performance after vestibular stimulation ($x = 4.8$, df = 4, $p = 0.009$). On repetition of the imagery task the day after vestibular stimulation performance was comparable to that at baseline.

One interpretation of the behavioural effect of vestibular stimulation on unilateral neglect is that it produces a non specific activation of the right hemisphere and decreases the imbalance caused by unilateral damage. We found, however, that more "left side" landmarks were registered after vestibular stimulation in all our patients in the visual field, although the number of the "right side" landmarks did not decrease. This does not support the hypothesis that unilateral neglect is due to an imbalance in an opponent system that controls lateral orientation.

Our results suggest that the effects of vestibular stimulation on unilateral neglect are only partly caused by facilitation of visual exploration of the relevant sector of space as a result of displacement of gaze or, more generally, by change in the relationship between outside stimuli and body coordinates: the results of the mental representation test suggest that the neuronal circuits underlying endogenous representation of egocentric space are modulated by vestibular projections.

The effects of vestibular stimulation on anosognosia support this supposition. One patient (case 3) had severe anosognosia at baseline evaluation: he denied his hemiplegia even after being asked to move the affected limbs: he grasped and raised his plegic upper left arm with the right hand. Under vestibular stimulation he acknowledged his motor deficit after being questioned specifically, but after about 30 minutes, when the effect of vestibular stimulation had disappeared, the anosognosia worsened with respect to the baseline: on being asked to move his left upper limb he only raised the right one. In this patient high level cognitive processes, for example memory or reasoning, were not able to reverse the anosognosia after transitory amelioration following vestibular stimulation. This behaviour suggests that awareness of self is strongly linked to high level cognitive monitoring processes, but arises from neuronal integration of information about the present state of the body. The effects of vestibular stimulation on both visuospatial representation and anosognosia are consistent with the hypothesis that vestibular inputs exert a basic influence on the representation of centred-space and the body.

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**Ceftazidine encephalopathy: absence status and toxic hallucinations**

We describe a case of absence status and non-epileptic hallucinations due to ceftazidine toxicity. Ceftazidine, a third generation cephalosporin, has structural similarities to penicillin.1 The electro-clinical pattern in the patient closely resembled the generalised epilepsy model induced in cats by intramuscular penicillin.

A 34 year old man had chronic renal failure due to membranous glomerulonephritis. He was being treated with haemodialysis and was admitted for a parathyroidectomy. The operation was uneventful, and serum calcium was maintained postoperatively with supplements of calcium. On the second post-operative day he developed a left lower lobe pneumonia. Intravenous cefotaxime (1 g twice hourly) and penicillin (1 million units six hourly) were administered. On the third post-operative day *Pseudomonas aeruginosa* was isolated from the sputum and cefotaxime was changed to intravenous ceftazidine (2 g twice hourly). On the fifth post-operative day he was confused, incoherent, and had generalised myoclonic jerks as well as frequent eyelid fluttering. He reported vivid hallucinations at this time. On the sixth post-operative day uncontrolled seizures were suspected and a further dose of ceftazidine was given. He then had grand mal tonic-clonic seizures. Serum calcium, magnesium, aluminium and glucose were normal. Blood cultures were negative.

An EEG showed continuous generalised three per second spike-and-wave activity that was abolished with 3 mg of intravenous clonazepam (figure). His confusional state immediately resolved and he was able to draw normal auditory hallucinations. They comprised vivid details of a man sitting in the room operating a complicated machine, the operation of which required the patient’s utmost concentration. Other hallucinations were of brightly coloured balloons trying to come through the cracks in the wall, and of lights attached to a silver chain which broke when he averted his eyes. He also heard familiar voices outside his room, and persistent knocking sounds. Ceftazidine and penicillin were ceased and tobramycin started. Hallucinations persisted for the subsequent two days during which diazepam was performed daily and his EEG did not show recurrence of the absence status. By the ninth post-operative day his mental state was normal. A cerebral CT scan was normal.

Serum ceftazidine levels were assessed using an anti-bacterial assay (*Bacillus subtilis*). Measurements on the fourth and fifth post-operative days yielded levels of 402, and 253 mg/ml (normal peak level 55 mg/ml). The presence of penicillin would have interfered with the accuracy of the assay, but the levels of ceftazidine were still considered toxic. On day eight, after three days of diazepam, the serum level was 34.4 mg/ml.

The electro-clinical pattern in this patient, with impaired conscious state, fine myoclonic jerking, and generalised spike-and-wave discharges on the EEG was diagnostic of absence status.2,3 A similar clinical picture was described previously in a patient with renal failure given ceftazidine, but an EEG was not done and absence status was not diagnosed.4 The immediate reversal of the clinical and EEG abnormalities by intravenous clonazepam demonstrates that the drug caused a true epileptic encephalopathy. This must be distinguished from the usual pattern of toxic or metabolic encephalopathy where there is diffuse slow activity on the EEG, with little or no epileptiform activity, and the only treatment is withdrawal of the offending agent.

The electro-clinical features of this case were similar to those of the feline generalised penicillin epilepsy model, where there is strong evidence for the primary abnormality residing in the cerebral cortex.5 In humans, massive doses of systemic penicillin are required to cause convulsions, even in the presence of renal failure. It is unlikely that penicillin contributed significantly to the neurotoxicity, in the presence of an intact blood-brain barrier, with the doses used. Given the structural similarities of penicillin and ceftazidine, we now suggest that the epileptic encephalopathy induced by ceftazi-
dime has a similar mechanism to feline generalised penicillin epilepsy. Vivid visual and auditory hallucinations were also a feature of our patient. Uncharacterised hallucinations without confusion have been previously noted with ceftazidime. The persistence of hallucinations for a few days after the withdrawal of the electro-clinical absence status strongly suggests that the hallucinations were not epileptic in nature and that these two neurotoxic side-effects have different mechanisms. The nature of the hallucinations closely resembled those of peduncular hallucinosis, which is usually associated with lesions involving the deep midline grey matter of the midbrain, hypothalamus and thalamus. We suggest that ceftazidime toxicity may result in a disturbance in the deep midline grey matter, causing a transient toxic peduncular hallucinosis.

Ceftazidime is not metabolised and is exclusively excreted by the kidney. Great care should be exercised in giving ceftazidime to patients with renal failure. Hallucinations should raise the question of toxic peduncular hallucinosis. A confusional state should be immediately investigated with an EEG to confirm the presence of absence status which can then be specifically treated.

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Spatial delirium following a right subcortical infarct with frontal deactivation

Reduplicative paramnesia, environmental delusion, disorientation for place, and spatial delirium are terms designed to a peculiar organic syndrome of delusional belief for a patient to be “relocated” in a place different from the actual one. This syndrome may be observed during recovery from confusional states after severe traumatic brain injury but also as a relatively isolated symptom after a focal lesion of the right hemisphere. About 16 cases with a documented focal lesion have been described in the literature. We present a case documented with CT, MRI and CBF study, in which lasting spatial delirium followed a small right subcortical infarct.

An 85 year old hypertensive right handed man, with no previous psychiatric history, and a former manager of an oil refinery, developed a fronto-basal intracerebral haematomas in December 1998 while on oral anticoagulant from a myocardial infarction. Neurological outcome was uneventful and a control CT scan showed resolution of blood collection. In February 1989, he was re-admitted with a recent left hemiparesis. Examination showed moderate predominately brachio-facial deficit, brisk tendon reflexes and absent Babinski’s sign. A mild left hemianopia was found with Goldman perimetry. There was no sensory loss. The patient was alert but mildly ananosognosic for his left sided weakness. He was oriented to time, place and persons; language and memory were unimpaired. However, although he acknowledged that he was in hospital, he expressed a strong belief that he was in Belgium (rather than France). Blood pressure was 140/80, heart auscultation was normal with sinus rhythm and no cervical bruit was heard. Weakness resolved subtotally within a week and the patient was discharged home. He was seen two months later because of paranoid delusions about his spatial location. While at home in Lyon, he was convinced that he was actually in another city which varied from day to day, but was mainly in Europe.

Thus he thought he was in Prague one day, in Brussel another, in Denmark, or in Morocco. According to the patient’s statement, these locations were stages on a mandatory tour planned for his rehabilitation. Each morning, he prepared to leave which resulted in a distressing effect on his family. In the different places, the patient was in familiar surroundings. The patient could describe the scenes very precisely, but he was not embarrassed though he could offer no explanation for them. Duplication or triplication of places occurred. The theme of the delusions were remarkably similar, and attempts at rationalisation failed to persuade him to the contrary.

His daily behaviour was normal except for this spatial disorder. Mental status was remarkably preserved in a clinical setting. Temporospatial orientation was normal. Speech was fluent and coherent. Immediate span was 6 for digits and 5 for spatial locations. WAIS scoring was in the high range (119), with verbal IQ (122) and performance IQ (111). Free drawing or copying of a cube was performed well. There was no visual agnosia, especially for faces and places, no loss of visual imagery, and no left hemispatial neglect. He was able to place main towns on a map of France easily. Environmental orientation was perfect. However, visuo-constructive abilities and spatial organisation

Figure 1. CT scan before (top) and after (bottom) onset of spatial delirium. The left frontal haematoma seen initially cleared and a right subcortical infarct appeared secondarily.
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