used by Dr Davies is 0.5%. This is the currently estimated risk of seroconversion after a needlestick injury of HIV positive blood – this point requires emphasis as it leads directly on to the need to have an estimate of population prevalence of HIV.

Secondly, the best available population estimate of HIV prevalence in the UK is derived from voluntary blood donation screens. The UK prevalence derived from this source is 0.0016% (British Medical Journal, 1988). Using a simple probability calculation, the risk of seroconversion after needlestick injury with blood of unknown HIV status is 1 in 12.5 million. We can build assumptions into this calculation, e.g. that the prevalence of HIV in a psychiatric hospital population is, say, 100 times that in the general population; the risk from a single random needlestick injury then becomes 1 in 125 000. I do not 'dismiss' this risk, but attempt to view it in relation to, for example, the 1 in 1000 risk of a child dying before its first birthday (Office of Population Censuses and Surveys, 1986*a*) the 2 in 1000 risk of a man aged 45–54 dying of a coronary heart disease (Office of Population Censuses and Surveys, 1986*b*) and the 6–30% risk of hepatitis B seroconversion after needlestick injury with infected blood (*Population Reports,* 1986). Furthermore, using a simple binomial model it would require in excess of 85 000 events to produce a greater than 50% probability of at least one seroconversion.

I shall not follow Dr Davies' practice of confusing terms whose meanings are widely held to be different. Screening is not the same as assessment, and certainly not the same as "assessment" under the 1983 Mental Health Act. It is a pity that Dr Davies has not assimilated the cogent arguments by Dickens (1988) on the legal rights and duties of health professionals; this is surprising, as Dr Davies himself cited Dickens' article. Of equal importance are the ethical arguments for and against involuntary screening. Walters (1988), in a reasoned and eloquent article, concluded: "Mandatory screening programmes other than those involving persons who voluntarily donate blood, semen, or organs are not morally justifiable at this time". Taken together, these papers present the case for a voluntary screening programme and emphasise the essentially voluntary relationship which ought to exist between doctor and patient.

Finally, it is precisely because Dr Davies and I hold genuinely different opinions about the best approach to the problems presented by HIV infection that I cannot join with him in a trivialising of the debate – summed up in his recycled phrase "where will all this nonsense end?" In particular, there is a pressing need for anonymous screening for HIV so that better population prevalence figures are available for monitoring trends and planning services – informed debate on this and other relevant issues are not nonsense. Such debate is, in fact, an essential part of developing valid and acceptable practice and policy responses to the greatest health risk of our time.

J. B. CONNELLY

Department of Community Medicine
St Mary's Hospital Medical School
London W2

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Childhood and adolescent depression

SIR: I found the articles by Angold (*Journal,* May 1988, **152,** 601–617; *Journal,* October 1988, **153,** 476–492) on childhood and adolescent depression to be thoughtful and informative. However, I was somewhat surprised to see that the only reference to my work in this area was to misquote the rating scale that I developed while in Edinburgh. The scale has no reference to "wandering behaviour", although this term was included on a list of variables taken to form an operational definition for depression in childhood. As it turned out, the current RDC criteria and my operational definition are remarkably similar.

One of the major points made by Dr Angold is the importance of taking the history directly from the child, i.e. that children are generally reliable informants if they can get some help in putting their situation and feelings into words. Mood self-rating scales for children seem to be quite useful for this purpose.

Another of his important conclusions is to be careful in investigating mood phenomena in children who present with conduct disorders or who have serious psychosocial difficulties.

My work (Birleson, 1981; Birleson *et al,* 1987) would strongly support these assertions if quoted correctly.

PETER BIRLESON

Royal Children's Hospital
Flemington Road
Parkville, Victoria 3052
Australia

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Positive symptoms of schizophrenia

SIR: Frith & Done (*Journal*, October 1988, **153**, 437–443) propose that the positive symptoms of schizophrenia arise from a failure in transmission, from goal-setting areas to a central monitor, of willed intentions that form the basis of self-generated action. They suggest that when self-generated actions are noted by the monitor, but not understood as such because of failure of advanced warning of willed intention, then these actions are attributed to external events.

Such a mechanism could underlie the "permeability of the ego-world boundary" mentioned by Schneider as possibly causing passivity phenomena (Koehler, 1979). However, there is another implication of this hypothesis which is more difficult to place: to lose awareness of willed intention to an extent sufficient to cause a florid positive schizophrenia may be to lose recognition of oneself as an independent thinking being. If the central elements of a singular identity are retained in the absence of awareness of the self-generated nature of activity, then Descartes' phrase, "I think therefore I am", would need to be restated as, "I respond therefore I am". This is not a correct statement, since the ability simply to respond does not require an individual sense of consciousness.

Jaspers (1959) considered the effect of psychopathology on awareness of existence, and concluded that there are circumstances where *cognito ergo sum* is no longer a valid experience, particularly in the presence of derealisation and depersonalisation.

Such observations lead to the prediction that the more severe are the positive symptoms expressed by a schizophrenic patient, the more likely it becomes that the patient will be experiencing severe symptoms of depersonalisation or derealisation. This is generally not the case. One explanation for this would be that only the transmission of willed intentions relating to selected goals is impaired. This would account for the observation in many cases, particularly of paranoid schizophrenia, that positive symptoms are only experienced in a part of the patient's experience as a whole. The question then becomes: why is there an abnormality in this particular area of self-generated behaviour?

HOWARD RING

The Maudsley Hospital
Denmark Hill, London SE5

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SIR: Frith & Done (*Journal*, October 1988, **153**, 437–443) draw attention to the lack of a psychological theory for the positive symptoms of schizophrenia. They propose what appears to be a simplistic model to explain the phenomena of auditory hallucinations. They believe that the patient is "talking to himself" but believes the voices he is hearing are from an outside source.

What they do not consider is the form or content of auditory hallucinations commonly seen in schizophrenia. For example, how would this theory explain two voices discussing the patient, one of which may be male, the other female? This would certainly not reflect "normal psychological processes", whether or not it was labelled "my own". Similarly, the content of auditory hallucinations in paranoid schizophrenia is often abusive and derisory; our understanding of this is not advanced by the theory.

It is admirable that experimental tests of monitor failure are possible, but surely the theory must first embrace those symptoms commonly seen in clinical practice.

BRIAN R. TIMNEY

Adolescent Unit
Northern General Hospital
Sheffield S5 7AU

Sample Size and CT Scans in Schizophrenia

SIR: Smith *et al* (*Journal*, November 1988, **153**, 667–674) remind us that the use of high-tech research instruments such as computerised tomography (CT) often hides basic methodological flaws, such as the choice of bogus control groups. Ironically, however, the results of the authors' own elegant meta-analysis of published CT studies in schizophrenia contain the seeds for criticism of their own study: the use of sample sizes too small to test hypotheses is another much-perpetrated sin. The figures derived by Smith *et al* from previous studies show that lateral ventricular size (as measured by VBR) in schizophrenic subjects exceeds that of healthy controls by about 30%. Entering these figures and an approximation of the authors' value for overall standard deviation (± 3.1) into a power analysis shows that, in order to be even 80% confident that a two-tailed test will produce a statistically significant difference, at least 60 patients and 60 controls are needed. It is not surprising,

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