Catatonia: the tension insanity

SIR: Johnson in his lecture article (*Journal*, June 1993, **162,** 733–738) suggests that catatonia should be regarded as a neuropsychiatric syndrome in which an abnormal mental state is associated with 'cataleptic phenomena'. Cataleptic phenomena, as used by Johnson, essentially refer to features of catatonic stupor, with immobility, maintenance of posture and mutism constituting a diagnostic triad. Such a view of catatonia would virtually exclude those 'catatonic' conditions in which only the excited state is present.

Catatonia is generally regarded as having two phases – retardation (stupor) and excitement – with sometimes rapid alternation between the two. Although less common, the excited form may be the only state present. Morrison (1973) studied 250 cases of catatonia and found that 110 were predominantly retarded, 67 predominantly excited, and the remainder in a mixed state. It is interesting to note that the three-state concept of catatonia – retarded, excited and mixed – is incorporated into the ICD–10 diagnostic guidelines (World Health Organization, 1992) for a new category: organic catatonic disorder.

Morrison's study also showed that retarded and excited patients differed not only in obvious presentation but also in course and prognosis. Retarded patients were significantly more often negativistic, mute, rigid and cataleptic – features corresponding to Johnson's cataleptic phenomena – whereas excited patients were more frequently impulsive, combative and denudative. Excited patients were also characterised by an abrupt, rapid onset, were more likely to be greatly improved at discharge, and were more often recovered at follow-up. Moreover, they were more frequently diagnosable as having affective disorder, mostly mania.

Nevertheless, considerable symptom overlap was also noted between the two subtypes. No one symptom was limited to one type, and of those special symptoms supposedly 'typical' of catatonia, only four – mutism, negativism, posturing and staring into space – were present in half or more of the total patients. Such symptom overlap, coupled with the fact that the two states may co-exist or alternate rapidly, would argue against separating them as two distinct syndromes. They are best considered distinct subtypes subsumed under the catatonic syndrome.

The past 20 years have seen no comparable large-scale symptomatology studies. Research attention to the two subtypes of catatonia has been scanty. No distinction is often made between the two states, or interest is concentrated only on the retarded form. There have been a number of reports in recent years on the use of drugs such as diazepam (McEvoy & Lohr, 1984), lorazepam (Salam *et al*, 1987) and carbamazepine (Rankel & Rankel, 1988) in the treatment of catatonia (psychogenic- or neuroleptic-induced), but they are all for the stuporous state. A re-evaluation of the excited form of the syndrome as a catatonic subtype is indicated. To remove it from the catatonic syndrome at the present stage would appear unjustified.

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WAI-YU JOSEPH

Tokanui Hospital
Private Bag
Te Awamutu
New Zealand

Low serum cholesterol and suicide

SIR: Hawton & Cowen (*Journal*, June 1993, **162,** 818–825) consider the suggestion that the link between low cholesterol and suicide is secondary to the reduced appetite and weight loss which may occur with depressive illness. In an attempt to clarify the links between cholesterol and depression, we studied six depressed men over the age of 40 with no history of ischaemic heart disease (IHD), before treatment and on recovery, and six age-matched male controls for whom blood was obtained at the same interval.

Depression is associated with an increased risk of death from IHD (Rabins *et al*, 1985) and one might therefore expect that patients with depressive illness would have high plasma cholesterol, high low-density lipoprotein cholesterol (LDL-C), low high-density lipoprotein cholesterol (HDL-C), or altered HDL subfraction distribution (Griffin *et al*, 1988).

In our study, there was a non-significant fall in total plasma cholesterol in the depressed group on recovery (mean $\pm$ s.d.: 203.5 ± 34.9 v. 175.4 ± 25.9 mg/100 ml) and there was a significant fall in LDL-C in recovered depressive patients (mean $\pm$ s.d.: 102.1 ± 18.1 v. 77.4 ± 17.6 mg/100 ml; $P = 0.036$). There is thus some support for the hypothesis that recovery from depression is associated with changes in circulating cholesterol concentrations, though whether these would be independent of, or central to the changes which result in clinical recovery remains to be established.

Lowering of cholesterol to reduce the rate of IHD has been associated with increased deaths from suicide and accidents (Engelberg, 1992). Elliott (*Journal*, June 1993, **162,** 818–825) suggests that the overall result of diminished brain cholesterol would be reduced 5-HT (serotonin) activity by increased pre-synaptic 5-HT uptake or decreased post-synaptic transmission.

A lowering of total plasma cholesterol on recovery from depression might therefore inhibit the facilitation of 5-HT transmission generally considered to be the result of antidepressant treatment, if it were mirrored in the brain, but the cholesterol content of HDL may be more important for brain function. In our study there was no significant change in HDL on recovery (mean ± s.d.: 39.9 ± 10.3 v. 38.5 ± 10.1%).

The activity of the 5-HT pump is increased by low cholesterol, as is Na/K ATPase activity (Papahajopoulos *et al*, 1973). Red-cell Na/K ATPase is known to be low in depressed patients, and to increase on recovery (Naylor *et al*, 1980). Further studies of cholesterol levels in depression with concomitant observation of 5-HT receptor function, optimally by provocation tests, would be of value in clarifying the relationships of cholesterol to 5-HT activity, Na/K ATPase activity, and the risk of IHD in depressive illness.

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*F. COULTER

Dykebar Hospital
Grahamston Road
Paisley PA2 7DE

F. M. CORRIGAN

Argyll and Bute Hospital
Argyll

B. MOWAT
E. R. SKINNER

Marischall College
Aberdeen

*Correspondence

Eye-movement desensitisation to overcome post-traumatic stress disorder

SIR: I read with considerable interest the article by Spector & Huthwaite (*Journal*, July 1993, **163,** 106–108) because over the past two years or so I have been attempting to use this procedure in my clinical work. Unfortunately, examples of post-traumatic stress disorder usually of a severe nature are all too prevalent in the area where I practise. In general I follow the procedure outlined by Shapiro (1989) but would tend to give more than the 20 saccadic movements in each set.

Initially I experienced some resistance from the patients, so now I would generally give them a short discussion on the association between post-traumatic stress disorder and rapid-eye movement sleep as outlined in the paper by Ross *et al* (1989). It is a time-consuming procedure both for the patient and the therapist, but in my opinion it can form a valuable part in the treatment of a potentially crippling condition.

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J. B. WALSH

South Tyrone Hospital
Carland Road
Dungannon
County Tyrone BT71 4AU

How well are 'cured' anorexic nervosa patients?

SIR: Windauer *et al* (*Journal*, August 1993, **163,** 195–200) rightly stated that there was little support for my earlier contention (Hsu, 1988) that "for those who recovered from anorexia nervosa, normal weight bulimia nervosa (not major depression) is the most common diagnosis". The preliminary data from our own long-term follow-up study also indicated that eating disorder not otherwise specified (ED NOS) is the most common diagnosis, not bulimia nervosa (Hsu *et al*, 1992). From the data presented by Windauer *et al*, I would think that ED NOS is also the most common diagnosis among those who no longer meet criteria for anorexia nervosa.

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