and non-debt group (37% and 44% respectively, $\chi^2=0.03$, d.f.=1, NS). The comparable proportions for the control group were 43% and 2% (Fisher exact, $P=0.006$).

This absence of a difference in depression between those in and not in debt in the DSH group is at variance with the Hatcher's report. Methodological differences, a difference in the proportion of males, the inclusion of those other than self-poisoners and bias introduced by nursing staff who did not give questionnaires to all DSH patients may explain this. The similarities in ages and the proportions in debt are however striking.

This study found that a brief questionnaire was acceptable for both DSH and this control group. The proportion of questions answered about debt was high and was representative of the other questions. This method may enable large control groups to be studied. In addition this study supports the hypothesis that debt is more common in the DSH population. To draw further conclusions from these numbers is hazardous although the comparative levels of depression associated with debt in both DSH and control populations warrants further investigation.

HAMER, D., SANJEEV, D., BUTTERWORTH, E., *et al* (1991) Using the Hospital Anxiety and Depression Scale to screen for psychiatric disorders in people presenting with deliberate self-harm. *British Journal of Psychiatry*, **158**, 782–784.

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Risk of HIV for women who inject drugs

SIR: Gossop *et al* (*BJP*, January 1994, **164**, 101–104) suggest that women may be at higher risk of relapse to drug use than men because of their closer social attachment to other drug users. Preliminary results from our survey of opiate users presenting for treatment in south London are broadly in line with those of Gossop *et al*. However, we found that women who inject drugs may also be at greater risk of HIV infection than men.

Women comprised 24% of a sample of 97 opiate-dependent drug users questioned about sexual and injecting behaviours. They were more likely to be living with a drug user (61% compared with 31% of men; $\chi^2=6.6$, $P<0.05$) and to have a sexual partner who was an injecting drug user (70% compared with 22% of men; $\chi^2=18.2$, $P<0.0001$). There were no significant differences between the proportions of men and women who had recently injected drugs, but women were more likely to have injected themselves with equipment that had been used by someone else (30% of women compared with 11% of men; $\chi^2=5.2$, $P<0.05$). For both sexes, the majority of equipment sharing episodes involved their regular sexual partner, with whom condoms were used by only 20% of male and 25% of female clients.

In drug-using couples, women may have less opportunity to negotiate safer sexual and injecting practices, because men play a more active role in procuring drugs and injecting equipment (Klee, 1993). This particular aspect of relationships between male and female drug users has implications for HIV prevention strategies.

KLEE, H. (1993) HIV risks for women drug injectors: heroin and amphetamine users compared. *Addiction*, **88**, 1055–1062.

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Dysphagia in the neuroleptic malignant syndrome

SIR: The neuroleptic malignant syndrome (NMS) is a recognised complication of psychotropic drug use with a variable outcome (Bristow, 1993). We describe two cases where dysphagia with loss of a gag reflex was a major presenting feature.

Case 1: A 25-year-old male presented acutely psychotic and received zuclopenthixol, chlorpromazine and droperidol. He developed NMS with dysphagia, loss of a gag reflex followed by right lower zone pneumonia which required ventilation for 7 days, during which time he received dantrolene, bromocriptine and lorazepam (Gratz *et al*, 1992). His fever finally settled on imipenem. No organisms were grown. He was noted to have absent bowel sounds which returned after 9 days following the administration of cisapride. However his swallowing was unaffected by this and returned on day 25. An MRI scan of the brain during the dysphagia showed no evidence of brainstem damage.

Case 2: A 32-year-old male presented with dysphagia and fever and was noted to have a poor gag reflex. He had been receiving chlorpromazine and flupenthixol for 7 weeks. A diagnosis of NMS was made and he received dantrolene, bromocriptine and lorazepam. He developed signs of right lower zone pneumonia which responded to ciprofloxacin and erythromycin. By day 11 he could speak but still was unable to swallow; MRI scanning showed no brainstem abnormalities. He required a tracheostomy and feeding gastrostomy. He had a fatal cardiorespiratory arrest on day 16 due to bilateral pulmonary emboli.

Both patients developed complete dysphagia and reduced gag reflexes prior to the administration of medication for NMS, followed by the development of right lower zone pneumonia, probably due to aspiration. Dantrolene and lorazepam have muscle relaxing properties, and bromocriptine has been associated with reflux oesophagitis (Katzung, 1989). They could have slowed the recovery of the swallowing reflex and worsened aspiration. The first patient received cisapride, a pro-kinetic agent (Walker, 1994), which seemed to be associated with the return of bowel sounds although no improvement in swallowing occurred. Patients who develop NMS should have their gag reflex assessed early and if reduced or absent a nasogastric tube would be recommended to reduce the risk of aspiration.

BRISTOW, M. F. & KOHEN, D. (1993) How 'malignant' is the neuroleptic malignant syndrome? *British Medical Journal*, **307**, 1223–1224.

GRATZ, S. S., LEVINSON, D. F. & SIMPSON, G. M. (1992) The treatment and management of the neuroleptic malignant syndrome. *Neuropsychopharmacology-Biology-Psychiatry*, **16**, 425–443.

KATZUNG, B. G. (ed.) (1989) *Basic and Clinical Pharmacology*, pp. 264–275, 338–339. Englewood Cliffs, NJ: Prentice Hall International (USA).

WALKER, G. (1994) *ABPI Data Sheet Compendium 1993–4*, pp. 703–706. London: Datapharm Publications.

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Clozapine-induced neutropenia – or not

SIR: We wish to bring attention to a point of concern between clinicians and the Clozapine Patient Monitoring Service (CPMS), which may increasingly become an issue as more patients receive clozapine.

Because of the danger of neutropenia (3%; Veys, 1993) and its potentially fatal sequelae, it is absolutely right that regular blood counts be taken.

However, we have had experience of two patients whose survival prospects (through lower suicide risk) and quality of life were radically improved by clozapine. Despite suffering from severe schizophrenic illness unresponsive to conventional neuroleptics, they were able to be discharged from hospital. Both, unfortunately, then had one abnormal 'red alert' result. Because of the inevitable delay between blood sampling and the results, both patients had continued to take clozapine until the request for the emergency sample was made. In both cases these repeat samples taken 24 h after the 'red alert' sample showed a near doubling of the neutrophil count, with values well into the normal range.

It is known that neutrophil counts vary according to such factors as time of day, physical activity and presence of viral illness (Lewis, 1974). We understand the caution of the pharmaceutical company and the conditions of its licensing agreement, but is it not possible that one isolated result may be due to factors other than clozapine?

We do not question that patients who have had a true clozapine-induced neutropenia should not be rechallenged with the drug (Safferman *et al*, 1992), but we have seen the tragic consequences of stopping clozapine. Is it not over-reacting to stop clozapine on the basis of one blood result, considering the impact on the lives of patients denied its benefits?

As clinicians, in consultation with the patient, relatives and multi-professional team, we would be prepared to take the risk of continuing clozapine in these cases, but we are not permitted to do so. Would it not be possible to amend the regulations so that there must be two consecutive 'red alert' results in 24 h before clozapine is stopped?

LEWIS, S. M. (1974) The constituents of normal blood. In *Blood and its Disorders* (eds R. M. Hardesty & D. J. Witherall), pp. 3–68. London: Blackwell Scientific.

SAFFERMAN, A. Z., LIEBERMAN, J. A., ALVIR, J. M. J., *et al* (1992) Rechallenge in clozapine-induced agranulocytosis. *Lancet*, **339**, 1296–1297.

VEYS, P. (1993) A review of the possible mechanisms of Clozaril-induced neutropenia. *Clozaril Newsletter* (CPMS), **3**.

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Administrative problems limiting electroconvulsive therapy

SIR: In the case report of NMS with prolonged hospitalisation, tracheostomy, intubation, and artificial ventilation (*BJP*, January 1994, **164**, 120–122), Cape notes that the patient "also had one