could cause heightened cortical excitability and hence unilateral hallucinations.

(g) Hallucinations can sometimes lateralise to the good ear (Robinson, 1927). Electrical aural stimulation in cases of hallucinations and ear disease paradoxically affected the opposite ear (Ireland, 1893). In McCay & Healey's (1931) case a right radical mastoidectomy cured long-standing mental disorder including sinistral hallucinations. No brain disease was detected. A similar case of 'toxic insanity' came to post-mortem (Henderson *et al*, 1913). A man became very confused and irritable with hallucinations during an acute exacerbation of chronic otitis. There was considerable improvement of his mental condition with successful ear treatment, but he relapsed and died. There were old and new labyrinthine bone fistulas, but the brain was remarkably healthy.

(h) 'Temporal lobe' epilepsy can also arise from the ear (Ireland, 1893).

(i) Hallucinations can be attenuated by plugging the ear or induced by removing impacted wax (Ireland, 1893).

(j) Peripheral audiosensitivity can lead into hallucinations (Ireland, 1893).

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SIR: We were interested to read the letter of Khan *et al* (*Journal*, February 1988, **152**, 297–298) concerning a patient with unilateral complex auditory hallucinations. We report two further cases.

Case Reports: (i) A 58-year-old man was readmitted with his third episode of psychotic depression, characterised by depressed mood, loss of drive, grossly disturbed sleep, and loss of appetite and weight. Mental state examination revealed complaints of "a whistling noise", louder in his left ear, which he attributed to a "surveillance device", and derogatory "whispering voices" present only on the left side. During a previous admission the whistling had been accompanied by a single male voice As his mood improved, the voices faded and disappeared, leaving only a distant whistling in his left ear. Physical examination showed a marked sensorineural deafness in his left ear. He failed to attend for audiometry after discharge. All other investigations were normal.

(ii) A 47-year-old woman had initially been seen by psychiatrists at the age of 18 years because of a sensitive, inadequate personality. Alcohol dependence developed, complicated by grand mal epilepsy. She first complained of auditory hallucinations at the age of 33 years. During subsequent admissions, passivity phenomena, thought insertion, and broadcasting were documented at various times. Mental state examination during the most recent admission revealed a woman of low average intelligence, with slight blunting of affect. She described a persistent buzzing and a solitary, adolescent, female voice, both audible only in her left ear. Four EEGs over a period of 21 years showed diffuse slowing and bilateral spike activity with photic stimulation, most marked over the temporal leads. Audiometry showed bilateral high tone deafness; other investigations were normal.

In both these cases, unilateral hallucinations were present with hearing loss and ipsilateral tinnitus. In the first case the phenomenon appears to represent auditory hyperaesthesia during a depressive episode, leading to functional hallucinations (Hamilton, 1974). In the second case there was additional evidence of non-focal epileptiform activity which may have contributed to both the psychosis and the hallucinations.

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Inadequate Seizures with Electroconvulsive Therapy

SIR: Sharpe & Andrew recently published a Brief Report (*Journal*, January 1988, **152**, 134–136) citing the case of a severely depressed patient in whom treatment with electroconvulsive therapy was abandoned because of repeated failure to induce a convulsion. The authors suggested that failure to convulse may be an occasional and unrecognised cause of non-response to ECT, and concluded that adequate monitoring of seizure activity is required.

In my own centre a number of consultant staff independently observed that severely depressed patients were often not responding to ECT. This prompted a careful survey of ECT practice within the hospital over a period of 11 weeks, including monitoring of electrode placement, seizure duration, and concurrent medication (24 patients, 120 treatments).

Hospital practice is to use the lowest setting of current to produce a visible seizure, using an Ectron Duopulse Series 2A. If no seizure activity is observed, the stimulus is repeated once under the same anaesthetic, and a greater stimulus used for subsequent treatments (ECT setting 2 instead of setting 1).

Initially, seizure duration was monitored clinically by an independent observer; the interval between the stimulus and cessation of the clonic phase was measured using a stopwatch. Using this method, it was demonstrated that 55% of treatments had produced a seizure duration of less than 30 s (the arbitrarily defined cut-off point for missed or inadequate seizures used by many workers). Bilateral and unilateral electrode placements were used with approximately equal frequency. The percentage of unilateral treatments which produced an inadequate seizure was 62%, while for bilateral treatments the corresponding figure was 44% ($\chi^2 = 4.04$, d.f. = 1, $P < 0.05$). Five patients never achieved a seizure duration of 30 s or longer, and four of these were receiving unilateral treatments. Only four patients were receiving medication which might have elevated the seizure threshold, such as benzodiazepines.

It has been widely debated whether clinical monitoring of seizure duration in ECT is acceptable (Christensen & Koldbaek, 1982; Fink & Johnson, 1982; Fink, 1987). With the increasing sophistication of anaesthesia, cerebral seizure activity could be masked by the muscle relaxant. Because of this possibility, a two-lead portable EEG machine was subsequently employed to assess the validity of the clinical method. One patient receiving a course of right unilateral ECT was monitored throughout treatment using left tempero-parietal EEG leads. On no occasion did the clearly observable EEG spike discharges outlast clinical jactitation by more than 2 seconds. This finding is contrary to that of Christensen & Koldbaek (1982), and suggests that clinical observation is unlikely to underestimate seizure duration significantly. Furthermore, the discrepancy between the durations of seizures induced by bilateral and unilateral treatments remains.

This survey suggests that inadequate seizures with ECT may be very common, especially with unilateral treatments. Because of recent awareness of dose-related side-effects, clinicians have been encouraged to use the lowest current which will result in a seizure (Royal College of Psychiatrists, 1977; Pippard & Ellam, 1981). Has the pendulum swung too far, so that these 'threshold doses' are producing inadequate seizures?

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Diethylpropion, Bupropion, and Psychoses

SIR: I read with great interest the recent report by Carney (*Journal*, January 1988, **152**, 146–147) of psychoses associated with the use of the diet pill diethylpropion. We recently described four cases of psychoses which emerged during treatment with the unicyclic aminoketone antidepressant bupropion (Golden *et al*, 1985). Diethylpropion and bupropion share strikingly similar chemical structures; replacement of the former's diethyl group with a t-butyl group and the addition of a chloride atom to the meta position of the phenyl ring would yield the latter (Mehta, 1983).

We found that those patients who experienced psychoses in association with bupropion treatment demonstrated significant increases in plasma concentrations of the dopamine metabolite homovanillic acid, suggesting that perturbations in dopaminergic systems might underlie their toxic reactions (Golden *et al*, 1988). A similar mechanism of action might account for the psychotic symptomatology associated with diethylpropion.

Bupropion is still undergoing investigation in anticipation of its release for clinical use. Overall, we have found it to be a strikingly effective treatment for many patients who have failed to respond to conventional antidepressants. As in the case