clozapine's suspected epileptogenic properties (Heerlen & Kunze, 1979; Juul Povlsen *et al*, 1985). Herein, we report on one patient who developed spontaneous (tardive) seizures following only one electroconvulsive therapy treatment soon after clozapine discontinuation.

Case report: A 26-year-old man with a four-year history of chronic paranoid schizophrenia received up to 800 mg per day of clozapine with limited response as extreme anxiety, fearfulness and paranoia continued to impair his ability to function. At the time that ECT was recommended the patient was receiving clozapine (800 mg), propranolol (60 mg) and diazepam (20 mg) daily, which were then tapered over 14 days and discontinued, with the exception of diazepam which was reduced to 5 mg daily 72 hours before his first ECT session. The patient received bilateral ECT four days after his last dose of clozapine. Seizure duration monitored by two-lead electroencephalography (EEG) was 123 seconds. Recovery was remarkable for significant post-ictal confusion. The patient had two spontaneous *grand mal* seizures witnessed by staff on days four and six following this first and only ECT treatment. An EEG performed on day five after the first seizure and before the prescription of phenytoin was remarkable for mild defused slowing, with a 'questionable' focal abnormality in the right frontotemporal region. Non-contrast computerised tomography and magnetic resonance imaging were normal. The patient was ultimately discharged on maintenance phenytoin and clozapine with minimal improvement in his mental status, but no further seizure activity.

The inferences that can be drawn from this care are, however, unclear. We cannot rule out the possibility that clozapine stopped only four days before ECT in some way facilitated this patient's spontaneous seizures. However, other explanations are equally plausible. The patient had been on long-term benzodiazepine treatment which was reduced from 20 mg to 5 mg over two weeks; this may have been a contributing factor. It is also impossible to tell from an individual case whether this patient's seizure activity is significant in light of the reported one in 500 incidence of tardive seizure phenomena following ECT (Fink, 1977).

As a practical guide, given the suggestion of increased epileptogenic activity with the atypical neuroleptic clozapine, clinicians are advised to permit a drug-free period of 7–10 days following clozapine discontinuation before starting ECT. Theoretical risks of spontaneous seizure activity and prolonged seizure duration are raised which merit further study.

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Buspirone-induced mania

SIR: Buspirone, with its limited liability for abuse (Griffith *et al*, 1986), and lack of withdrawal effects (Tyrer *et al*, 1985), might be considered the drug of choice in the treatment of anxiety in patients with a history of alcohol or drug abuse. Such a patient, who developed mania after two weeks on buspirone, is presented.

Case report: A 28-year-old single man began abusing alcohol and heroin at 14 years of age. From 1984 onward he binged sporadically on alcohol, but remained off heroin. He presented for treatment in November 1989, with severe symptoms of anxiety. He was commenced on buspirone (10 mg b.d.), and also agreed to take disulfiram (400 mg daily). He took the buspirone regularly for two weeks, but took the disulfiram only intermittently. He drank on occasion during this period, but denies any other drug abuse. While on buspirone he described a 'floating feeling', and noted his thoughts going faster. Over the next few weeks he developed pressure of speech, flight of ideas, irritability, elated mood, and overactivity. There was no clouding of consciousness and he denied perceptional disturbances. Physical examination was normal. A drug screen was negative. The patient required high-dose neuroleptic medication to which he responded slowly. Neither he nor his family have any history of affective disorders.

To date there has been three published reports of buspirone causing mania. Two of these (Liegghio & Yeragani, 1988; McDaniel *et al*, 1990) involved patients who already had a diagnosis of bipolar disorder. In both, the introduction of buspirone precipitated a manic swing. In the third (Price & Bielefeld, 1989) a 38-year-old man with resistant depression and anxiety became hypomanic on the two separate occasions that buspirone was introduced.

Buspirone is thought to exert its effect by interaction with 5-HT1A receptors, and enhancement of dopaminergic and noradrenergic activity. These differential effects have been linked to the development of psychotic behaviour. The facilitation of dopaminergic function by buspirone may be more clinically significant than hitherto thought and is possibly the mechanism through which this drug induces mania.

In this case, it could be argued that the onset of mania had nothing to do with buspirone. However, this appears unlikely: the patient had been withdrawn a number of times from alcohol and had taken disulfiram in the past, both without adverse effects. There have been no reported interactions between buspirone and disulfiram. The temporal relationship of being on buspirone and becoming manic, would seem to preclude any role that alcohol may have had in the onset of his symptoms.

If buspirone does cause hypomania, its use in alcoholics, or drug addicts, although attractive, should be carefully monitored and the relative risk of precipitating a psychotic illness weighed against the attraction of using an anxiolytic which is reputed to be free of abuse or dependency.

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Educating the psychiatrist of the 21st century

SIR: As I read Cawley's Lecture (*Journal*, August 1990, **157**, 174–181), with its persuasive *tour d'horizon*, I found myself becoming increasingly restive. Finally, and doubtless because I live abroad, I spotted the trouble – his title was wrong. It should have been "Educating the British NHS consultant psychiatrist in the 21st century". If we hold psychiatry to be a discipline like others that transcends local administrations and national frontiers, the education of psychiatrists in general is another matter.

As psychiatrists go, the British consultant is a peculiar animal in ways that I can only outline here. He or she is employed in large hospitals and institutions, and would be classified as a hospital or a government psychiatrist in some other countries. He has concerns with 'management', and now 'audit', which are of no interest or incomprehensible to many psychiatrists elsewhere. He is one of a team of public employees that deals with the patient, and he does relatively little clinical work by himself, and even less of the kind sustained for months or years with individual cases, from their start to their finish. He may go into the outside world with his team but he does not belong to it, and therefore has scant familiarity with the main mass of psychiatric disorders, even though they are often as destructive to human welfare as the atypical fraction he meets in hospital. Indeed, as Professor Cawley puts it, he and his team "may have little direct experience of the vast majority of the clinical problems in the field in which they claim expertise".

Far from taking psychoanalysis near its centenary peacefully for granted as integral to the study of mind, culture, and society, the British consultant psychiatrist sees it still as totally controversial, if not crackpot, and of no everyday clinical relevance, while quite unaware that there are neighbouring countries (e.g. Germany) where psychoanalytic therapy is funded by the health service, widely available, and reinforced by university departments. Although seldom possessing serious knowledge of it, he can, as Professor Cawley sadly observes, be ferocious in belittling psychoanalysis, unless he belongs to a small eccentric minority who have a habit of forsaking psychiatry altogether for psychoanalytic practice amid a non-medical fraternity almost confined to London.

If we forget, as I fear Professor Cawley has done, that this animal is only one type of psychiatrist, but by no means the world over the common one, we can easily agree that it is absolutely essential for the psychiatrist in training to be schooled in "modern management techniques", in collaborative research with neuroscientists, epidemiologists, and others, and in sharing his patients with a multiform team, all of which could seem esoteric luxuries and bizarre priorities elsewhere. Also, we can then easily forget to ask the vital question whether this is the psychiatric animal that we want to go on producing at all.

However, once the question is asked, we are up against it because we seem to have no choice but to go on producing what we do. Professor Cawley looks for innovations in psychiatric training in Britain but he does not examine the obstacles to effecting any very substantial changes, let alone fundamental ones. As I have pointed out (Bourne, 1988), these obstacles are the more huge for being invisible because they are buried in the embryology of the British Psychiatry – not only in his basic medical education, in his prejudicial studies, and in his selection as a medical student in the first place, but in his schooling from