been seen to acknowledge the possibility that raters can make inferences about one another's rating behaviour by noting when pen moves to paper. Placing a screen between raters has been shown to result in a fall in the very high agreement about symptoms in joint interviews to the level for independent interviews (Robinson *et al*, 1982), which raises the intriguing possibility that this very high agreement reported here and elsewhere, for example in the International Pilot Study of Schizophrenia (World Health Organization, 1973), has at least two components: one a measure of agreement about symptoms by psychiatrists, the other a measure of sensitivity between psychiatrists.

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Folate, Vitamin B_{12} and Posture

SIR: Abou-Saleh & Chung-A-On (*Journal*, January 1987, **150**, 133) have reported a study of vitamin B_{12} and folate plasma levels.

We observed in 23 patients who had been lying down for at least 6 hours, that standing for 30–40 minutes increased vitamin B_{12} and folate plasma levels by more than 10%. Red cell folate levels decreased non-significantly by an average of approximately 2%.

The explanation of our observations is the following: as soon as the subject stands, fluid (up to 23% of plasma volume) leaves the circulation under the influence of hydrostatic forces, particularly in the lower limbs. As a result, there is an increase in concentrations of blood constituents such as red cells, proteins (Fawcett & Wynn, 1960; Hagan *et al*, 1978), and protein-bound substances such as calcium (Husdan *et al*, 1973), folate, and vitamin B_{12}, which do not readily pass through the capillary membrane. Hagan *et al* (1978) have shown that standing for 35 minutes increases venous haematocrit, haemoglobin, and plasma protein levels by 10%, 10%, and 20% respectively.

Our observations underline the need to control posture and other factors which may change plasma volume (Husdan *et al*, 1973; Abalan *et al*, 1987) in folate and vitamin B_{12} plasma level studies as in studies of other protein-bound vitamins.

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Transient Effect of Diazepam in Some Sub-acute Organic States

SIR: Aynsworth's report (*Journal*, January 1987, **150**, 110–112) of a patient with a catatonic state possibly related to viral encephalitis prompts us to comment on her finding in that patient of a brief return to apparent normality after intravenous diazepam. We note that she quotes previous reports in which intravenous barbiturate resulted in improvement in organic catatonic states.

Case report: A 19-year-old woman suffered severe hypoxic brain damage due to drug overdose, with a severe defect in short-term memory and attention span and a trans-cortical aphasia. At times she was mute or nearly mute. Grimacing and posturing was accompanied by disinhibited behaviour and apathy. For several months she refused to eat. Intravenous sodium amytal produced a striking temporary

improvement in her awareness, attention, speech, and interpersonal relationships. Oral diazepam resulted in a recordable improvement in her behaviour and level of activity over several weeks, and in a longitudinal cross-over study with placebo over several months the improvement was deemed attributable to the diazepam. Intermediate memory (recording and recall of verbally and visually represented material) improved slightly with diazepam and amytal.

We do not understand the mechanism of this finding in our patient or in Aynsworth's patient. The fact that improvement occurs in catatonic states with barbiturate or diazepam infusion should not necessarily lead clinicians to suspect hysterical mechanisms. In the female patient we report, the benefits of diazepam gradually wore off over six months. Although environmental changes have resulted in further improvements in her condition she remains in sheltered accommodation.

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Similar Incidence Worldwide of Schizophrenia: Case not Proven

SIR: The article by Sartorius *et al* (*Psychological Medicine*, November 1986, **16**, 909–928) is a summation of a prodigious amount of work and contains invaluable data. The two important conclusions reached, however, must be examined closely in the light of the diagnostic criteria and population cohorts which were used.

Firstly, we consider the significance of the "central" (by which they mean 'nuclear', Schneiderian) schizophrenia syndrome that the authors state occurs with approximately equal frequency worldwide. To study incidence data, the authors include all individuals in a given catchment area who made a first lifetime contact with any helping agency, hospital, clinic, or religious or traditional healer over the one-year collection period of the study with a disorder that met either broad (ICD–9 or CATEGO S, P, O) or narrowly defined (CATEGO S+, Schneiderian, nuclear) criteria for schizophrenia. The problems with these definitions are well known: neither the ICD–9 definition nor the Present State Examination (PSE)-based CATEGO classification requires a minimum duration of symptoms for diagnosis of schizophrenia. This appears to have led to the considerable diversity of the schizophrenic samples collected in participating countries, as illustrated by the large differences in age-specific incidence rates in them. For example, in their Table 6 it is evident that the percentage of first-contact schizophrenic patients in the 15–24 age group in Cali (70.8%) is more than three times that in Moscow (21.8%). Among 25–34 year olds, Ibadan (42.3%) has twice the percentage of Cali (21.4%). For 35–44 year olds, the range is even wider: 3.6% of the sample of first contact schizophrenics in Rochester falls in this age-group, compared with 6.5% in Agra, 19.4% in Dublin, and 22.3% in Moscow. Finally, Moscow reports that 20.4% of their total sample of first-contact schizophrenic patients fell in the oldest age-group (45–54 years), while only 1.2% of first-contact schizophrenic patients in Agra and 3% of the Cali sample fell in this range – a nearly 20-fold difference.

With respect to the clinical syndrome, again the differences are as striking as the similarities: while only 26% of patients in developed countries had acute onset, 49% had such onsets in developing countries. A diagnosis of acute schizophrenic episode was made in 40% of patients in less developed countries, but only 10% in developed countries, and the catatonic subtype was 10 times more common in developing countries. Indeed, inspection of their Fig. 2 indicates that in the developing countries more than 30% of all patients in the survey had been ill less than one month and 50% for only two months! While the differences in clinical presentation, such as more violence and excitement in developing countries and more paranoia and depression from developed lands, might be attributed to cultural differences, the great differences in mode of onset and age distribution between centres indicates that the schizophrenia defined by the authors presents great differences between the places studied. Indeed, what would be designated by RDC or DSM–III criteria as *acute reactive*, *brief* or *schizophreniform* psychosis is characteristic of a majority of admissions to psychiatric facilities in the developing countries. If diagnosed as schizophrenia, this may lead to the false conclusion that the same disorder has an equal prevalence around the world. Clearly, inclusion of this large number of acute psychoses of less than two months' duration, predominantly from the developing countries, will also bias the statistics by indicating a more favourable course for schizophrenia in developing countries. Inclusion of alcohol and drug-related psychoses by the authors will further blur the data, and may account in part for the large differences in age of first-contact schizophrenic patients between the centres.

The incidence data, claimed to show a similarity in all populations studied, are flawed in two respects. Firstly, only 8 of the 13 areas studied had sufficient