SIR: Mr Smeeton and Dr Wilkinson ask why we categorised our patients on the basis of their lifetime history. The answer is that this method seemed to us the simplest, and to rest on no assumptions about patterns of repetition. Their proposal to classify patients solely by the number of episodes in the preceding two years implies that such clustering is a salient and consistent feature of repetition, and while there is some interesting evidence that this might be so, we do not think that at present it provides a secure basis for classification. Moreover, contrary to another of their suggestions, it turns out not to be true that the number of parasuicidal episodes increases linearly with age simply as a function of increased time at risk. The older patients in our series show a different pattern of parasuicide from younger subjects; they are predominantly first-ever admissions, and major repetition is distinctly rare. Our present view is that such patients are quite a different group from the younger ones, and probably reflect differences both in psychopathology and in social contex.

Mr Smeeton and Dr Wilkinson raise the possibility of a birth cohort effect for parasuicide, much as proposed some years ago by Alderson (1974). They also indicate that formally testing such a model is scarcely feasible. But for what it is worth, we have found in our own data, which extend back to 1968, that the frequency of a prior episode in relation to age has constantly been much as reported in our recent paper. There is thus little support for a cohort effect. Alderson (1985) has come to the same conclusion for England and Wales.

Finally, they suggest the use of cluster patterns as a prognostic indicator among the major repeaters, referring to their own studies and work by others. This approach is eminently worth pursuing, but can only be applied to a small minority of all patients – less than 3% in their 1987 paper. Most repetitions will be generated by the much larger group of first-ever and 'minor' repeaters. Even for the major repeaters it would be unwise to base predictions solely on the past history of parasuicide and to ignore the many other aids to prognosis that are now available. Indeed, if we understand them correctly, Mr Smeeton and Dr Wilkinson themselves appear to recognise this point. At present there is no substitute for comprehensive psychiatric and social assessment in any attempt at prediction.

NORMAN KREITMAN
MRC Unit for Epidemiological Studies in Psychiatry
Royal Edinburgh Hospital, Morningside Park
Edinburgh EH10 5HF

PATRICIA CASEY
Regional Hospital, Wilton,
Cork, Ireland

References

ALDERSON, M. (1974) Self-poisoning – what is the future? *Lancet, i,* 140–143.
— (1985) National trends in self-poisoning in women, *Lancet, i,* 974–975.

Loss and theft

SIR: In Fishbairn's letter about shoplifting (*Journal,* June 1988, **152,** 845–846), he wrote about risk-taking having an antidepressant effect and wondered if this was the mechanism behind shoplifting. In some 90 cases which I have studied, the event which always precipitated the risk-taking behaviour was a loss, or a threatened loss, of a significant other held in a symbiotic relationship with the self, which was experienced as a catastrophe and necessitated that they were caught in the act. This behaviour is similar to the all-or-nothing mode of the narcissistic personality, and ensures that someone cares when it was felt that nobody cared at all.

An illustration can be seen in the behaviour of a tertiary-educated woman who had been a compulsive shoplifter and had been seen for a year for twice-a-week psychotherapy, and had not shoplifted over that period. In a session it was suggested by the therapist that consideration could be entertained to work towards the ending of therapy. An hour later I was rung by the police to say that she had been caught shoplifting and that her mental state was such that they thought she needed hospitalisation.

R. S. GILLEN
28 Ward Street
North Adelaide
South Australia 5006

The use of propofol for anaesthesia during ECT

SIR: Rands (*Journal,* January 1989, **154,** 125) is correct to draw attention to the adverse effect of propofol anaesthesia on seizure duration during electroconvulsive therapy (ECT). In a study involving 25 patients we observed that the use of methohexitone produced a median (QD) seizure duration of 33.0 (7.8) s, whereas the use of propofol significantly reduced this to 19.0 (9.0) s ($P < 0.01$) (Simpson *et al,* 1988). We concluded that propofol was not an appropriate agent for ECT anaesthesia, and that methohexitone should remain the standard agent. Since then, two other studies have shown that both observed seizures (Rouse, 1988) and cerebral electrical seizure activity (Dwyer *et al,* 1988) are attenuated by propofol.

In view of these findings we feel that the use of propofol anaesthesia for ECT should be

https://doi.org/10.1192/bjp.154.5.721b Published online by Cambridge University Press

discouraged, despite the obvious advantages of the drug for brief anaesthesia.

K. H. SIMPSON
R. P. SNAITH

St James University Hospital
Beckett Street
Leeds LS9 7TF

References

DWYER, R., MCCAUGHEY, W., LAVERY, J., *et al* (1988) Comparisons of propofol and methohexitone as anaesthetic agents for electroconvulsive therapy. *Anaesthesia*, **43**, 459–462.

ROUSE, E. C. (1988) Propofol for electroconvulsive therapy. A comparison with methohexitone. Preliminary report. *Anaesthesia*, **43** (Suppl.), 61–64.

SIMPSON, K. H., HALSALL, P. J., CARR, C. M. E., *et al* (1988) Propofol reduces seizure duration in patients having anaesthesia for electroconvulsive therapy. *British Journal of Anaesthesia*, **61**, 343–344.

Sex chromosomes and psychosis

SIR: The paper by Crow (*Journal*, November 1988, **153**, 675–683) raises many interesting points. He suggests that there is a pseudoautosomal locus on sex chromosomes for psychosis, but fails to mention whether the gene is dominant or recessive. We presume he has a dominant gene in mind, because one would not expect to find vertical concordance of sex with a recessive gene. The following possibilities occur to us for genes on the X or Y (underlining indicates presence of psychosis gene): paternal inheritance, (a) $\underline{X}Y \times XX = XX, \underline{X}Y, \underline{X}\underline{Y}$ ... (b) $\underline{X}Y \times XX = \underline{X}X, \underline{X}\overline{X}, XY$ ...; maternal inheritance, (c) $\underline{X}X \times X\underline{Y} = \underline{X}\underline{Y}, \underline{X}X$ ....

Dr Crow presents data to show same-sex concordance in sibs in schizophrenia, and has evidence for paternal transmission. This is consistent with (a) and (b). An excess of father-son over father-daughter transmission leads him to suggest a locus for schizophrenia with preferential mutation on the Y chromosome (situation (a)). Sib sex concordance is quoted for affective illnesses, as are excess of female-female sib pairs and a deficit of father-son transmission. These observations lead him to suggest preferential mutation on the X chromosome for affective illnesses (situation (b)). The lack of mother-daughter and mother-son difference is consistent with (c). We think it is an interesting hypothesis that explains certain cases of psychosis, but the model becomes strained if it attempts to account for all the epidemiological data. When Dr Crow tries to explain the greater severity in male schizophrenics he is referring to an X-linked semi-dominant model. We would also expect an excess of male schizophrenics and male-male pairs if we accept his model of preferential mutation on the Y chromosome for schizophrenia. He has attempted to explain these anomalies, but most importantly has failed to offer any evidence for paternal inheritance in affective illnesses which would be expected with a sex concordance in sibs. How does one explain the lack of same – sex concordance in the other sibling pairs? We would like to suggest that the reason may be genetic heterogeneity as well as variable penetrance.

A possible problem associated with genes in this region is the small size of the segment. Chromosomally, it cannot be larger than about 0.5% of the genome. Furthermore, the particular region that would give the exact concordance of sex must be a small segment on the border between the pseudoautosomal region and the region of the X- and Y-linked genes, because the obligatory crossover must occur proximally to the genes in question most of the time (>80%) for the concordance to be observed. Thus the total region in question must be less than 0.1% of the genome and possibly even smaller. This does not mean that the gene cannot be in this region, but it does mean that the possibility can be checked easily.

If we accept heterogeneity as a possibility, a case for X- and Y-linked genes can be made to give most types of concordance. Several types of sex-linked inheritance can be distinguished:

(a) X-linked lethals (often called sex-linked dominant): these are always passed on maternally, and would produce concordance of mother-daughter and sisters, as well as a deficiency of affected males, and a distorted sex ratio (2:1).
(b) X-linked true dominants: where male and female are similarly affected, would produce sister concordance from paternal transmission and no concordance from maternally affected individuals.
(c) X-linked semidominants: paternally passed on, genes would produce concordance of mild cases in the sisters and no affected sons. Maternally transmitted genes would show concordance of mild cases between the sisters and the mother, and concordance of severe cases between brothers.
(d) X-linked recessives: would produce, if passed on from the mother, concordant brothers; if passed on paternally, no affected individuals would be produced.
(e) Y-linked genes: always passed on paternally, and concordance is complete between all males.

We realise that no single hypothesis will account for all the data. However, it is possible that a gene or