British Journal of Psychiatry (1988), **153**, 843–854

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Editor: Ian Pullen

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Definition of Suicide

SIR: Farmer (*Journal*, July 1988, **153**, 16–20) need not be distressed at not possessing or discovering an ideal definition of suicide – that is to say, one that is both theoretically cogent and robust in practice. No-one has. The reason, I suggest, for this imperfect state of affairs is that while motivation is certainly central to the designation of a death as suicidal, yet for all our concern as psychiatrists about why people do the things they do, we do not possess any clear theory of motivation. There is no generally accepted view as to what types of motivation people have, of how to deal with non-conscious determinants of action (i.e. whether intent and motivation are synonymous), of how clearly to distinguish immediate from ultimate goals, of how to think about hierarchies of motivation, or of how to operationalise our definitions, such as they are. The problem is a very general one and not specific to suicide, and as usual we have to be content with approximations and the risk of misclassification in borderline instances. Progress is nevertheless possible.

Professor Farmer rightly concerns himself with how suicide is defined and investigated by various agencies, especially those that generate official statistics. No-one doubts the need for caution in using (any) official data, but it is worth commenting that differences in definition are eminently researchable. Investigators can – indeed have – contrast legal decisions with those reached by psychiatrists, have organised the exchange of death records between different jurisdictions to determine if doing so results in significant differences in verdicts, have examined the local effects on suicide rates of a change in coroner, have compared suicide rates of immigrant groups with those of their nation of origin, and so forth. The results suggest that on the whole, suicide statistics remain serviceable within certain defined limits. To put the matter metaphorically, it is consensually agreed that the bath contains a baby as well as the bath water.

But my main concern is Professor Farmer's veteran campaign concerning the relation between parasuicide and completed suicide. He hints that the distinction is largely artefactual, given that young women metabolise drugs more effectively than men. It would be instructive to see a properly calculated analysis along these lines, using the extensive data now available. Beyond this, Professor Farmer belabours the issue of the dichotomous versus the unitary view of the parasuicide/suicide relationship. As I understand it, the work of Stengel, Kessel, and others shows that the two groups of patients differ on a large number of characteristics, including their basic epidemiology; on other variables they resemble each other, such as being involved in acts of self-damage. Moreover, there may be an overlap in the sense that a small proportion of parasuicides can be construed as 'failed' suicides. Could I be enlightened as to whether I hold a dichotomous or unitary theory, and what is gained by the distinction?

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Postpartum Mania

SIR: The recent paper on puerperal psychoses by Platz & Kendall (*Journal*, July 1988, **153**, 90–94) was of considerable interest to us, because the method which they used was similar to the one which we published several years ago (Kadrmas *et al*, 1979). However, some findings were different. In their study, unlike ours, there was no significant increase

in readmission rates in puerperal manics over controls. Our manic patients would have included patients who were diagnosed as schizoaffective, manic using the Research Diagnostic Criteria (RDC). There was an unequivocal increase of first-rank symptoms in our postpartum manics when compared with non-postpartum manics. The RDC would have called such patients schizoaffective, manic. Considerable data suggest that mania and schizoaffective, mania are manifestations of the same disease (Clayton, 1982).

Dr Platz and Professor Kendall reported twelve patients with manic disorder and six with schizoaffective disorder. I do not know how many of those schizoaffectives were schizoaffective, manic, although this is an important point. The simple fact that the readmission rate for schizoaffective patients was more like that of the manic patients than of the depressive patients suggests that most of them may have been manic. These 18 puerperal patients had 36 readmissions (2.0 per patient) and the controls 56 (3.1 per patient), a 55% increase. The duration for the combined puerperal patients was 16.2 weeks per patient, compared with 22·2 weeks per patient for controls (a 68% increase). It should be tested as to whether these differences are significant.

In the discussion, the authors suggest that childbirth may be a "uniquely potent" precipitant of psychotic illness, and that it is plausible "that it should be capable of precipitating episodes of illness in women with only a moderate genetic or constitutional predisposition to affective disorders". I do not think this follows. If one assumes that childbirth is a "uniquely potent" precipitant, all bipolar patients, including those that have episodes independent of childbirth, should have mania following childbirth. In fact, a significant proportion do not (Reich & Winokur, 1970). This suggests an alternative explanation: i.e. that puerperal psychoses in some proportion may have a different illness from that which is ordinarily seen as non-postpartum mania.

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Stuttering and Anxiety

SIR: I was interested in the paper by Drs Andrews and Craig on prediction and outcome after treatment for stuttering (*Journal*, August 1988, **153**, 236–240), but, as a psychiatrist who stutters, I feel competent to suggest a clarification of their premise that stuttering is not associated with anxiety or nervousness. The psychometric evidence the authors cite supports the view that stuttering does not indicate trait anxiety, but says nothing to counter the possibility that the phenomenon indicates situational anxiety. Indeed, a consideration of the suggested aetiology of the disorder indicates that it would not be surprising if a stutterer stuttered more when anxious. Drs Andrews & Craig tell us that stuttering is a consequence of inefficient sensory motor integration of speech at the cortical level, and that this is why various tactics, such as adopting a masking tone, are effective, at least temporarily, in reducing the frequency of stuttering. From this, then, it seems reasonable to suggest that conditions which affect cortical function generally may affect the frequency of stuttering. One such condition would be the alerting consequence of the adoption of the fight/flight posture, which is also characterised by the symptoms of situational anxiety. The intensity of the fight/flight position diminishes when the decision to flee or to fight is made, and this would explain why stutterers regain a degree of fluency after this decision. For example, when he or she is angered and goes on the offensive in an argument, or when a difficult interview over which he or she has no control comes to an end, the stutterer tends to become more fluent. An analogy with epilepsy may be relevant; the stutterer is more likely to stutter when tired, just as the epileptic is more likely to have a fit. This suggests that just as fatigue reduces the epileptic threshold, so too it reduces cortical efficiency in carrying out the sensory motor integration tasks required for fluent speech.

This model has implications for the interpretation of Drs Andrews & Craig's results. They identify learning skills to control stuttering as "probably the most important factor" in long-term successful outcome of treatment. This model would suggest that these skills are in fact specific anxiety management techniques, aimed at increasing cortical efficiency by adopting learned patterns of response to a fearful situation. In this context, therefore, it is not surprising that duration of treatment significantly correlates with improvement.

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