In humans, areas in the left inferior frontal and right superior parietal cortex become active both when producing and when seeing finger movements in others.¹ Could similar mirror activity arise in a purely sensory context, such that a person, due maybe to inhibitory failure, may experience pain in a finger or limb when seeing sudden trauma (for example, a blow) to a corresponding area in another person? We report the anecdotal account, from a widow, of her late husband's apparent experience of such "mirror pain" or, as we would suggest, "allodynia".

The deceased, a long time smoker, died in late February 1993 with the diagnosis of "extensive metastatic carcinomatosis", antecedent cause, "carcinoma of the right lung". (The widow, however, questions the lung cancer diagnosis, and claims that symptoms of serious rheumatoid disease involving cervical spine and dysphagia were misinterpreted.) As a consequence of increasing pain and stiffness beginning in the neck and upper body, and chest symptoms, he underwent radiography of the cervical spine and chest in August 1990, disclosing opacity in the right lung and slight tracheal deviation; he had increasing difficulty swallowing with food inhalation. Unwillingly, he underwent radiotherapy in early November 1990 to alleviate dysphagia, although according to the widow subsequent gastroscopy indicated that this may have been unnecessary.

He was reported to be very sensitive to touch; even the slightest hand contact gave the impression of sharp fingernails. Of particular interest was his widow's recent observation that "If I slightly knocked my finger, spontaneously showing him, he would immediately grasp his own finger and say "don't do that" (meaning not to show him); He actually felt it. If I merely commented (that I had knocked my finger), there was no such reaction". In interview, she recounted other similar events. The experience was suddenly immediate and intense, and, apparently, qualitatively similar to the hypersensitivity occasioned by actual contact. She had initially contacted one of us (JLB) after hearing a radio broadcast by him of phantom limb phenomena, and wondered whether an analogous mechanism of some kind may have been operating with her late husband.

Although mirror motor neurons may be fundamentally important in learning to act, an adaptive role is far less obvious for perceiving another's pain. Perhaps during infancy avoidance of noxious stimuli is facilitated by early recognition of pain in others. Alternatively the phenomenon may merely be adventitious consequence of disruption of convergent sensory systems. Thus hyperalgesia, where a light touch induces an unpleasant sensation in the same person, is typically attributed to dysfunction of convergent sensory neurons in the neuraxis, though any of several CNS levels may be involved. However where, as here, a separate person is implicated, there may be additional limbic involvement, given the rather intensely emotional aversive aspects of the sensory experience.

Unfortunately no CT or MRI seem to have been performed of the brain, but it is probable that there was fairly widespread CNS involvement. He had also, apparently, experienced head trauma in the war. It would be of interest to know whether similar "allodynia" has been seen after known damage that includes left inferior cortex (opercular region), or the rostralmost region of the right superior parietal lobule.⁵ It would

also be interesting to get persons, normal or hyperalgesic, to note reactions to noxious stimuli in others while judging the intensity of mildly aversive tactual stimuli they receive themselves.

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Acquired hepatocerebral degeneration: full recovery after liver transplantation

Liver diseases may give rise to variable degrees of neurological impairment, which mostly consist of the syndrome of hepatic encephalopathy, due to the toxic effect of ammonia on the brain during episodes of liver decompensation. In a minority of patients, repeated episodes of liver failure can lead to a chronic progressive encephalopathy, not necessarily related to hyperammonaemia, known as acquired hepatocerebral degeneration (AHD).¹ The pathogenesis of AHD is unclear, but the relation with the acute form of hepatic encephalopathy seems a crucial point. Cerebral deposition of manganese may have a pathogenetic role. The disease may

appear after one or more episodes of hepatic coma or, rarely, become manifest in the absence of them. Neuropathology typically discloses degenerative changes in the basal ganglia. The modern techniques of neuroimaging disclose these lesions *in vivo*.² The clinical picture varies, neuropsychiatric changes and movement disorders usually being prominent. The syndrome is poorly responsive to medical therapy, thus being considered largely irreversible.

We report on a patient with AHD who was cured by liver transplantation.

A 59 year old man came to us in November 1997 for a neurological consultation before inclusion in the waiting list for liver transplantation. He had a history of chronic hepatic disease—alcohol and HCV related liver cirrhosis—which had begun some years before. No familial hepatic or neurological diseases were reported. In 1995 he had an episode of hepatic encephalopathy, consisting in somnolence and confusion lasting 36 hours. At the time of examination, the patient had stopped alcohol consumption 1 year before; liver failure was grade C-10 of the Child-Pugh classification. Copper balance was normal. Neurological examinations and EEG gave normal results. The patient was put on the waiting list for liver transplantation. In February and March 1998, he had two episodes of mild ascites with signs of encephalopathy (confusion and asterixis), both reversed by medical therapy. In April 1998 the patient began to complain of sleep disorders, tremor, dysarthria, motor slowness, and subtle cognitive dysfunction, not reversed by medical therapy for hepatic dysfunction. On 6 June 1998, his neurological suitability for liver transplantation was reconsidered. He seemed alert, oriented, and cooperative, with a slight slowness of psychomotor activity. The neurological examination showed hypomimia, dysarthria, bradykinesia, oral dyskinesia, and mild bilateral hand tremor. Neuropsychological examination showed a remarkable impairment of information processing control (attention, vigi-

Neuropsychological testing before and after liver transplantation (LT)

Test	Cut off	Score* before LT	Score* after LT (3 months)	Score* After LT (12 months)
Information processing control:				
Attentional matrices (visual search)	31	9	40.2	47.2
Trail making form A: time	93	84	66	27
Trail making form B: time	282	275	168	81
Stroop: word				
Time	38	76	19	15
Errors	2	0	0	0
Colour				
Time	35	58	29	31
Errors	1	4	1	0
Colour/word				
Time	80	124	130	85
Errors	9	10	13	22
Letter A cancellation: errors	9	24	15	5
Digit symbol substitution	19	12	19	18
Auditory reaction times (ms)		281	220	197
Visual reaction times (ms)		423	340	339
Word fluency (F, A, S)	17.3	17.3	26.3	29.3
Raven's coloured matrices (1947)	18.9	19.3	20.3	22.3
Memory:				
Digit span	3.7	4.5	5.5	5.5
Corsi's blocks	3.7	3.7	3.7	3.7
Immediate visual memory	13.8	12.4	16.4	13.4
Rey's 15 words:				
Short term	28.5	37.4	49.4	45.4
Long term	4.7	11.2	12.2	9.2
Paired associate learning	6.5	10	11.5	11
Story recall	4.7	10.6	12.1	4.5
Supraspan spatial learning	5.7	4	14.6	8.7

*Corrected for age and schooling when needed.

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 (especially 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 AHD⁵ 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 trans-

plantation 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 reports²⁻⁵ 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 cardiorespiratory 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 study⁶ 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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