patients with a lifetime history of problem drinking was no more severe than in control patients. This is in contrast to another study (Dixon *et al*, 1992) where in recently admitted psychotic in-patients alcohol abusers had significantly higher TD scores than non-abusers. The following case report may help to explain these different results.

Case report. A 37-year-old woman with a 16-year history of psychiatric disorder diagnosed latterly as bipolar affective disorder was readmitted to Crichton Royal Hospital on a Sunday evening. She was expressing suicidal ideas, had auditory hallucinations, and had been drinking more heavily than usual for two to three weeks; she admitted to three to four litres of vodka per week. As she had run out of money she had no drink on the Saturday or Sunday. She had been receiving flupenthixol decanoate for three and a half years; the constant dose over the previous 12 months was 150 mg weekly. On the Sunday evening there was no evidence of dyskinesia. The following day there was gross orofacial, neck and upper limb dyskinesia, which if formally rated would certainly have been scored 'severe' on the AIMS global scale (US Department of Health, Education and Welfare, 1976). Gross dyskinesia persisted throughout her stay of eight weeks, during which time there was no evidence of alcohol consumption. The dose of flupenthixol decanoate was reduced to 120 mg weekly. The patient went home on pass for a weekend before discharge. Over the Saturday and Sunday she admitted to consuming over 10 pints of beer. She returned to hospital on the Monday morning; there was no evidence of dyskinesia. Because of disruptive behaviour, she was discharged the same day, but agreed to attend the day hospital. She is again abstaining from alcohol, and dyskinesia has reappeared.

Although Duke *et al* give detailed information, it seems that most of their patients were assessed outside hospital, and therefore had ready access to drink. Continuing alcohol consumption may have masked dyskinesia and contributed to lack of difference in severity of TD between problem drinkers and control patients. This is in contrast to the Dixon study, where patients were not assessed until towards the end of their hospital in-patient stay.

This case report suggests either that alcohol can diminish the severity of dyskinesia produced by antipsychotic medication, or that 'withdrawal' dyskinesia can occur with alcohol as with neuroleptic medication. The latter is perhaps more likely; dyskinesia is common in detoxified alcoholic patients (Lucey & Dinan, 1992).

DIXON, L., WUEIDEN, P. J., HAAS, G., *et al* (1992) Increased tardive dyskinesia in alcohol abusing schizophrenic patients. *Comprehensive Psychiatry*, **33**, 121–122.

LUCEY, J. V. & DINAN, T. G. (1992) Oro-facial dyskinesia and alcohol dependence syndrome. *Psychological Medicine*, **22**, 79–83.

US DEPARTMENT OF HEALTH, EDUCATION AND WELFARE (1976) Abnormal Involuntary Movements Scale In *ECDEU Assessment Manual* (Ed. W. Guy). Rockville: US Department of Health Education and Welfare.

J. HALLIDAY

Department of Clinical Research
Crichton Royal Hospital
Dumfries DG1 4TG

Cannabis consumption and schizophrenia

SIR: The May issue of the *BJP* contains two papers about schizophrenia, and substantial numbers of the patients studied in both were taking psychoactive substances, in one alcohol, and in the other cannabis and other drugs. The basis on which the diagnosis of schizophrenia was made is therefore called into question.

Duke *et al* (**164**, 630–636) identified 352 patients with schizophrenia, and of the 271 who completed the alcohol assessment, 22% were problem drinkers. The 81 patients who did not complete the assessment are likely to have included some problem drinkers, and the remainder will have included substantial drinkers, where the alcohol intake did not amount to a 'problem'. They used Feigner diagnostic criteria (although apparently not exclusively), and one of these is the "absence of alcoholism or drug abuse within one year of onset of psychosis". Although Feigner does not consider this an essential criterion, I believe that for anyone with psychosis taking a psychoactive substance, no diagnosis can be made until after they have been free of the substance for a significant time, in case of alcohol usually about two weeks. This may not be easy to ensure, and the authors should therefore be asked whether the diagnosis was made at a time when the subjects were free of alcohol and should state how they ensured this. Otherwise, the diagnosis of schizophrenia is untenable.

Martinez-Arevalo *et al* (**164**, 679–681) state that patients were included in their study if the diagnosis fulfilled DSM–III criteria, and they report that 30 of the 62 patients regularly consumed cannabis at the beginning of the study, while many others were taking other drugs or alcohol. DSM–III states that, "The diagnosis is made only when it cannot be established that an organic factor initiated and maintained the disturbance". The authors should be asked whether the diagnosis was made at a time when the subjects were free of cannabis and of other psychoactive substances, and should state how they ensured this. Otherwise, the diagnosis of schizophrenia is untenable. The same question could be asked of most of the papers they quote.

Van Praag (1992) cautioned against "a system of diagnosing mainly grounded on symptoms detached from aetiology". The failure to consider aetiology when making a clinical diagnosis is common. In the case of schizophrenia, the error is both common and costly. The cost of the unnecessary treatment must be enormous in drugs, manpower and to the community services, the patients suffer from unnecessary side-effects and often cannot work, and, by doctors purporting to offer 'treatment', they are actually prevented from getting well, since they are not confronted by the fact that their fearsome experiences are self-induced, that they will get well if they stop taking the substance responsible, and that appropriate help is available for them to do this if they wish. As I wrote once before (Cohen, 1992), "The problem is common . . . and perhaps it is time the College tackled it . . ."

COHEN, S. I. (1992) Patients repeatedly admitted to psychiatric wards. *Psychiatric Bulletin*, **16**, 664.

VAN PRAAG, H. (1992) Reconquest of the subjective. *British Journal of Psychiatry*, **160**, 266–271.

SAMUEL I. COHEN

8 Linnell Drive
London NW11 7LT

Cocaine, psychiatric admissions, and HIV

SIR: Gossop *et al* (*BJP*, May 1994, **164**, 660–664) suggest that there may be a relationship between cocaine use and HIV infection. Psychiatric patients have been identified as a potentially high-risk population for HIV infection because of their likely impairments in judgement and an increased exposure risk (Cournos *et al*, 1991).

In Trinidad and Tobago, smoking crack is the predominant mode of cocaine use, with no intravenous use reported. In a two-year review of HIV testing in St Ann's Hospital, the sole psychiatric hospital there (Infection Control Unit, 1993), it was found 6.8% of those tested were HIV positive (53 of 782). Of the HIV-positive patients, however, 31 (58.5%) were admitted with cocaine-related problems. This suggests that cocaine use in psychiatric admissions may further increase the risk for HIV infection.

With crack cocaine use increasing in the UK, particularly among the Caribbean population, there is some cause for concern, and preventive strategies for both cocaine abuse and HIV infection should be developed for this group.

COURNOS, F., EMPFIELD, M., HOWARTH, E., *et al* (1991) Seroprevalence among patients admitted to two psychiatric hospitals. *American Journal of Psychiatry*, **148**, 11225–11230.

INFECTION CONTROL UNIT (1993) *HIV Testing in St Ann's Hospital. Psychiatric Update*. Trinidad: St Ann's Hospital.

GERRARD HUTCHINSON

Bethlem Royal Hospital
Beckenham
Kent BR3 3BX

SIR: I have worked at the Substance Abuse Prevention and Treatment Centre (SAPTC) in Trinidad, West Indies (population 1.2 million). This facility offers patients using cocaine a six-week in-patient treatment programme.

I reviewed the notes of new admissions for 1986 and 1987. There were 125 admissions in 1986 and 127 admissions in 1987 which met the DSM–III–R criteria (American Psychiatric Association, 1987) for cocaine dependence. All of them listed smoking as their most recent mode of intake, and while a few had used the intranasal route, virtually none had ever injected. (There has never been a culture of intravenous drug use in Trinidad.)

It is impossible to make direct comparisons with the findings of Gossop *et al*, given what is effectively a single route of administration in a cohort presenting to a specialised treatment facility. However, although Gossop *et al* report low dependency in their sample of crack smokers in the community, the SAPTC experience does suggest that using crack cocaine by smoking alone can result in severe dependence. Indeed, in each year there were many patients who were seen but not admitted simply because of a lack of beds, and the figures above thus underestimate the dependency problem.

Cocaine use in Trinidad was not an issue until about the 1980s when a ready supply of cocaine became available, and the problem has since mushroomed. Whatever the pattern of use in the community in the UK now, with the targeting of Europe by suppliers of crack cocaine as reported in the press and increasing availability, there is likely to be the emergence of a large group with severe problems of dependence. A major issue will be the nature of services offered to them.

AMERICAN PSYCHIATRIC ASSOCIATION (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM–III–R). Washington, DC: APA.

D.A. RATAN

Leicestershire Mental Health Service NHS Trust
Psychiatric Unit
Leicester LE5 4PW

Cost-effectiveness of antidepressants

SIR: Jönsson & Bebbington (*BJP*, May 1994, **164**, 665–673) calculate that total costs per patient for