several errors in table 2. Fluconazole is not given as 400 mg four times a day for acute cryptococcal meningitis therapy but rather as 400 mg/day; fluconazole is not given as 200 mg four times a day for suppressive therapy but rather as 200 mg/day; pyrimethamine is not given at 50–100 mg four times a day for acute toxoplasmosis therapy nor is folinic acid at 10 mg four times a day or sulfadiazine 4–8 g four times a day but rather pyrimethamine 50–100 mg/day, folinic acid 10 mg/day, and sulfadiazine 4–8 g/day; pyrimethamine for suppressive therapy is not given at 25–75 mg four times a day but rather as 25–75 mg/day and folinic acid should be given at a dose of 10 mg/day; the toxoplasmosis prophylactic dose of trimethoprim 160 mg with sulfamethoxazole is one tablet per day. Finally, the statement on page 552 "antiretroviral therapy can only improve ADC symptoms" is no longer correct. Significant improvement in ADC symptoms, signs, and function (to the point where some patients can return to full-time work) is now possible with highly active antiretroviral therapy.[2]

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Statistics and analysis of the Camino ICP monitor

We have concerns about the mathematics, accuracy of the data, and conclusions in the paper by Martinez-Manas *et al*.[1] This paper reports on a prospective study of the accuracy and complications of the Camino intracranial pressure monitor.

The authors have been lax in their use of English, failing to differentiate between their use of the words "patients" and "probes". This would not be such a problem if they had only reported on one probe per patient, which should have been part of the protocol of the study. They have also used the verbs "to calibrate" and "to zero" interchangeably when in fact they mean "to zero"; thus the devices need to be "zeroed" before insertion not "calibrated".

The paper reports on 108 probes in 101 patients. Details of patients should relate to 101 individuals therefore; for instance, there could not be 65 males and 43 females. There are numerous mistakes throughout the paper in the basic calculation of percentages. For instance 66 cases of head injury (fig 1) out of the 108 indications for monitoring do not account for 71% of implantations and three positive cultures from 16 subdural devices do not account for 10.7%. Furthermore, the precision suggested by the use of decimal places in reporting percentage data is totally unwarranted.

There is also concern about the failure rate of probes from the authors' analysis of infection rates and zero drift, which was performed on only 63%, and 52% of the total number of inserted probes respectively. The protocol should have included procedures to minimise this. It may be that there was a high failure rate of the catheters but this is not reported. More details should be given to ascertain whether any bias is likely to have been introduced by excluding so many probes.

Figure 3 suggests huge drifts (-24 mm Hg to +35 mm Hg) that are clinically significant and unacceptable, with 39% of probes tested failing to comply with the manufacturer's specifications. The authors demonstrated that there was no correlation between duration of monitoring and zero drift which is in agreement with previous work.[2] However, the authors fail to highlight the fact that regardless of the duration of monitoring, 23% of probes tested had a zero drift of $\geq \pm 10$ mm Hg, which is clearly unacceptable.

The representation of the data as mean, median, and SD in table 3 is misleading as it is clear from fig 3 that there is a wide distribution of both positive and negative offsets. Consequently, a near zero mean drift is likely to occur even though the magnitude of the zero drift in individual cases is large. Clinically, it is the zero drift from a single patient that is important and not the zero drift of a series of probes.

The recommendation to change the catheter if a long monitoring period is expected to allow for rezeroing is not held up by the data shown in fig 3, which would suggest that there is more likely to be a larger zero drift than the manufacturer's specification in the early days.

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BOOK REVIEWS

Abnormal Cortical Development and Epilepsy: from Basic to Clinical Science. By R SPREAFICO, G AVANZINI, and F ANDERMANN. (Pp 324, £39.00). Eastleigh: John Libbey,1999. ISBN 0 86916 579 5.

One of the most interesting fields of research in epilepsy in the past 5 years or so has concerned cortical dysgeneses. In some series of chronic epilepsy, overt dysgenesis underlies 15% of all epilepsies, and more subtle forms might account for some apparently cryptogenic cases. This book is therefore timely. It is a record of the proceedings of a conference held in Venice in October 1997, within the framework of the Mariani Foundation Colloquia in Childhood Epilepsy. The book is organised into sections on cortical development, animal models, electroclinical imaging and neuropathological studies, genetics, and surgical treatment. The faculty and chapter authors are distinguished figures in this research field largely from the United States, Canada, and Italy.

The recognition of the importance of these conditions in epilepsy has been due to the introduction of structural MRI and also the advances in understanding of the processes of cortical development. The second field particularly is one in which advances are being made rapidly, both clinically and in the laboratory, and the authors and editors do a superb job in marshalling this information into a readable and well organised form. I found many of the chapters exceptionally interesting. The heavy emphasis on molecular genetics and pathology is appropriate in this area and is a model for how the modem topics of epilepsy should be approached.

My only reservation about the book is that in this fast moving field some of the basic science and genetic data are already out of date,