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.

https://doi.org/10.1192/bjp.172.4.364b Published online by Cambridge University Press

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Moncrieff, J. (1997) Lithium: evidence reconsidered. *British Journal of Psychiatry*, **171**, 113–119.

Wolf, T., Müller-Oerlinghausen, B., Ahrens, B., *et al* (1996) How to interpret findings on mortality of long-term lithium treated manic–depressive patients? Critique of different methodological approaches. *Journal of Affective Disorders*, **39**, 127–132.

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Suicide prevention

Sir: Lewis *et al* (1997) provide an interesting contribution towards a strategy for preventing suicide in the general population. Their conclusion, that high-risk strategies will have only a modest effect on population suicide rates, would seem impossible to contradict on the basis of available data. However, their emphasis on prevention in deliberate self-harm patients seems less well founded as no effective preventive measures have yet been documented. They do not refer in detail to other risk factors including depression and other mental disorders. If we assume that the prevalence of depression in the general population is approximately 5%, versus a conservative estimate of 50% in completed suicides, this would, using the same method as the authors, give a population attributable fraction of approximately 0.3. Studies generally find that approximately half to two-thirds of mental disorders in patients who consult the general practitioner go undetected. It would therefore seem logical that a possibility for prevention would be through training general practitioners in detecting and treating depression and other mental disorders.

The authors also note that high-risk strategies which focus on post-discharge suicide would be expensive compared with most other medical interventions. However, this would be equally true for services for deliberate self-harm patients or any other high-risk strategy. The only argument that, in financial terms, would justify any intervention aimed at preventing suicide would probably be that there are likely to be other beneficial effects beyond 'merely' saving lives (e.g. improving the quality of life and social trajectory for psychiatric patients).

Lewis, G., Hawton, K. & Jones, P. (1997) Strategies for preventing suicide. *British Journal of Psychiatry*, **171**, 351–354.

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Suicidal ideation and short-term risk of suicide

Sir: Morgan & Stanton (1997) state that 83% of in-patient suicides report suicidal ideation. Moreover, many of these individuals who proceed to suicide are symptomatically much improved and they suggest this is misleading. Findings from our recent Australian study of 103 in-patient suicides over a four-year period (Shah & Ganesvaran, 1997) support these findings. In our study, unstable suicidal ideation (i.e. daily fluctuation in suicidal ideation), in contrast to stable suicidal ideation (i.e. continuously either suicidal or not suicidal), was strongly associated with suicide.

Suicidal ideation is not static. Rather, it is a balance between self-destructive and self-preservative wishes at that point in time. Stengel (1977) suggested that victims want neither to live nor to die, but desire both at the same time, usually one more than the other. Some patients, particularly those with recurrent relapses and resistance to treatment, may be perceived by staff as manipulative, provocative, unreasonable, over-dependent and feigning disability; this led to the concept of terminal malignant alienation (Morgan & Priest, 1991). Patients with fluctuating suicidal ideation are particularly likely to fall into these categories. This results in staff criticism and a lower level of support, leading to alienation. The combination of fluctuating suicidal ideation and alienation can lead to failure in the recognition of seriousness of suicide risk. Moreover, this may lead to observation complacency and absconding or less scrutiny while granting leave. In our Australian series 36% committed suicide after absconding and 35% while on approved leave.

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— & Stanton, R. (1997) Suicide among psychiatric in-patients in a changing clinical scene. Suicidal ideation as a paramount index of short-term risk. *British Journal of Psychiatry*, **171**, 561–563.

Shah, A. K. & Ganesvaran, T. (1997) Inpatient suicides in an Australian mental hospital. *Australia and New Zealand Journal of Psychiatry*, **31**, 291–298

Stengel, E. (1977) *Suicide and Attempted Suicide*. Ringwood, Victoria: Penguin.

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Minor psychiatric morbidity in NHS workers

Sir: While the extensive survey by Wall *et al* (1997) covered many NHS workers and appears to have validated many of the smaller-scale studies, the sampling methods used will have excluded many of those at highest risk of work-related psychiatric morbidity. The exclusion of staff "not in a position to respond" includes those on sickness absence and those who have moved because of staff turnover. Both increased rates of sickness absence and high staff turnover can be used as indicators of occupational stress within an organisation (Cooper, 1996). Exclusion of staff redeployed within trusts excludes those who have been moved because of stress-related disorders and ignores the fact that changes in role and responsibility can be major stressors in their own right. Gathering further information about these groups would help to overcome these methodological problems.

Finally, the clinical implications drawn from the study focus on tertiary level interventions, provided through occupational health departments, rather than the primary and secondary level interventions (mental health promotion and mental illness prevention) recommended in the *ABC of Mental Health in the Workplace* (HMSO, 1996).

Cooper, C. (1996) Stress in the workplace. *British Journal of Hospital Medicine*, **55**, 559–563.

HMSO (1996) *ABC of Mental Health in the Workplace: A Resource Pack for Employers*. London: HMSO.

Wall, T. D., Bolden, R. I., Borrill, C. S., *et al* (1997) Minor psychiatric disorder in NHS trust staff: occupational and gender differences. *British Journal of Psychiatry*, **171**, 519–524.

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https://doi.org/10.1192/bjp.172.4.364b Published online by Cambridge University Press