and can spot other people's grosser errors (Austin, 1961). Most of us rapidly become quite lost, however, if we venture to describe the rules that we automatically follow. It is one thing to talk with concepts, but quite another to talk about them: that is the business of philosophers.

The central issue in criminal trials is whether defendants did something that they ought not to have done, without an adequate excuse. If they did then it is considered to be right to hold them responsible for their behaviour, blame them for it, and punish them. The defences that are raised are excuses – "I didn't know what I was doing" (McNaughton rules), "I was not in control of my actions" (automatism), and so on. All this, as Dr Fenwick observes, is to do with mental phenomena.

The defendant's medical condition is relevant to this process only insofar as it provides an excuse for what was done (except in those special cases where medical evidence bears on whether he/she did the *actus reus*). Here we get into the area of the relations between mental phenomena and cerebral phenomena. Philosophers have argued over the details of this area at great length without reaching any very satisfactory conclusions, but for most practical purposes in the witness box one can say in a loose sort of way that cerebral phenomena cause mental phenomena. What the doctor must do is explain to the court in ordinary language what the medical findings are (some of these statements are likely to be about the defendant's brain and some about the defendant's mind) and how they illuminate the defendant's state of mind and actions at the time of the offence. If a doctor mixes "brain words" with "mind words", as in "guilty brain" or "hypoxic mind", the members of the jury will think that the doctor is speaking metaphorically or uttering nonsense.

Doctors and lawyers will always speak rather different languages. What matters is that they should use language precisely and attempt to keep in touch to some extent with each other's ways of thinking.

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Life events and relapse in bipolar disorder

SIR: Dr McPherson and colleagues (*BJP*, September 1993, **163,** 381–385) comment on life events and relapse in bipolar affective disorder. However, there are limitations in the usefulness of the ideal of life events as factors in disease causality.

In a recent retrospective analysis of 36 bipolar patients admitted to our hospital over 12 months, we discovered significant life events in 12. However, a diagnosis of substance abuse was found in 18 of the 36, and a history of non-compliance with medications for lengthy periods before admission in 17. No assessment was made of the effect of the latter two factors on the illness process itself. Of the 12 patients with significant life events preceding admission, two had a substance abuse problem combined with non-compliance with treatment, and a further four had problems with one of these two factors. Therefore 6 of the 12 patients experiencing life events also had confounding factors influencing their illness. We agree with McPherson *et al* that compliance may well be a confounding variable in the evaluation of the effects of life events on the rate of relapse.

Our observations suggest that the prevalence of substance abuse and problems with compliance are high among bipolar patients who describe life events preceding their hospital admission. We feel that research into the relative effects of these two factors on relapse rates is required, and indeed study of the effects of these conditions on life events themselves might also be of benefit.

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Is there a lithium withdrawal syndrome?

SIR: Professor Schou (*BJP*, October 1993, **163,** 514–518) examines the evidence for a lithium withdrawal syndrome. His argument, which is based on terminology and the definition of the term 'rebound', is indeed very convincing. He describes rebound as a phenomenon leading to a temporary increase in the frequency of an episodic disorder following discontinuation of a specific treatment. A good example of a rebound phenomenon in an episodic disorder is seen in the treatment of epilepsy. Abrupt withdrawal of the anti-epileptic results in either status epilepticus (rebound in intensity) or increased frequency of epileptic attacks. This follows immediately on withdrawal and is commoner in those who have received the anti-epileptic for a long time. According to this example, there are several factors which

must be present to constitute a 'rebound': abrupt withdrawal of the drug; a relatively long period of treatment before withdrawal; an increase in the frequency and/or intensity of the episodes; a brief interval between withdrawal and relapse.

Several studies have found relapses after lithium discontinuation. The older studies (Baastrup *et al*, 1970) examined the efficacy of lithium in the prophylaxis of manic–depressive psychosis and therefore the temporal relationship between drug withdrawal and onset of illness was not closely examined. More recently there has been evidence to suggest that abrupt withdrawal of lithium can lead to a rapid recurrence of affective symptoms, especially mania (Mander & Loudon, 1988). These studies have shown that many patients relapse within two weeks of drug withdrawal, that these relapses warrant urgent hospital treatment, and that these patients have received lithium for an average of over three years – all evidence in keeping with the above criteria and strongly in favour of a rebound phenomenon, contrary to the views expressed by Professor Schou.

An abstinence phenomenon with lithium withdrawal is to be expected given the effect this drug has on various systems in the body. Therefore, it is somewhat surprising that no somatic or physiological symptoms have been described in relation to its withdrawal (Balon *et al*, 1988). Moreover, the symptoms of nervousness, irritability, insomnia and labile mood which have been observed after lithium withdrawal are unlikely to be abstinence phenomena, as Professor Schou has pointed out. It is also interesting to note that Christodoulou & Lykouras (1982) demonstrated a reduction in symptoms such as hand tremor, fatigue and increased tendon reflexes, which are commonly associated with withdrawal states following lithium discontinuation.

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SIR: The article by Professor Schou opens up the discussion on the possible drawbacks of lithium treatment. His suggestion that the data from the pooled analysis by Suppes *et al* (1991) be plotted on a semilog scale is a good one, and when I did this it appeared that there are two slopes, indicating a steeper withdrawal phase over the first three months.

An interesting study that Professor Schou did not consider is that of Coxhead *et al* (1992), in which half the patients (randomly and blindly) were suddenly switched from lithium to carbamazepine. As carbamazepine has been shown to be as effective a prophylactic as lithium, one might expect that there would be a similar rate of recurrence. On the other hand, if lithium withdrawal markedly increased the vulnerability to recurrence, then one would expect that, immediately after the switch, considerably more of the patients who were switched would experience a recurrence. The results pointed to the latter conclusion, with 7 of the 15 patients switched to carbamazepine relapsing within two months, compared with only 2 of the 16 patients who remained on lithium. At 12 months the drugs appeared to be equivalent in efficacy: eight of the carbamazepine group and nine of the lithium group had relapsed. This suggests that it is the most vulnerable patients who are 'picked off' by lithium withdrawal.

I think that it is not only possible that lithium withdrawal is a cause of recurrence in bipolar disorder, but that it may be a common cause. Along with two colleagues, I reported an observational study of bipolar patients, selected by consecutive admission, looking at the relationship of life events to recurrence (Hunt *et al*, 1992). In the course of this two-year study, 9 of the 18 who were on lithium (but not on carbamazepine or antipsychotics) suffered a recurrence; four became ill within six weeks of stopping lithium, suggesting that a significant proportion of recurrences might be due to rebound mania on withdrawal of lithium.

It would seem likely that if this effect does occur with lithium it would also occur with other drugs which are effective in the prophylaxis of bipolar disorder. I know of no well conducted trial that has examined this possibility, but have seen one case in which the withdrawal of carbamazepine was associated on two consecutive occasions with rapid recurrence and an overall marked shortening of cycle length.

In my view, given the current state of knowledge, it would appear to be advisable only to start treatment with lithium (or carbamazepine) if both the patient and doctor understand that though long-term treatment is likely to improve outcome,