Suicide has previously been reported (Bebbington, 1976), but we are not aware of a report of fire setting associated with this condition, thus indicating that this may be a rare occurrence. However, fumigation is often used by these patients and it seems essential to explore any attempts or intentions by them to use fire or smoke to combat the infestation, and to consider admission in order to protect both the patient and neighbours. Many such patients relapse when treatment is stopped, which raises legal and ethical difficulties when long-term treatment is attempted.

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Is there a Lithium Withdrawal Syndrome?

SIR: The letter from Hawkins & Shepherd (*Journal*, February 1987, **150**, 273) contained a number of inaccuracies and misconceptions.

Firstly, they state that the words 'withdrawal' and 'relapse' are not interchangeable. I make it clear in my opening sentence that the paper is concerned with "the possibility of relapse being caused by drug withdrawal". Their criticism would seem to be an unnecessary exercise in semantics.

Secondly, they state that to show a withdrawal effect it is necessary for there to be a "fall in the relapse rate lower than expected for a period after the withdrawal syndrome has ended". This is clearly wrong. The question is whether there is an *increased* risk of relapse in the withdrawal period in addition to that which would be expected taking into consideration the natural history of the disease process. This is not the same thing.

Thirdly, they state that the theoretical relapse rate "can never be known", and ask "How can one distinguish a withdrawal state causing relapse and relapse alone?" Perhaps they are not aware that this is why control groups are used in order to estimate the theoretical relapse rate. In the control group only 8% of patients relapsed in the first three months, compared with 28% in the experimental group. This difference cannot be explained by the experimental group being at higher risk, because after three months the relapse rates are identical.

Finally, no firm conclusions can be drawn from a retrospective study, hence the title poses a question. Correspondence in the same issue (*Journal*, February 1987, **150**, 264–265) has highlighted the fact that despite increasing use of lithium there has been an increasing readmission rate for mania at a number of different centres. There has been no satisfactory explanation for this. One possibility is that repeated lithium withdrawal increases the number of relapses, and I think this topic deserves further consideration.

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BITE: Self-rating Scale for Bulimia

SIR: The paper by Henderson & Freeman (*Journal*, January 1987, **150**, 18–24) is a useful and timely development in the scientific study of eating disorders. However, four points require further consideration.

Firstly, the authors need to clarify whether they regard the BITE as a screening test or as a diagnostic instrument. The statement that "subjects achieving a high score have a high probability of meeting... criteria for bulimia" indicates clearly that it is a screening test. However, the observations that the BITE "can be used to *identify* binge-eaters in a given population" (our emphasis), and that it provides the information necessary to make a DSM–III diagnosis of bulimia, suggest that the authors also consider it to be a diagnostic instrument.

Secondly, the criterion for caseness is unclear. In study 1, the only criterion given for the patient group is that they were binge-eaters at "various stages of treatment". This is too imprecise – an operational definition is a central requirement for work of this nature. The absence of such a definition from study 1 is all the more mysterious since one was used in study 2. Where diagnostic criteria are considered, it is not always clear which are meant. DSM–III criteria were used in studies 2, 3, and 4, while the authors conclude, in their instructions for administration, that high scorers have a high probability of meeting "DSM–III criteria for bulimia *and* Russell's (1979) criteria for bulimia nervosa" (our emphasis). There is a crucial difference between the two: DSM–III criteria are relatively broad, in contrast to Russell's criteria which require evidence of a morbid fear of fatness. By which criteria does the BITE identify cases?

Thirdly, the authors adopted a case-control strategy. This method gives rise to over-optimistic estimates of the validity coefficients. As Williams *et al* (1980) noted:

> "a group of symptoms selected on the basis of the ability to discriminate between two distinct populations, i.e. 'known ill' and 'known well', may be effective in classifying respondents who happen to come from one of those groups. However, in epidemiology we are not presented with individuals who clearly belong to one of these two groups: we are presented with individuals whose probabilities of illness are distributed along a continuum. Instruments which can distinguish clearly between distinct caseness groups, i.e. well-separated locations on the continuum, need not necessarily perform well in classifying individuals from various and intermediate probabilities of illness."

Another problem with the case-control approach is that since the prevalence of caseness in the study population is set at 50%, the resulting positive predictive value will be considerably higher than that appropriate to the use of the same test in a population where the prevalence is much lower than 50% (Williams *et al*, 1982), as is invariably the case with eating disorders.

Fourthly, there are several methodological points which require clarification. For example, why did the control group in study 1 contain both men and women, whereas the patient group consisted only of women? How were the sub-scales derived? How were the cut-off points decided upon? Where does the proposed lower cut-off (10), which appears in the discussion but not the results, come from? This cut-off is claimed to be relevant in the identification of sub-clinical groups: how can this be so, when no such patients were studied?

The authors are premature in their claim that the BITE is "a tested, valid questionnaire". For example, they say that "the modified BITE produces neither false positives nor false negatives". This is much too sweeping a claim, based as it is on one relatively small validation study. While this questionnaire may fulfil an important need, more development work is required.

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Predictions of Outcome in Depressive Illness

SIR: Carney *et al* (*Journal*, January 1987, **150**, 43–48) claim that their study supports the dualist theory of classification of depressive illness. They reach this conclusion on the basis of the finding that their sample of depressive in-patients was not normally distributed on the Newcastle scale and the finding that outcome after two weeks differed between the endogenous and the neurotic groups. Their conclusions about both of these findings are open to different interpretations.

Firstly, depressed patients who are admitted to hospital are extremely unlikely to be a representative sample of all depressed patients: they have generally failed to respond to general practice or out-patient treatment with antidepressant medication. These non-responders will contain disproportionate numbers of patients with severe neurotic and severe endogenous features, the first group being relatively immune to physical treatments, the second group requiring more vigorous physical treatments. Thus, it is hardly surprising that depressed patients admitted "on clinical grounds" do not show a normal distribution of scores on the Newcastle scale.

Secondly, their conclusions about differing outcome between the two groups derives from a comparison of measures before and after fourteen days of a trial of antidepressant medication. Outcome is thus confused with treatment response. As the authors state, albeit in a different context: "the wisdom of attempting to base conclusions about diagnosis and classification on the response to a particular treatment is basically unsound".

Thus, these findings provide no convincing evidence for the dualist theory of the classification of depressive illness.

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The Impact of a Liaison Psychiatric Service on Patterns of Referral in a General Hospital

SIR: It is interesting to read of a change in referral rate associated with the organisation of a liaison psychiatric service (Brown & Cooper, *Journal*, January 1987, **150**, 83–87). However, it would be misleading