performance after vestibular stimulation (t = 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 task, though 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 dissipated 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 on 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 self-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 twelve 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 twelve 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 sepsis was 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 describe his 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 dialysis 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 μg/ml (normal peak level 55 μg/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 dialysis, the serum level was 34.4 μg/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-

Figure EEG performed on the sixth post-operative day. There was continuous generalised epileptic activity associated with a confusional state and subtle generalised myoclonic jerks. Following the administration of intravenous clonazepam, the epileptiform activity disappeared and the confusional state and myoclonic jerks immediately resolved.
dime has a similar mechanism to that of fenelzine in the patient's mental state. We also noted a distinct peculiarity organic syndrome that was observed in the patient. This syndrome was characterized by delusions, hallucinations, and other neuropsychological abnormalities. The presence of these symptoms was consistent with the literature on this subject. 

We investigated the patient's medical history and found that he had been treated with ceftazidime for a long period. This treatment was accompanied by a transient increase in the frequency of his symptoms, which was consistent with the literature on this subject. The patient was treated with ceftazidime for a long period, which was accompanied by a transient increase in the frequency of his symptoms, which was consistent with the literature on this subject. The patient was treated with ceftazidime for a long period, which was accompanied by a transient increase in the frequency of his symptoms, which was consistent with the literature on this subject.
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