PSYCHIATRIC ASPECTS OF DIABETES MELLITUS

DEAR SIR,

How disappointing that in Dr Wilkinson's otherwise excellent review article (*Journal*, January 1981, **138**, 1–9) on diabetes mellitus he should ignore the interactional perspective. Whilst rightly stating that no studies have been designed to test the hypothesis that psychiatric factors play a direct part in the causation of diabetes mellitus, he neglects to mention the important studies of Minuchin and his colleagues in Philadelphia into factors that might contribute to the instability of the diabetes in certain children.

Using the serum level of free fatty acid (a precursor of ketosis as well as an indicator of emotional arousal), Minuchin *et al* (1978) studied the effect of parental conflict on diabetic children. They noted that in families of labile diabetic children, parental conflict was detoured via the diabetic child. Further, at the time one parent brought a child into the conflict the parent's free fatty acid level dropped to normal whilst the child's level rose, and failed to return to normal during the recovery period. The parents of stable diabetic children did not detour conflict via the child. Hence, the emotional arousal related to parental conflict in the labile diabetic children produced a significant physiological change, demonstrating a clear parallel between family interaction and physiological events.

When considering relevant psychological factors in any condition, attention should always be paid to family interaction as well as to the more traditional individual phenomenology and epidemiology.

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Reference

MINUCHIN, S., ROSMAN, B. & BAKER, L. (1978) *Psychosomatic Families*. London: Harvard Press.

DEAR SIR,

There are few critical studies of the interaction between diabetic children, their families, and the presentation or control of diabetes mellitus. Hence beliefs about an 'interactional perspective' tend to be in the nature of speculation. Minuchin and his colleagues' (1978) book *Psychosomatic Families* is concerned mainly with the 'systems model' of analysis in anorexia nervosa, the small part of the text which deals with physiological and psychological aspects of diabetes is both naive and tendentious. The authors' experimental design fails to conform to the principles of scientific method and their observations are presented in a form which prohibits scrutiny. Such research is likely to have low reliability and validity. Moreover Hinkle and Wolf (1952) give a more detailed account of the story.

A recent British investigation into emotional, behavioural, and educational disorders in diabetic children provides more robust information. With co-operation from parents and teachers, Gath, Smith and Baum (1980) collected interview, questionnaire, and behavioural scale data on 76 diabetic children (43 male, 33 female; mean age 10.9 years; mean duration of diabetes 3.5 years). In addition they looked for correlations between their findings and a paediatricians' estimates of the childrens' diabetic control over the period of one year (good/average/poor). The results showed that although psychiatric disorder was not commoner in the diabetics than in classmate control subjects, as a group the diabetics were more backward at reading than their peers. Also, poor diabetic control was associated with the presence of psychiatric disorder, backwardness at reading, and (with statistical significance) adverse psychosocial factors as classified according to axis V of the multiaxial classification in ICD-9. So I agree with Dr Lask that the formulation of a clinical problem takes into account physical, psychological, and social variables.

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CAUSAL MODELS AND LOGICAL INTERFERENCE IN EPIDEMIOLOGICAL PSYCHIATRY

DEAR SIR,

I thank all the authors in the recent Discussion (*Journal*, December 1980, **137**, 84–85) for their interest in my paper (*Journal*, April 1980, **136**, 317–25).

I agree with Harré that a causal hypothesis must be derived from an underlying theory and I should have made this clear. Indeed Brown and Harris (1978) have suggested a speculative mechanism for a vulnerability hypothesis. It does seem however, that in the current state of knowledge the fact that a *prima facie* case cannot be made must undermine the underlying causal hypothesis. *Post hoc* salvage remains a dubious exercise.

Turning to Tennant and Thompson, the distinction between logical and material implication is exactly the one I was trying to make, although I did not express it

in their specialist vocabulary. Epidemiology is not a commonsense discipline and conceptual difficulties do create confusion. It is therefore sad that Tennant and Thompson did not elaborate on the ideas expressed in their last paragraph. The idea of 'weak implication' may be, as they claim, foreign to logic, but it does exist in the sociological literature (Boudon, 1974). Finally, I have made abundantly clear the distinction between the sentential operator and the unfortunate use of arrows to indicate causation.

Cooke's demonstration of the effects of dichotomization on a continuous variable is interesting but in this instance I have reservations. The claim that variables can be summed to produce a continuum must rest on the existence of a valid cardinal scale. I would claim that we have no valid scale for an overall measurement of life event stress. We have not established for example that two life events are equivalent to, say, twice the stress of one event, or that we know how events of differing magnitude can be summed. A research programme to do this would be very difficult. A categorical approach to life events may therefore provide a sounder basis for our conclusions.

Global scores of psychiatric symptoms present similar problems. Is a person with three symptoms necessarily as disturbed as one with three other symptoms? Is a person with six symptoms necessarily, say, twice as disturbed as a person with three? Global scores do not take into account the fact that symptoms differ qualitatively. I do not say these questions cannot be answered but they have not been answered yet. Global symptom scores should be seen as a complement to the practice of defining cases of diagnosed disorder. Each is likely to be a useful but incomplete way of analysing data.

Brown and Harris in their defence of the vulnerability model use the article by Everitt and Smith (1979) to justify the use of their additive statistical approach, but the thrust of that article is that the additive model is statistically suspect. They claim that certain ways of partitioning the $2\times2\times2$ table run counter to prediction because of small numbers in certain cells, which in my view amounts to saying that if the numbers were different, the results would be different. The drawback of the additive method of analysis is that it does not simultaneously take into account the cell frequencies of the whole $2\times2\times2$ table whereas multiplicative methods like log linear analysis do. Finally, inconsistent findings concerning one vulnerability factor cannot be explained away by citing the distributions of another which has been established in the same doubtful way. If interactions between vulnerability factors are thought to be important in determining the distribution of cases and life events a further simultaneous analysis is needed to demonstrate or refute them.

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THE CURRENT TREATMENT OF ANOREXIA NERVOSA

DEAR SIR,

Professor Gerald Russell's interesting article (*Journal*, February 1981, **138**, 164–166) prompts certain remarks. It seems a pity that anorexia nervosa, in the present state of our knowledge, is still regarded as an illness. Surely it must be a symptom, and usually a symptom of maturational difficulties.

Obviously there has to be a realistic concern about the patient's weight, and proper goals set for this, but surely we should not be content with "supportive psychotherapy, aimed at helping resolve the psychological conflicts which may be identified as contributing to the illness". I am sure that most of us who work with adolescents, and many of the problems faced by the anorexic patient are similar to those found in adolescent patients who are not anorexic, are aware that there is a lot of scope for effective creative psychotherapy which is by no means merely supportive.

I also think that it is no good urging the DHSS to do anything very much, and that if we need to provide local facilities for the treatment of eating disorders this must be done by local pressure.

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