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Correspondence

Contents: Substance-induced psychosis/Public attitudes to mental illness/SSRI and sympathomimetic interaction/'Psychopaths' in special hospitals/Use of vecuronium to prevent suxamethonium-induced myalgia after ECT/HIV and mental illness/Biological psychiatry and reductionism/Antipsychotic drug-induced dysphoria/Bias towards chronicity in schizophrenia?/Accepting voices

Substance-induced psychosis

SIR: Poole & Brabbins (1996) draw attention to an area of psychiatry where our conceptual confusion may have serious consequences for patients. They are right to assert that drug use should not be uncritically assumed to be the cause of associated psychoses. Nevertheless, their scepticism about the existence of "true drug-induced psychosis" risks throwing out the baby with the bathwater.

It is our clinical impression that the behavioural profile of "schizophrenia" has changed over the years. We do not recall having to manage so many restless, impulsive, combative and dangerously violent psychotic patients in a setting of serious and often sociopathic personality disorder, as are presenting today. Indeed, we used to feel confident in teaching our students and assuring the public that violence was rare among sufferers from schizophrenia. Difficulty in containing these patients in acute general psychiatric units leads to many having to be accommodated in secure units, special hospitals and, increasingly, private hospitals specialising in their care. The cost to the NHS must be enormous. Relative resistance to anti-psychotic drugs is frequently observed in these patients and unusually high doses have often to be used to bring under control crises dangerous for the patients as well as the staff and the community. This, of course, carries its own dangers. Discussion with many colleagues confirms our impression that, although these patients display many of the positive symptoms of schizophrenia there is a paucity of negative ones and they rarely progress to the characteristic schizophrenic defect state, despite many relapses. This itself suggests a possibly different aetiology.

The genetic and other factors, including substance abuse, in causation of this complex disorder probably differ in *certain significant respects* from the causes of the schizophrenic illness originally observed, studied and defined by Kraepelin, Bleuler and Schneider. Smith & Hucker (1994) emphasise that toxicological screening alone may give misleading results and they believe that the frequency of substance abuse in "schizophrenic" patients is under-estimated. In our view, the role of substance abuse in the apparently changing clinical picture of "schizophrenia" warrants further systematic enquiry. Despite the grave problems they cause the cases we described are a minority. Schizophrenia may be getting a bad name undeservedly.

POOLE, R. & BRABBINS, C. (1996) Drug induced psychosis. *British Journal of Psychiatry,* **168,** 135–138.
SMITH, J. & HUCKER, S. (1994) Schizophrenia and substance abuse. *British Journal of Psychiatry,* **165,** 13–21.

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SIR: In their editorial, Poole & Brabbins (1996) rightly say that psychiatrists lack clarity in their understanding of the relationship between psychosis and drug use, that the literature is extensive but flawed, the published studies rarely related mental state to toxicological findings and that there are several "obstructions to clarity" in this field.

The main obstruction to clarity is widespread failure to consider the point which is central to the whole subject, namely that in making a differential diagnosis of psychotic disorder in any person who is taking or may have taken drugs, there is no way of making a diagnosis of, say, schizophrenia or mania until after the patient has been free of drugs for 1 to 2 weeks, occasionally longer. Until then, the assumption must be that the drugs may be the cause of the psychosis (Cohen, 1995). With few exceptions – such as the review by Smith & Hucker (1994) – failure to consider this is evident throughout the literature and