Correspondence

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Princess Diana and post-traumatic stress

Sir: Some aspects of the public reaction to the death of Princess Diana illuminate the medicalisation of distress as a contemporary cultural trend, driven in part by the tendency of trauma models to transform the social into the biomedical. First, an apparent increase in consultations with general practitioners for depression was reported (Morris, 1997). More tellingly, Shevlin *et al* (1997) administered the Impact of Events Scale to an opportunity sample of 205 respondents three weeks after the death. Their finding that 28–32% had a "clinically significant reaction" to this event says more about the poor specificity of models of post-traumatic stress than anything else. In my experience, check-lists of post-traumatic stress disorder (PTSD) will recruit cases as readily from people who have fallen off their bicycles as from victims of catastrophic violence and war (Summerfield, 1995). A lack of precision in distinguishing between subjective distress and objective disorder is likely to be exacerbated by the recent reformulation of PTSD in DSM–IV. The criteria for traumatic stressors are widened to include the experience of hearing the news that something bad has happened to someone close or significant. That someone can apparently be Princess Diana; but if this kind of ordinary human emotionality and fellow feeling fits a biomedical paradigm, there is something wrong with the paradigm. Is there a lesson here for the trauma field?

Morris, B. (1997) GPs called into action to help nation recover from shock of Diana's death. *British Medical Association News Review*, September 24, 18.

Shevlin, M., Brunsden, V., Walker, S., *et al* (1997) Death of Diana, Princess of Wales. *British Medical Journal* **315**, 1467.

Summerfield, D. (1995) Addressing human response to war and atrocity: major challenges in research and practices and the limitations of Western psychiatric models. In *Beyond Trauma. Cultural and Societal Dynamics* (eds R. Kleber, C. Figley & B. Gersons). New York: Plenum.

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Effectiveness of lithium

Sir: Moncrieff (1997) continues her efforts to awaken the world to what she must consider a nearly universal delusion; namely, that lithium works for acute mania, bipolar prophylaxis and antidepressant augmentation. While she makes some valid points, the substance of her review is overshadowed by selective inattention to study results and by her assumption that an imperfection in study design is a fatal flaw that invalidates all conclusions (except in those studies she cites to support her position).

There is no question that early lithium/placebo studies of acute mania were limited in scope and design, and would not meet current research standards. None the less, they consistently found lithium superior to placebo despite designs that may well have made it *more* difficult to show drug/placebo differences.

Moncrieff's interpretation of the Bowden *et al* (1994) double-blind comparison of divalproex (n =69), lithium (n=36) and placebo (n=74) in acute mania requires clarification. While the amounts of supplementary medication used were not mentioned, no neuroleptics were allowed at all, maximum doses of chloral hydrate and lorazepam were restricted, neither drug was given during eight hours before behavioural assessments, and neither drug was used beyond day 10 of the three-week study. Moncrieff's most striking omission was not discussing the study design, which excluded patients previously treated with divalproex but admitted 146 patients previously treated with lithium. Prior treatment had been effective and tolerated in only 39% of this group. This design flaw is seldom mentioned when claims are made that divalproex and lithium have equal antimanic efficacy. When treated with lithium, previous responders improved 15.3 points on the Mania Rating Scale compared with only 4 points in the placebo group (and 7.4 points in the divalproex group).

Moncrieff argues that lithium prophylaxis is ineffective, yet the meta-analysis by Davis *et al* (1993) of 10 placebo-controlled studies found that the difference in relapse rate between lithium and placebo (55%) had a statistical significance of $P<10^{-29}$. Admittedly, the suggestion that mania precipitated by abrupt lithium withdrawal may have artefactually increased drug/placebo differences in discontinuation trials has some merit. However, even with gradual discontinuation, eventual relapse rates are still quite high (Baldessarini *et al*, 1997).

Next, while Moncrieff states that "... little advantage can be seen in patients who are taking lithium compared to those who are not," there is considerable (although not absolutely conclusive) evidence that lithium prophylaxis substantially reduces mortality rates (Wolf *et al*, 1996).

Moncrieff's misinterpretations of data supporting the value of lithium augmentation for treatment-resistant depression have already been addressed (Bernadt & Stein, 1997). While not every study has been positive and while none is of perfect design, the use of lithium is the best established of all augmentation strategies.

Finally, lithium causes side-effects and lithium toxicity can kill, but what was described in 1894 as an "old but flourishing blunder" for the treatment for gout is now the standard against which challengers to the mood disorders throne must be compared. If lithium were abandoned by psychiatry, what could possibly take its place?

Baldessarini, R. J., Tondo, L., Floris, G., *et al* (1997) Reduced morbidity after gradual discontinuation of lithium treatment for bipolar I and II disorders: a replication study. *American Journal of Psychiatry*, **154**, 551–553.

Bernadt, M. & Stein, G. (1997) Lithium: evidence reconsidered (letter). *British Journal of Psychiatry*, **171**, 484.

Bowden, C. L., Brugger, A. M., Swann, A. C., *et al* (1994) Efficacy of divalproex vs lithium and placebo in the treatment of mania. *Journal of the American Medical Association*, **271**, 918–924.

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