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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