

imipramine and paroxetine treatment are similar, despite a fivefold difference in the actual costs of medication. This is because the real difference between the costs of 12-week courses of paroxetine and imipramine (£78.12) is small compared with the high drop-out rates (42% for paroxetine and 53% for imipramine) and the consequent substantial costs that a 'treatment failure' then incurs (£488 plus cost of other drug).

While the proposed model is stable despite variations in possible costings, it is worth noting that the actual choice of model is also important. For example, the central assumption that all drop-outs (no matter at what point during the 12-week course they occur) incur the full cost of a treatment failure may be difficult to justify. Early drop-outs (due to adverse effects, particularly likely with imipramine) may allow rapid switching to alternative medication and so be far less costly than drop-outs later in the course of therapy (for example due to lack of efficacy, which affects both drugs more equally). Although no model can ever be perfect, it is important to recognise that the assumptions underlying any given model will influence the results.

As the model allows for a relapse rate of 25%, perhaps some comment should be made about the place and costs of maintenance antidepressant treatment. Most clinicians would advise this after two antidepressant-responsive depressive episodes within 12 months (the time course of the model). The cost of one year's treatment with paroxetine (£412.45) is higher than that of imipramine (£73.00) and even approaches the cost of not treating the patient (a treatment failure). As drop-out rates for prophylactic treatment (once patients are stabilised) are unlikely to differ as they may for acute therapy, cost-effectiveness of maintenance therapy may be quite different from acute treatment. As more studies emerge stressing the importance of prophylactic medication in preventing relapse (e.g. Frank *et al*, 1990; Old Age Depression Interest Group, 1993), taking into account the continuing costs of such treatment will become increasingly important when deciding initial choice of medication.

Finally, one possibly minor point is that Table 2 states that only 5% of drop-outs subsequently attend out-patient departments (variable 10). However, the calculations in Table 3 suggest that all drop-outs attend for five out-patient sessions each, at a total cost of £180. Either there is a typographical error in Table 2 or the actual cost of out-patient treatment would only be 5% of figure stated, resulting in a substantially reduced cost of treatment failure of only £317. The effects of this would be to

reduce the costs of both drugs, although the effect would be greater (by some £20) on imipramine.

FRANK, E., KUPFER, D. J., PEREL, J. M., *et al* (1990) Three-year outcomes for maintenance therapies in recurrent depression. *Archives of General Psychiatry*, **47**, 1093–1099.

OLD AGE DEPRESSION INTEREST GROUP (1993) How long should the elderly take antidepressants? A double-blind placebo-controlled study of continuation/prophylaxis therapy with dothiepin. *British Journal of Psychiatry*, **162**, 175–182.

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Delusion of inanimate doubles

SIR: Castiloe & Berman (*BJP*, May 1994, **164**, 693–696) ask whether the delusional duplication of inanimate objects is an entirely separate entity from duplication of persons but, as yet, there is little reason to suppose that it is. Perception of inanimate objects appears to employ the same neuroanatomical mechanisms as perception of people (Anderson, 1988).

Substitution of inanimate objects occurs in 5–15% of reported cases of delusional misidentification (Anderson & Williams, 1994). Around 60% occur without duplication of person. Doubles are usually, but not invariably, inferior, and this may simply reflect congruence with the prevailing mood state, which is typically of a suspicious, persecutory type.

The cases reported by Castillo & Berman were all elderly women; we have found a tendency for substitution of inanimates to be associated with greater age and female sex, more so than substitution of person (Anderson & Williams, 1994). Sufferers often live alone, and this may contribute to the primacy of inanimate over animate objects, but late-life paranoid disorders without misidentification also show a preponderance of socially isolated women.

The authors correctly refer to similarities with reduplicative paramnesias, which are traditionally considered distinct neurological phenomena. This distinction may be no more than historical artefact, and there is clearly a family of overlapping recognition disorders, including reduplicative paramnesias, classic person misidentifications, substitution of inanimate objects, and visual agnosias.

We were disappointed that Castillo & Berman explained this phenomenon by psychodynamic and psychoanalytical symbolism (Anderson, 1990). If this phenomenon (it is inappropriate to call it a syndrome) has importance, it is in challenging the

acceptability of psychological explanations of delusional misidentification (Anderson & Williams, 1994). Authors proposing psychological pathogenesis accept it is difficult to explain object misidentification by psychological mechanisms (Epoch, 1986) and the assertion that it is simply an extension of duplicated people (Colman, 1933) is immediately falsified by substitution of inanimate objects without person duplication.

'Delusional gross replacement of inanimate objects syndrome' is neither an accurate nor appealing description for a phenomenon which appears to be a variant of the Capgras' phenomenon and which may, for the time being, be most conveniently and simply referred to as the delusion of inanimate doubles.

- ANDERSON, D. N. (1988) The delusion of inanimate doubles: implications for understanding the Capgras' phenomenon. *British Journal of Psychiatry*, **153**, 694–699.
- (1990) Understanding Capgras' syndrome. *British Journal of Psychiatry*, **157**, 622–623.
- & WILLIAMS, E. (1994) The delusion of inanimate doubles. *Psychopathology* (in press).
- COLEMAN, S. M. (1993) Misidentification and non-recognition. *Journal of Mental Science*, **79**, 42–51.
- ENOCH, M. D. (1986) Whose double? In *The Delusional Misidentification Syndromes* (ed. G.N. Christodoulou), pp. 22–29. Basel: Karger.

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Unipolar mania in non-Western cultures

SIR: The plea of Shulman & Tohen (*BJP*, April 1994, **164**, 547–549) for resurrecting the concept of unipolar mania is particularly germane from a cross-cultural perspective.

In 1904, Emil Kraepelin visited several non-Western societies as part of an international lecture tour. During the course of his stay in Indonesia, he noted that there appeared to be an absence of depressive disorders – a fact that he attributed to diet and climate. Today, unipolar mania is still common in many non-Western cultures. In Nigeria, for example, recurrent mania without depression was noted to be "the rule rather than the exception" (Makanjuola, 1982). Of 50 Chinese patients attending the first lithium clinic in Hong Kong, 18 manifested only manic episodes during their affective relapses (Lee, 1992). A higher prevalence of mania than depression was also found in China and other non-Western cultures such as Africa, Indonesia,

and the Caribbean (Singer, 1975). In CCMD-2 (*Chinese Classification of Mental Disorders*, 2nd edition), the official nosologic system widely used in China (Young, 1989, p. 533), a category (code 31) of 'manic disorder' which requires the absence of previous depressive episodes can be found. Not uncommonly, my medical students asked me why Chinese patients with multiple episodes of mania should be regarded as 'bipolar' according to Western nosology.

Although systemic data are lacking, the possibility certainly exists that unipolar manic patients may exhibit particular neuroradiological, polysomnographic or other biological correlates, and a different clinical course, as well as treatment experience and response. In Hong Kong, limited evidence suggested that while thyroid autoimmunity occurred in a proportion of manic-depressive patients, they were absent in patients with unipolar mania. This may support the view that depression, not mania, has an adverse effect on cellular immunity (Lee *et al*, 1992). It is unclear whether the absence of depressive episodes would reduce suicide in this subgroup of 'bipolar' patients, but in eastern India, unipolar mania is associated with significantly increased affective morbidity (Khanna *et al*, 1992).

Major epidemiological surveys using standardised Western instruments have not made a distinction between unipolar mania and bipolar disorder. Given that the non-Western cultures make up 80% of the world but are poorly prepared to publish in the English literature, more attention to unipolar mania is clearly in order.

- KHANNA, R., GUPTA, N. & SHANKER, S. (1992) Course of bipolar disorder in eastern India. *Journal of Affective Disorders*, **24**, 35–41.
- LEE, S. (1992) The first lithium clinic in Hong Kong – a Chinese profile. *Australian and New Zealand Journal of Psychiatry*, **26**, 450–453.
- CHOW, C. C., WING, Y. K., *et al* (1992) Thyroid abnormalities during chronic lithium treatment in Hong Kong Chinese – a controlled study. *Journal of Affective Disorders*, **26**, 173–178.
- MAKANJUOLA, R. O. A. (1982) Manic disorder in Nigerians. *British Journal of Psychiatry*, **141**, 459–463.
- SINGER, K. (1975) Depressive disorders from a transcultural perspective. *Social Science and Medicine*, **9**, 289–301.
- YOUNG, D. (1989) *Chinese Diagnostic Criteria and Case Examples of Mental Disorders*. Hunan University Press: Hunan (in Chinese).

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