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Standardised Assessment of Personality Disorder in Mental Handicap

SIR: We previously reported on the reliability of Mann's standardised assessment of personality disorder in mental handicap (Mann *et al*, 1981; Ballinger & Reid, 1987), and we subsequently described a survey of 100 patients in a mental handicap hospital, using this scale (Reid & Ballinger, 1987). We were interested to see if the presence of personality disorder had a predictive value, and we reviewed the placement of the patients one year later. In the year after assessment, 25 of the 100 patients had been discharged, mainly to hostels. Of the 44 patients with no personality problems, 11 (25%) had been discharged; of the 34 with mild traits only (Grade I), 13 (38%) had been discharged; and of the 22 patients with definite personality disorder (Grade 2), only 1 (4.6%) had left hospital. Thus, patients with personality disorder were less likely to be discharged ($\chi^2 = 8.08$, d.f. $= 2$, $P < 0.05$), suggesting that personality disorder detected by this method of assessment was of value in predicting likely discharge.

BRIAN R. BALLINGER
ANDREW H. REID

Royal Dundee Liff Hospital
Dundee DD2 5NF

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Behaviourally Disturbed HIV Patients

SIR: Much has been written about the deleterious effects on patients of the rundown and closure of our large psychiatric hospitals. It is ironic that this process is accelerating at a time when we are facing a new and worrying group of disorders consequent on the Human Immunodeficiency Virus (HIV). The nosology of HIV-related psychiatric disorder is still poorly understood, and in planning services for such patients we are still highly dependent on educated guesses as to the scale and nature of the problem. Perhaps the main cause for concern is that group of patients who show disturbed behaviour requiring in-patient management.

A number of patterns of psychopathology may be discerned which result in disturbed behaviour: (a) disinhibition in early HIV encephalopathy leading to amplification of premorbid sociopathic traits; (b) more profound dementia resulting in the release of primitive behaviours as seen in other types of dementia; (c) functional psychosis in which the delusions and hallucinations produce fear and aggression; and (d) anger and resentment in patients who perceive themselves as having nothing to lose. This, in particular, may produce an urge to transmit the virus to others. In all of these cases there is a definite and significant risk of infection to those in contact with them.

Such patients will clearly require management in conditions of greater security than is available on most acute admission wards, and this will almost certainly mean detention under the Mental Health Act.

It would, in my opinion, be improper to expose other, HIV-free, detained patients to the risk of infection with this lethal agent, and hence we must be thinking in terms of specialised units. In the 'old days' it would have been a relatively simple matter to refurbish a ward in a psychiatric hospital to cater for the security needs, and with the large pool of nursing staff available, great flexibility and rapid response to ward requirements would be possible. In the new district general hospital units, the problems are much greater. If specialised units are to be available for disturbed HIV carriers, new buildings and staff will be required which means new money, and in considerable quantities. We simply do not know enough about the scale of the problem and its likely development to estimate the number of beds and staffing levels required and, to a considerable extent, one gets the impression that this problem is being tacitly ignored by planners.

In Plymouth, we are attempting to address the problem of HIV-positive behaviourally disturbed patients. We know of three definite cases of HIV encephalopathy, and the 'guesstimate' is that this will rise to 40 or more in a couple of years. We have no idea what proportion of these will require secure provision (one of the three known to us might well have benefited from this had it been available) and for how long they will need it. I would be most interested and grateful to hear from anyone who is involved in planning services for HIV patients in their district or

region and to swap ideas on this rather ill-defined yet important problem.

D. R. DAVIES

Moorhaven Hospital
Bittaford
Ivybridge
South Devon PL21 0EX

The Pathogenesis of Depersonalisation: A Hypothesis

SIR: Depersonalisation/derealisation are common symptoms of anxiety and of affective disorder, and are not uncommon features of other psychiatric syndromes. They are also common in various organic states (e.g. hypoglycaemia) and are frequently likened by patients to dreaming, which occurs at a time of demonstrable alteration in cerebral activity. Furthermore, these phenomena are disorders of perception, which suggests some organic basis. Is it possible that depersonalisation/derealisation are *always* manifestations of demonstrable organic abnormality in the brain?

Hyperventilation, too, is a common symptom of anxiety, and depersonalisation is common in hyperventilation. Anxiety is frequent in most psychiatric syndromes, and it might be reasonable to suppose that whatever the primary psychiatric diagnosis, it is only the patient who is anxious and hyperventilating who develops depersonalisation. In such patients the depersonalisation would be the result of the changes in metabolism and cerebral blood flow produced by the hyperventilation. The hypothesis would be that only those patients who were over-breathing would be depersonalised, whilst those patients who were not over-breathing would not suffer depersonalisation. At the same time the proviso has to be made that not all patients who are over-breathing would necessarily experience depersonalisation, as there might be some individual variation in the propensity to develop this symptom.

Having made this hypothesis, I set out to test it. As a first step I began to look for patients with depersonalisation who were not over-breathing, with a view to comparing various measures in them with patients who were over-breathing. It has, however, proved to be increasingly difficult, if not impossible, to find such patients who were not over-breathing. I think this may be because, since I have become aware of the hypothesis, I am not overlooking hyperventilation in such patients, whereas previously I might have been. Thus the investigation might not turn out to be as easy as it seemed at first, not an unfamiliar situation. In the meantime, however, the idea appears to have spread in this hospital and I have found over the past few months or more that it has been quoted to me as 'received knowledge' in teaching sessions with undergraduates and with junior doctors. For this reason I seek the hospitality of your columns to go on record as its originator.

SAMUEL I. COHEN

The London Hospital Medical College
Turner Street
London E1 2AD

'Neuroleptic Malignant Syndrome' Without Neuroleptics

SIR: In support of the suggestion by Singh & Maguire (*Journal*, December 1987, **151**, 863) that the term neuroleptic malignant syndrome (NMS) should be revised, we report a fulminating case, exhibiting all of the diagnostic criteria proposed by Levenson (1985) but which occurred when lithium and phenelzine were employed in therapeutic doses. The patient had never taken neuroleptic drugs.

Case report: A 42-year-old woman presented to casualty with a rapid onset of restlessness, sweating, and confusion. She had a history of depression with intermittent agitation and some phobic symptoms of several years duration. Her medication comprised the following: phenelzine (15 mg three times daily), lithium carbonate (800 mg daily), L-tryptophan (1 g daily), diazepam (2 mg three times daily), and triazolam (0.25 mg daily). Phenelzine had been commenced six weeks previously, replacing clomipramine which had proved ineffective over four months. Relatives believed that the patient took her medications only as prescribed.

Within three hours she was comatose. Pupillary and corneal reflexes were lost. Trunk and limbs were hypertonic and held in rigid hyperextension. Tendon reflexes were brisk, but plantar responses were flexor. Temperature rose from 38.5°C on admission to 42.5°C four hours later. She had a tachycardia and became hypotensive.

A diagnosis of NMS was made and she was treated with intravenous dantrolene (60 mg three times daily), commenced within four hours. Body temperature returned to normal within 14 h; blood pressure and heart rate were controlled with dopamine and practolol.

Investigation showed mild leucocytosis and initially normal biochemical parameters of hepatic, renal, and muscle function. Serum creatine phosphokinase became elevated, reaching a peak of 41 355 U/l (normal values 24–175) on the third day. Cerebrospinal fluid was normal. Blood and urine cultures were negative. Intravenous benzylpenicillin and gentamicin were commenced before results of these became available.

Severe disseminated intravascular coagulation occurred after 12 h. Acute renal failure and continuing infusion of blood products necessitated treatment by peritoneal dialysis. Mechanical ventilation was instituted. By day five, elevated transaminases and alkaline phosphatase indicated severe hepatocellular damage. Profound hypoglycaemia