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