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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