

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