male pharmacists clearly suggests that these must be significant factors. On the other hand, if being a doctor or a doctor's wife carries with it a special kind of distress loading, clearly the physician is not unique in this either. Being an unskilled worker may be a worse predicament. By the same token, there appear to be some professions (e.g., the clergy, politicians) that are spared excessive risks of suicide. If we are to understand why doctors and their wives are at higher risk for suicide, we might benefit from undertaking in-depth comparisons of their demographies, lifestyles and value systems with those of other occupational groups.

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References

SAKINOFSKY, I. (1980*a*) Suicide in doctors and wives of doctors. *Canadian Family Physician*, **26**, 837–844.
——(1980*b*) Suicide in doctors and their wives. *British Medical Journal*, **281**, 386–387.

Mania Following Bereavement

SIR: In his letter (*Journal*, August 1986, **149**, 244) Bridges raises interesting points apart from, rather gallingly, misreading our intendedly ironic "Freudian" reference (*Journal*, April 1986, **148**, 468–70). He claims that in opposing grief and mania we create a false paradox in our patient's manic sequel to bereavement. If it is argued that grief and mania are not opposites then, by Bridges' own statements, grief and depression should have no relation. The disjunction of grief and depression is a little more problematic. The phenomenological closeness of these conditions is embedded in DSM-III, which requires the phenomena of "major depression" not to be caused by grief (p. 214). I do not believe DSM-III necessarily embodies 'truth', but it does conform to a body of respectable opinion, as do concepts of pathological grief, and to the extensive literature on bereavement and depression. Grief may be a normal experience, but it is not clearly differentiated from depression except by the presence of bereavement or loss. Bridges' placement of "happiness" as the polar opposite of grief is no less suspect than our opposition of grief and mania, or his separation of grief and depression. Only a psychiatrist who dealt with the extremes of illness would have the luxury of seeing such clear separations.

One of the worst sins of the analytic movement was to treat the hypothetical entities of "defences" as real or phenomenological entities. Bridges refers to "mani[a]-an illness" with a conviction that similarly treats the hypothetical entity of illness as a real or phenomenological entity. Moreover, psychoanalytic theory offered trite and circular 'explanations' for many mysteries of human behaviour and thereby closed them to investigation for decades. To speak of "specific vulnerability/non-specific stress' creates an illusion of explanation which threatens to do the same.

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The Prognosis of Depression in Old Age

SIR: Baldwin & Jolley (*Journal*, November 1986, **149**, 574–583) point out that "much thought needs to be given to research methodology in this field." I could not agree more; the authors' retrospective study of case notes can hardly be regarded as rigorous or comparable with a prospective follow-up study of the type I conducted. It is well recognised that case note reports of outcome frequently bear no relation at all to the mental states discovered in face-to-face interviews and no doubt the same vagaries of reporting afflict doctors' notes in Manchester as they do in East London. Baldwin & Jolley did, however, interview those alive for their long-term study. They reported a mortality rate (35%) remarkably similar to the 37% mortality rate of the East London cohort over a 4 year period (in press). If the dead are excluded, then 40% of their patients remaining alive were either 'continuously ill" or suffering from "depressive invalidism". No different from Post's findings and not much different from mine!

It seems to me that where our methodology is similar, the results are similar, though perhaps they view the same results with more optimism. Baldwin & Jolley's pint pots are always half-full, whereas mine are half-empty!

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Depression in School Phobia

SIR: We note with concern the comments of Weinberg *et al* (*Journal*, March 1986, **148**, 335). Some of the phraseology suggests that their views may be influenced by considerations beyond the substance of the Newcastle method. They enunciate standards which are easier to apply to the work of others and also over-emphasise shortcomings which we have

already listed. In the selection of diagnostic criteria, we had pointed out (p. 353) that, as there was no control group (normal controls), the items identified would not necessarily discriminate between those who were well and those who were depressed.

As to his other points: (i) We believe it is valid to attempt to distinguish between neurotic disorders in childhood with and without depression, but have stated that further work needs to be undertaken to establish whether our formula can be applied more widely. Recent research on a fresh population has validated the formula. (ii) Reliability has been previously reported (p. 355 and Berney *et al* (1981). (iii) We are aware of the evidence for a genetic basis for bipolar affective disorder, but the case for this is less strong in the milder depressive disorders which present in out-patient practice. (iv) The theory of an underlying substrate of right cerebral dysfunction is controversial and cannot be supported as yet.

We regret the confusion about the origin of the Weinberg criteria, which derive from the Washington University research criteria. However, other workers (Puig-Antich, 1980) have already pointed to serious methodological flaws in the use of these criteria by Weinberg. By RDC criteria we imply the variation used by Puig-Antich. The current Newcastle Depression Project has looked again at the various sets of diagnostic criteria in relation to a clinic population (Kolvin *et al* – in preparation).

We also note with interest the comments by Atkinson (*Journal*, March 1986, **148**, 335–336). There are some major as well as some simple misunderstandings of the procedures used. We disagree with some of the views expressed. He also seeks extensive statistical details which are not usually published.

Comparison of groups (Table I). It is axiomatic that statistical critics should get their formulae right. On Atkinson's own analysis, a 31-item total global depressive score would lead to a probability of 1.5 and there is no such thing! Further, the formula would need to be modified when using higher levels of significance.

Initially our data in Table I was presented both as percentages and as means and standard deviations. Subsequently we used the device of the best cut merely for the ease of presentation. We agree that in the comparison of the groups it would have been best for us to make a cut on *a priori* theoretical grounds. The above use of the term 'best cut' when comparing groups has given rise to confusion.

Best cut approach. We agree this is best confined to the technical sense of identifying the optimum point for discrimination on a scale used for screening purposes. We merely revised the threshold of the cut so as to identify the optimum point for the purpose of discriminating cases from non-cases. Making a cut on *a priori* theoretical grounds does not allow the emergence of the most sensitive discrimination. This is a legitimate technique and does not inflate the discriminant efficacy of the measures in question.

Item analysis. The ideal sample would have included children matched for age and sex from the general population, but this was not feasible.

Use of weights derived from discriminant function analysis. Discriminant function analysis was used to explore a series of hypothesised classifications. Data from our multivariate analyses did not contribute to our diagnostic formula. We have not used weights derived from discriminant function analyses. This is a laborious technique which offers no clear advantages over simpler methods (Goldberg, 1972).

Principal component analysis. (i) This is a robust technique well suited to clinical data which may not fit classical assumptions of multi-normality. Atkinson cites a ratio (of subjects to variables) of 5 : 1, but others recommend a ratio of not less than three or four times as many observations as variables (Taylor, 1977). It is true that we interpreted this rather liberally in relation to social and family background data, but we were cautious in the conclusions we drew. While in PCA the ideal sample size should be relatively large, we again contend that it can be used as an exploratory tool on a moderately sized sample. Dogmatic statements about sample size suggest an inflexibility in the use of statistics. (ii) In the first PCA the correlation matrix was only moderately consistent with an underlying structure and our criterion for determining the number of components was that of a variance which stood out as far larger than the rest. This is a pragmatic and arbitrary approach but more sophisticated techniques are inappropriate in a small sample (Lawley & Maxwell, 1971). In the second PCA the correlation matrix proved more consistent with an underlying structure and the first two components accounted for a substantial amount of the variance. A retrospective analysis, using the rule whereby the number of factors extracted is equal to the number of Eigen values greater than unity, supported our decision. We did not use rotation because the '*n*' was moderate. While some of the factor loadings which were noted were rather low, this does not detract from the differentiating bipolar second component which contrasts affective symptomatology with premorbid personality traits.

As clinical investigators we have presented the early steps in the development of a set of diagnostic criteria – fully aware of the pitfalls of our method. We were equally aware both of the importance of using scarce opportunities to their full, and of the

necessity to ensure further validation and replication on fresh samples.

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References

BERNEY, T. B., KOLVIN, I., BHATE, S. R., GARSIDE, R. F., JEANS, J., KAY, B. & SCARTH, L. (1981) School phobia: a therapeutic trial with clomipromine and short-term outcome. *British Journal of Psychiatry*, **138**, 110–118.

GOLDBERG, D. P. (1972) *The Detection of Psychiatric Illness by Questionnaire*. Maudsley Monograph. Oxford: Oxford University Press.

LAWLEY, D. N. & MAXWELL, M. A. (1963) *Factor Analysis as a Statistical Method*. London: Butterworth.

PUIG-ANTICH, J. (1980) Affective disorders in children. *Psychiatric Clinics of North America*, **3**, 403–424.

TAYLOR, C. C. (1972) *Principal Component and Factor Analysis in Exploring Data Structures*, Vol. 1. (eds C. A. O'Muircheartaigh & C. Payne). New York: Wiley.

Psychiatric Morbidity and the Mentally Handicapped

SIR: Day (*Journal*, December 1985, **147**, 660–667) reported that 30% of mentally handicapped residents aged over 40 years and 20% of those admitted to a psychiatric day hospital for the mentally handicapped aged over 40 years had a significant psychiatric disorder. This study contains artefacts which lead to an over-estimation of the prevalence of psychiatric disorder.

No information is given concerning reliability. Poor reliability may have occurred at the time of diagnosis, during transcription of diagnoses from case notes, when diagnoses were re-classified into five super-ordinate categories (e.g., into the categories "psychosis" vs "psychosis"), and when diagnoses were made from case notes only (as in 6% of cases).

If the aim of the study was to estimate the current prevalence of psychiatric disorder then the inclusion of people with a history of psychiatric disorder clearly inflates this. For example, people may have previously suffered a single episode of an illness or may have been treated and no longer show the disorder.

Day gives no formal definition of "behaviour disorders" although he refers to examples of this. Whilst some instances of such behaviours have been shown to be associated with specific disorders (e.g., Lesch-Nyhan syndrome) it is not clear what *proportion* of such behaviours are indicative of psychiatric morbidity. Some behaviour disorders are associated with painful physical illness such as otitis media, undetected dental abscesses and chronic nasal infection. Others may be considered examples of learned behaviours. Behaviour disorders are clearly of heterogeneous origins and only some are psychiatric in nature (Jacobsen, 1982).

Finally, Day includes a number of offensive and troublesome behaviours as psychiatric morbidity. These include behaviours such as wandering, stealing, and public masturbation. Such problems may not reflect psychiatric morbidity in all cases but a variety of other problems such as lack of privacy, poor learning history or an environment which maintains aberrant behaviours.

Behaviour disorder accounted for 50.5% of psychiatric disorders in the long-stay residents and 35.5% of the day hospital admissions. If a substantial proportion of these could not be demonstrated to be psychiatric in nature then the prevalence of psychiatric morbidity would fall substantially.

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JACOBSEN, J. W. (1982) Problem behaviour and psychiatric impairment within a developmentally disabled population. I: Behaviour frequency. *Applied Research in Mental Retardation*, **3**, 121–129.

The Effect of Sulpiride on Negative Symptoms of Schizophrenia

SIR: Activating or disinhibitory effects of neuroleptics given in low doses to patients with schizophrenia were described when these drugs were first introduced to psychiatry (Delay *et al*, 1957), but subsequent experience with conventional anti-psychotic drugs has failed to provide convincing evidence of a useful dose-related bipolarity of effect. From the time of its original use in psychiatry, however, subsidiary effects of sulpiride – described in a variety of terms such as "anti-autistic" or "thymo-analeptic" have repeatedly been noted (Collard, 1969), and neuropharmacological studies (Sokoloff *et al*, 1980; Brown & Arbuthnott, 1983) suggest sulpiride may show a clinically useful separation of dose-related effects on psychiatric symptoms, low doses having activating and/or antidepressant properties, while higher doses are effective against positive symptoms of schizophrenia.

Since there are no controlled studies in this area, we have undertaken a double-blind comparison of normal versus low dose sulpiride in patients with chronic schizophrenia characterised predominantly by the negative symptoms poverty of speech and flattening of affect.