

## Correspondence

Editor: Ian Pullen

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### Personality and outcome of depression

SIR: Andrews *et al* (*Journal*, July 1990, 157, 13–18) present a very interesting study of personality and long-term outcome in depression, but there are alternative explanations to the data presented.

The authors follow the tradition of regarding endogenous and neurotic depression as a dichotomy. Their study shows the negative influence of personality on the outcome of neurotic depression (ND) but not endogenous depression (ED). Andreasen has indicated factors which support a dichotomy view (Andreasen, 1980). The findings by Professor Andrews *et al* exemplify one of these.

The paper also suggests that endogenous and neurotic depression have different courses with 'episodic' or 'more insidious' features respectively. The data presented is open to different interpretation. A total of 57% of their ON group (other neurosis presenting as depression) have drifted into ND by the end of the study. This suggests a high degree of overlap between the two groups. It is therefore just as reasonable to sum the two groups together as to make a possibly artificial division between them. Doing this the combined ND + ON group may then be compared with the ED group. In the combined group ( $n = 127$ ), 16% remained well, 71% suffered a recurrence of some degree and 18% were incapacitated during the follow-up period. In the ED group ( $n = 66$ ) these figures are 15%, 60%, and 16% respectively. These findings seem more remarkable for their similarities than their differences. From a clinical perspective both ED and ND appear to have similar courses.

The authors also suggest that the characteristics of ND more closely resemble ON than ED. In view of the diagnostic drift from ON to ND this is perhaps not surprising.

In the view of the Newcastle group which Professor Andrews *et al* favour, a personality inadequacy is one factor defining neurotic rather than endogenous depression (Carney *et al*, 1965). This study demonstrates that in depression, where personality factors are initially an aetiological factor, they remain a factor influencing the future recurrence of illness. Where personality factors are less important, in an initial endogenous diagnosis, they remain less of an influence in the outcome of the illness.

Overall, the courses of ND and ED appear only a little different in outcome. What differences there are can be explained by the modifying effects of personality. The case for a dichotomy is not proven.

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**AUTHORS' REPLY:** We have published two papers on the long-term outcome of our cohort of depressive patients (*Journal*, June 1988, 153, 752–757 and *Journal*, July 1990, 157, 13–18). Your correspondent appears to have overlooked the former paper in which we report that the morbidity at 15 years was the same for patients with endogenous or neurotic depression. We thank your correspondent for restating what we have already published and indeed stated again in the abstract of the second paper: "diagnosis at index admission did not predict overall outcome" (1990, p.13). We noted that despite the similarity in outcome, "patients with endogenous depression, an apparently stable diagnosis, had longer index admissions, were readmitted sooner, but

spent less time ill than patients in either of the neurosis groups" (1990, p.13). Personality abnormality as judged at index admission accounted for 20% of the variation in these measures in the neurotic but not in the endogenous group.

We have long argued that attempting to establish the independence of two syndromes on the basis of symptoms alone is foolish (Andrews *et al*, 1973). Syndromes that appear clinically distinct should be differentiated on the basis of aetiology, treatment response and/or natural history. In the present paper we simply report subtle differences in natural history, but not outcome, and relate these to a presumed aetiological factor (personality) that seems to act differently in the two disorders. Parker *et al* (1987) have previously shown that the parenting of our patients with endogenous depression differed from the parenting of patients with neurotic depression and that this was unlikely to be a reporting artefact.

We agree with Dr Aspin that "the case for a dichotomy is not proven", but there are now three pieces of evidence derived from just this one study that "endogenous and neurotic depression are two illnesses". We claimed no more.

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#### Violence in Alzheimer's disease

SIR: The series of articles on psychiatric phenomena in Alzheimer's disease by Burns *et al* (*Journal*, July 1990, **157**, 72–94) was illuminating. In this letter I would like to raise two issues.

First, the use of  $\chi^2$  test with some analysis appeared to be inappropriate as the expected frequency in the cells of some of the  $\chi^2$  tables was less than 5. Examples of this included Table II on p. 74, Table III on p. 84 and Table IV on p. 89. The more appropriate test would have been the Fisher's exact probability test (with a unidirectional hypothesis).

Second, the above study reported that 20% of patients exhibit aggressive behaviour and this figure

is comparable with that of other studies (Swearer *et al*, 1988). Some of my own unpublished work had indicated comparatively low levels of violence (seven violent incidents in one year) on a long-stay psychogeriatric ward. The reported relationship between aggressive individuals and presence of temporal lobe atrophy on neuroradiology is fascinating. This relationship is consistent with reports of violence and aggression associated with electroencephalogram (EEG) abnormalities focused on temporal lobes (Tunke & Dermer, 1977). There are other reports of association between violence and aggressive behaviour and abnormal EEGs (Maletzky, 1973; Shah, 1989). The latter study was among mentally handicapped patients, a population which may be comparable with Alzheimer's disease patients.

There are reports of successful treatment with phenytoin (Maletzky, 1973) and carbamazepine (Tunke & Dermer, 1977) of violent and aggressive patients without overt epilepsy but with non-specific EEG abnormalities. There are also reports of successful treatment with carbamazepine of aggressive and violent behaviour among patients with dementia (Au Essa, 1986).

Aggressive and violent behaviour is not uncommon in Alzheimer's disease, causes considerable distress among carers, and is often an indication to admit to a long-stay psychogeriatric bed, with consequent iatrogenic disorders. In view of this one wonders if there is a case to use carbamazepine in such patients, which is increasingly used in psychiatry and is relatively free of side-effects (Tobiansky & Shah, 1989). At the very least there must be a strong case for an appropriately controlled trial of carbamazepine in such patients.

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