

had been registered for less than three months. The medical records were available for four of the patients registered for 3–12 months and for all the patients registered 12 months or more. Thus, one-quarter (95% CI 14–38%) of non-urgent new referrals to a psychiatric outreach clinic had been registered with their GP for less than three months and in no case were the general practice medical records available.

This finding suggested that any association was as likely to be with the lack of medical records as with new registration. In routine cases it takes 8–12 weeks for the new GP to obtain the medical records and this is, on average, four weeks longer when the patient was originally registered outside the Lothians (Primary Care Medical Records, Lothian Health). Most of the delay is accounted for by the time it takes the previous general practice to send the medical records to the new health board.

Verhaak, P. F. N. (1993) Analysis of referrals of mental health problems by general practitioners. *British Journal of General Practice*, **43**, 203–208.

Wilkinson, G. (1989) Referrals from general practitioners to psychiatrists and paramedical mental health professionals. *British Journal of Psychiatry*, **154**, 72–76.

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Lipid-lowering drugs and mortality

Sir: In their review paper, Boston *et al* (1996) reported that increased mortality with cholesterol lowering has been associated with drugs that do not cross the blood/brain barrier, such as cholestyramine, and those that do, such as statins. However, concern about lipid lowering was prompted by studies of drugs other than statins, namely gemfibrozil and cholestyramine (McLoughlin & Clarke, 1989) and there is evidence that statins are safer. In the West of Scotland Coronary Prevention Study of men with high plasma cholesterol but no history of myocardial infarction, pravastatin reduced the risk of coronary events and the associated deaths without increasing the risk of death from non-cardiovascular causes (Shepherd *et al*, 1995). In a study of myocardial infarction patients who did not have high cholesterol levels, pravastatin reduced non-fatal coronary events with no significant differences in overall mortality or non-cardiovascular mortality (Sacks *et al*, 1996). In

the Scandinavian Simvastatin Survival Study there was a significant reduction in risk of death in the simvastatin group in patients with a previous history of coronary artery disease, an effect apparently independent of baseline serum cholesterol (Scandinavian Simvastatin Survival Study Group, 1995). Further evidence for the safety of statins comes from Wardle *et al* (1996) who found no changes in tension, anxiety, anger, hostility or depression in patients taking simvastatin compared with those on placebo. The beneficial effects of statins may be due to actions other than cholesterol lowering and the lack of effect on non-cardiac mortality, which contrasts with gemfibrozil and cholestyramine, may also be independent of cholesterol lowering.

There is unlikely to be any ethical way in which to study in humans what other biochemical factors may be altered by the different groups of lipid-lowering drugs. Raised cholesterol concentrations in adults are related to indices of impaired growth during late gestation (Barker *et al*, 1993) while suicide has been linked with low weight gain in infancy (Barker *et al*, 1995). Attempts to define biochemical variables associated with violence (including suicide) in adulthood may thus be complicated by an interaction with early nutrition.

Barker, D. J. P., Marty, C. N., Osmond, C., et al (1993) Growth in utero and serum cholesterol concentrations in adult life. *British Medical Journal*, **307**, 1527.

—, **Osmond, C., Rodin, I., et al (1995)** Low weight gain in infancy and suicide in adult life. *British Medical Journal*, **311**, 1203.

Boston, P. F., Dursun, S. M. & Reveley, M. A. (1996) Cholesterol and mental disorder. *British Journal of Psychiatry*, **169**, 682–689.

McLoughlin, I. & Clarke, P. (1989) Lipid-lowering drugs (letter). *British Journal of Psychiatry*, **154**, 275–276.

Sacks, F. M., Pfeffer, M. A., Moye, L. A., et al (1996) The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels. *New England Journal of Medicine*, **335**, 1001–1009.

Scandinavian Simvastatin Survival Study Group (1995) Baseline serum cholesterol and treatment effect in the Scandinavian Simvastatin Survival Study (4S). *Lancet*, **345**, 1274.

Shepherd, J., Cobbe, S. M., Ford, I., et al (1995) Prevention of coronary heart disease with pravastatin in men with hypercholesterolemia. *New England Journal of Medicine*, **333**, 1301–1307.

Wardle, J., Armitage, J., Collins, R., et al (1996) Randomised placebo controlled trial of effect on mood of lowering cholesterol concentration. *British Medical Journal*, **313**, 75–78.

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Perpetrators of child sexual abuse

Sir: We wish to challenge the unreferenced statement by Hilton & Mezey (1996) that the most common form of sexual abuse is father–daughter incest. Data published recently from our community study of adult New Zealand women showed that when 251 women reported child sexual abuse (CSA) of all types occurring when they were under 16 years of age, 22 recalled the perpetrator to be a father or father figure (i.e. stepfather, adoptive father or mother's live-in boyfriend), 75 recalled other relatives (of whom 21 were brothers), 116 acquaintances of the family and 38 strangers unknown to the girl (Romans *et al*, 1996). When accounts of CSA were restricted to those which involved contact with the girl's genitalia, the perpetrators included 22 father figures, 62 other relatives, 31 family acquaintances and 15 strangers. A similar profile was found for all CSA occurring when the victim was aged 12 years and under: the reported perpetrators included 17 fathers/father figures (12 biological fathers and five stepfathers), 69 other male relatives (of whom 16 were brothers) 81 acquaintances of the family and 26 strangers (unpublished data).

We are not the first group studying CSA to report such a pattern of perpetrator identity. Wyatt (1985) reported the following perpetrator percentages among 158 White American women: father/father figure 6%, other relatives 13% (of whom brothers comprised 3%), family acquaintances 30% and strangers 51%. The equivalent figures for the 147 African-American women in that study were father/father figure 10%, other relatives 19% (of whom brothers comprised 3%), family acquaintances 34% and strangers 37%.

All studies, of which we are aware, reporting a random community design, show substantial numbers of non-father relatives and friends or acquaintances of the victim's family to be perpetrators. Reliance cannot be placed on official judicial, health or welfare figures because of the now well-documented low rates of reporting. The large and important perpetrator categories of non-father biological relatives and family acquaintances need to be considered as they usually account numerically for more CSA than father figures, and the CSA they inflict can be intrusive and repeated (Romans *et al*, 1996).

We believe that it is important to correct this common misconception for at least two reasons. It may produce unwarranted scepticism by the clinician when dealing with

patients who talk about CSA by relatives and family friends, and it certainly leads to a misdirection of preventive efforts. In a field so characterised by media distortion, it is important not to add to the misinformation which abounds.

Hilton, M. R. & Mazzy, G. C. (1996) Victims and perpetrators of child sexual abuse. *British Journal of Psychiatry*, **169**, 408–415.

Romans, S. E., Martin, J. L., Anderson, J. C., et al (1996) The 'anatomy' of female child sexual abuse: who does what to young girls? *Australian and New Zealand Journal of Psychiatry*, **30**, 319–325.

Wyatt, G. E. (1985) The sexual abuse of Afro-American and White-American women in childhood. *Child Abuse and Neglect*, **9**, 507–519.

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Urine screening for drugs

Sir: We read with interest the letter by Craig (1996) concerning spurious amphetamine test results in the presence of trazodone. In the National Drug Treatment Centre Board, Ireland, we also routinely screen for amphetamines using EMIT-based Syva assays and have recorded false positives. These false results occur both in the presence of phenothiazines, specifically thioridazine and chlorpromazine, and also with trimipramine.

We have also had difficulty with Syva EMIT benzodiazepine tests with false negative results in the presence of short-acting benzodiazepines such as flunitrazepam. Using the Syva assay up to 10% of samples in this clinic were recorded as negative for benzodiazepine, which on re-testing with a more sensitive technique were detected as positive for benzodiazepine.

The patient group served by the clinic, the majority of whom are opiate addicts, show increasing abuse of substances which they are aware are proving difficult to detect in urine tests. Thus the importance of clinical assessment in cases where amphetamine or flunitrazepam abuse is suspected cannot be underestimated. While urine screening has a role in the treatment of an opiate-addicted population, it must be accepted that current assays are not without limitations.

We would concur with Dr Craig that where possible confirmatory testing is

advisable when using the amphetamine test; however, the most reliable test for confirmation is high-pressure liquid chromatography (Robertson & Drummer, 1995), which is not universally available in the clinical setting.

Craig, R. J. (1996) Urine screening for drugs and trazodone (letter). *British Journal of Psychiatry*, **169**, 669–670.

Robertson, M. D. & Drummer, O. H. (1995) High-performance liquid chromatographic procedure for the measurement of nitrobenzodiazepines and their 7-amino metabolites in blood. *Journal of Chromatography B: Biomedical Applications*, **667**, 179–184.

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Acute clozapine overdosage

Sir: We have observed survival to a high-dose intentional ingestion of clozapine in a young woman. The mortality rate in acute clozapine overdosage is approximately 12% according to the prescribing information provided by the manufacturer, Sandoz. Fatal cases are mostly associated with cardiac insufficiency and aspiration pneumonia and occur with dosages higher than 2 g (Le Blaye *et al*, 1992; Worm *et al*, 1993; Ines *et al*, 1994).

A 34-year-old woman with schizoaffective disorder, depressive type (DSM-IV) intentionally ingested 6–8 g clozapine after nearly three months of clozapine monotherapy (daily dose range 300–400 mg). At admission to the emergency ward one hour later, she presented in a deep coma, with mydriasis, plantar reflexes in flexion and no apparent focal neurological lesions. Blood pressure, heart rate, laboratory data, blood cell counts, ECG and chest X-ray were normal. She was lavaged and standard resuscitation therapeutic procedures were executed; cardiocirculatory and respiratory parameters as well as diuresis were monitored. At 14, 18 and 26 hours from ingestion plasma levels of clozapine and desmethylclozapine were, respectively, 5.7, 3.7 and 2.8 mg/ml and 1.55, 1.2 and 1.06 mg/ml. On the sixth day sudden hypotension with abundant haematemesis appeared, continuing for two days in spite of treatment with somatostatin. The gastroscopic examination showed an erosive haemorrhagic gastritis located in the antrum. After 13 days she was discharged in a good physical condition.

Le Blaye *et al* (1992) reported 150 cases of clozapine overdose; the most frequent symptoms were impaired vigilance (from somnolence to coma) and tachycardia. Major complications were aspiration pneumonia and electrocardiographic changes (but severe arrhythmia rarely occurred). Although cases have been reported of full recovery after ingestion of high doses (Le Blaye *et al*, 1992) and very high plasma levels (>9000 ng/ml) (Ines *et al*, 1994), it has been considered that a dose of 400 mg in a patient not previously treated may be life-threatening and that an overdose of 300 mg in a previously treated patient may result in coma (La Blaye *et al*, 1992). The present case is quite typical in phenomenology: the patient developed a state of deep coma; there were no ECG changes nor cardiac complications. The occurrence after one week of an erosive haemorrhagic gastritis was interpreted as a non-specific, stress-related phenomenon, although it cannot be ruled out that this was in connection with the clozapine overdosage.

The acute toxicity profile of clozapine is similar to that of other neuroleptics, except for extrapyramidal symptoms (Le Blaye *et al*, 1992). There are reports of clozapine-induced delirium reversed with physostigmine (Schuster *et al*, 1977); the present case, however, did not show severe central or peripheral anticholinergic symptoms. It appears, in accordance with the literature (Le Blaye *et al*, 1992), that early treatment of overdose complications is successful under a standard approach, making the case for clozapine as a relatively safe drug.

Ines, S., Mahal, A., Moss, J., et al (1994) Intoxication with clozapine: plasma levels above 9000 ng/ml. Typical clinical picture – diagnostic confusion. *Neuropsychopharmacology*, **10** (suppl. 2), 1225.

Le Blaye, I., Donatini, B., Hall, M., et al (1992) Acute overdosage with clozapine: a review of the available clinical experience. *Pharmaceutical Medicine*, **6**, 169–178.

Schuster, P., Gabriel, E., Kuefferle, B., et al (1977) Reversal by physostigmine of clozapine-induced delirium. *Clinical Toxicology*, **10**, 437–441.

Worm, K., Kringsholm, B., Steentoft, A., et al (1993) Clozapine cases with fatal, toxic or therapeutic concentrations. *International Journal of Legal Medicine*, **106**, 115–118.

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