Failure to Convulse with Electroconvulsive Therapy

SIR: Sharpe & Andrew (*Journal*, January 1988, **152**, 134–136) report on a case of a 32-year-old woman in whom it was not possible to induce a seizure despite 15 ECT applications. The author's are to be commended on the changes in technique such as the move from unilateral to bilateral, reduction in methohexitone dosage, extensive pre-oxygenation, and the use of chlorpromazine, all of which were designed to alter fit threshold.

The case raises the question as to whether we have moved too far in the direction of low energy stimuli in the design of ECT machines. The authors report energy in joules, but it is more conventional to refer to energy in terms of millicoulombs. The two machines that were used are not capable of delivering more than approximately 500 millicoulombs even at their maximum setting. This applies to most modern machines, including those manufactured in the United States. There is one machine available – the Neurotronics (NTS) Machine – which at its maximum setting will deliver 2000 millicoulombs. There may be a case for having a wider range of stimuli to allow for the occasional patient with a very high fit threshold to be successfully treated.

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SIR: Sharpe & Andrew (*Journal*, January 1988, **152**, 134–136) dismiss drug effects as a possible cause of the increased convulsive threshold in their patient, on the grounds that amitriptyline and chlorpromazine are known to reduce seizure threshold. While this is indisputable for spontaneous convulsions, tricyclic antidepressants antagonise electrically-induced seizures in animals (Spencer, 1976). Drug effects in these two situations thus appear to be quite different. In my experience, recognition of this in Canada led to the routine practice of discontinuing tricyclic antidepressants when giving ECT. Sharpe & Andrew do not indicate the concurrent dose of amitriptyline, but one alternative not tried in their case was discontinuation of all psychotropic medication during ECT.

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Hypomania and Epilepsy

SIR: Barczak *et al* (*Journal*, January 1988, **152**, 137–139) failed to find a "single case report of hypomania and epilepsy in the English-language press." I would refer them to Ferguson & Rayport (1984). In a series of 50 patients with a combined diagnosis of temporal lobe epilepsy and psychosis, Flor-Henry (1969) made a diagnosis of manic-depressive psychosis in 9 cases on the basis of periodic elation and depression with preservation of the personality.

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FLOR-HENRY, P. (1969) Psychosis and temporal lobe epilepsy: a controlled investigation. *Epilepsia*, **10**, 363–395.

Hypomania following Complex Partial Seizures

SIR: The recent report by Barczak *et al* (*Journal*, January 1988, **152**, 137–139) describes three cases of hypomania occurring as an interictal phenomenon in patients with complex partial seizures. The authors comment that the association is uncommon and note the apparent relationship with right-sided seizures. We describe a further similar case.

Case report: The patient is a colourful character, well known to the Liverpool psychiatric services, who was born in Trinidad of Portuguese parents. He first came to attention in 1975 at the age of 54 with a series of admissions, prompted by the police, following his disinhibited behaviour in public. He was described at the time as overactive, with marked flight of ideas, using clang associations and rhyming. He was also restless and sexually disinhibited, although cognitively remaining alert, responsive, and orientated. At the time he was maintained on phenytoin, which had been used to treat idiopathic epilepsy of long standing.

The following 12 years have resulted in more than 20 admissions to psychiatric wards, and a clear pattern has evolved. The patient has a single, or series of, grand mal convulsions which have been observed by staff on occasions. There then follows a usually brief period of postictal confusion and irritability, followed later by an elevation in mood, pressure of speech, flight of ideas, grandiosity ("I'm able to pour sunshine over the world"), sexual disinhibition (asking nurses, in Portuguese, to go to bed with him) and motor over-activity with sleeplessness. With treatment by neuroleptics, this hypomanic state settles within a few days, the patient usually returning to an euthymic state.

Serial EEGs have revealed a right-sided temporo-frontal lobe focus suggestive of subcortical epilepsy.

Attempts to control the epilepsy have tended to be thwarted by poor compliance, and there is more recent evidence of some EEG deterioration. We think that this case is another example of the association of right-sided epileptic activity with hypomania, occurring as an interictal phenomenon.

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SIR: I was very interested in the article by Barczak *et al* (*Journal*, January 1988, **152**, 137–139) describing three cases of hypomania following complex partial seizures. It reminded me of the following patient whom I saw originally two years ago. She had moved here from London.

Case report: She was a 40-year-old married woman with a five-year history of complex partial seizures since just before the birth of her second child. She had been investigated in Kent and later at the Maudsley Hospital, where investigations including EEG suggested an epileptic focus in the left temporal region, resulting from cerebral cortical venous thrombosis. A typical attack consisted of her becoming very quiet, then making repetitive hand movements, plucking at her clothes, smacking her lips, and speech becoming mumbled. The attacks were fairly frequent despite treatment with carbamazepine. In the past she had also experienced tonic/clonic seizures.

I was asked to see her urgently by the neurologist. Her husband had become very worried about her behaviour in the previous week. She had become increasingly emotional, her mood swinging from tears to euphoria. She had become preoccupied with religion and the fate of the world. One day, while taking the children to school, she became convinced that the school was evil, because its name was similar to the word hell. She believed the children were going to be murdered. She also had insomnia. Her husband reported that she had had a similar episode prior to her admission to the Maudsley, which culminated in a drug overdose. This second episode settled in a few days and she reverted to her usual self.

A few months later her husband telephoned me saying she had had three seizures, following which she had started acting strangely again. She had become very giggly and had not slept for two nights. She was again preoccupied with the end of the world and also guilt feelings about her past. Later she had become tearful and unable to cope normally. Once more, this lasted a few days before she reverted to her normal self.

It seemed to me at the time that these were short-lived hypomanic illnesses. I saw her last a year ago after an incident when she was reported to have dropped her trousers at a school function. This occurred when she was having frequent seizures, and seemed to be a form of automatism. Unfortunately her seizures remained frequent despite treatment with sodium valproate and carbamazepine. She has now moved to another area.

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Diethylpropion Hydrochloride-Induced Psychosis

SIR: We were most interested to read of the two cases of diethylpropion hydrochloride-induced psychosis reported by Carney (*Journal*, January 1988, **152**, 146) and we would like to draw attention to the similarities between those histories and two more patients who developed psychotic phenomena after taking appetite suppressants.

Case reports: (i) Mrs S., a 42-year-old computer operator, was referred to the psychiatric services for the first time after a two-week history of behavioural disturbance and paranoid ideation. She had multiple florid delusions, 2nd and 3rd person auditory hallucinations, and felt compelled to act upon the commands of these hallucinatory voices. Ideas of reference were marked; she believed that the television was spying on her and that people were staring at her. She was visually hallucinating. She had taken diethylpropion hydrochloride intermittently during the preceding year and was using it as an aid to weight loss before her illness developed. Her symptoms responded to chlorpromazine; she remains well at six months' follow-up, drug-free.

(ii) Mrs T., a 35-year-old divorcee with no past or family history of psychosis, presented with a one-week history of insomnia, restlessness and agitation, formed visual hallucinations, unformed auditory hallucinations, and persecutory ideation. Her symptoms began approximately 24 hours after ingestion of 25 × 30 mg capsules of phentermine taken as an impulsive gesture. On questioning, she believed that people had been spying on her through the windows of her flat. Her friends had been appearing before her eyes and then disappearing again. In 1983 she had been diagnosed as suffering from bulimia nervosa; there were no symptoms of eating disorders on this occasion. Three days prior to visiting the Emergency Clinic her GP had prescribed thioridazine (50 mg t.d.s.), and her symptoms had begun to recede from this point. She failed to attend for follow-up five days later.

A functional psychiatric disorder was unlikely in these cases because of the ages of onset, the negative family histories, and the patient's well-integrated premorbid personalities with preserved social functioning. There was a clear temporal relationship between drug usage and onset of symptoms, which resolved rapidly when the drug was withdrawn. These drug-related florid paranoid illnesses with