AUTHOR'S REPLY: Dr Pantelis articulates well a paradox which continues to dog our thinking about schizophrenia, but which is built-in to current classification systems: that schizophrenia is a syndrome, but a syndrome with a presumed organic aetiology. As with other complex, common diseases in medicine – such as ischaemic heart disease, epilepsy, or Alzheimer's disease – schizophrenia will turn out to have a variety of genetic and non-genetic causes. Identifying subtypes which seem to have demographic and symptomatic similarities will facilitate the search for these causes: perhaps an operationally-defined "neurodevelopmental" subtype is an example of this, as would be strongly familial subtypes, late-onset subtypes, subtypes secondary to other central nervous system diseases, and so on.

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The myth of suicide prevention by general practitioners

SIR: I thank Dr Hawton and Professor Morgan (*Journal*, March 1993, **162,** 422) and Dr McCabe (*Journal*, February 1993, **162,** 270) for drawing my attention to Rutz *et al*'s (1989) study in Gotland to support their optimism about suicide prevention by general practitioners. I was obviously wrong in suggesting that statistically significant differences would be difficult to find without large samples over long periods of time.

However, a follow-up study (Rutz *et al*, 1992) allows the original data to be set in temporal context. I have combined the data reported from these two studies in Figure 1. The interventions clearly coincided with random variations in a rate which had started to decline, for unknown reasons, in the late 1970s. This highlights the methodological problems I was trying to emphasise in my letter (*Journal*, October 1992, **161,** 574). Although Rutz *et al* managed to arrange the data to produce various statistically significant differences, Figure 1 clearly shows that the intervention had no impact at all on suicide rates in Gotland.

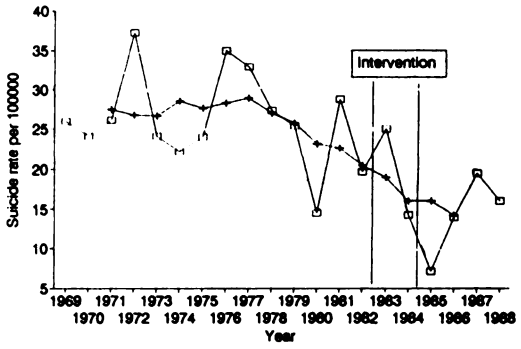

Fig. 1 Suicide in Gotland (Rutz *et al*, 1989, 1992) (□ = annual rate; + = five-year moving average).

I also thank Dr Hawton and Professor Morgan for drawing my attention to the incidence of some general medical conditions in general practice. Despite similar incidence rates, the difficulty in recognition of immediately potential suicide is obviously much greater than for these conditions. Quite apart from differences in volunteering relevant symptoms, someone's unrecognised Crohn's disease one month will still be available for belated recognition the next; the unrecognised suicidal patient will be dead.

I appear to have given the impression that I am opposed to improved recognition and treatment of psychiatric disorder in general practice. If the improvement requires the diversion of resources (energy and money) from the assessment and management of severe psychiatric disorder by secondary health care teams then this impression is correct. Let psychiatrists be satisfied with the organisation and funding of their acute services, in which the incidence of suicide is higher and more likely to be directly amenable to improvements, *before* telling general practitioners what to do to prevent these rare events in their patients.

RUTZ, W., VON KNORRING, L. & WALINDER, J. (1989) Frequency of suicide on Gotland after systematic postgraduate education of general practitioners. *Acta Psychiatrica Scandinavica*, **80,** 151–154.

——, —— & —— (1992) Long-term effects of an educational program for general practitioners given by the Swedish Committee for the Prevention and Treatment of Depression. *Acta Psychiatrica Scandinavica*, **85,** 83–88.

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Affect as a neurobehavioural probe in the evaluation of hypofrontality

SIR: I read with interest the article by Berman *et al* (*Journal*, February 1993, **162,** 183–192). The authors have used sensorimotor and cognitive tasks (Wisconsin Card Sorting test) as neurobehavioural probes to differentiate the pathophysiological mechanism of hypofrontality in schizophrenia and depression. Besides cognitive tasks, affect also has been recommended as a probe to elucidate the association

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between regional brain activity and behaviour in psychiatric disorders (Gur *et al*, 1992). In my opinion 'affect' could also have been used as a probe to accomplish the objective of this study.

The rationale for considering affect as an appropriate probe in the evaluation of prefrontal activity comes from the literature that substantiate the role of frontal lobe in emotional behaviour (Akert, 1964; Robinson *et al*, 1984). Further, in view of the fact that psychopathology of emotional disturbance in schizophrenia and depression varies significantly, one may assume that these conditions may be associated with differential frontal lobe activity to emotional challenge, complementing the observed finding that showed abnormal regional cortical blood flow in frontal lobes following cognitive activation in schizophrenic patients compared with depressive patients.

The application of this strategy using emotional challenge would also allow us to observe the differential frontal activity to emotional and cognitive tasks in normals. But the non-availability of standardised emotional tasks that can be used as a probe limits the practical application of this strategy. However, valuable guidelines provided by Gur *et al* (1992) regarding conceptualisation, construction and standardisation of new neurobehavioural probes would be helpful in the development of the procedure and application of emotional challenge in future research.

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GUR, R. C., ERWIN, R. J. & GUR, R. E. (1992) Neurobehavioural probes for psychologic neuroimaging studies. *Archives of General Psychiatry*, **49**, 409–414.

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Clinical studies of the dementias

SIR: It was flattering to be so quoted by Dr Brayne (*Journal*, April 1993, **162**, 439–446). While presenting an interesting epidemiological view of clinicopathological studies in dementia, some comments are necessary to put the issues in perspective for the general psychiatric reader. She is correct to highlight the importance of the sampling frame in such studies, but the sources of bias to which she refers pale into insignificance when one considers the proportion of people who come to post-mortem, an issue not addressed by the author. We achieved a positive response rate of about 75% which we believe to be reasonable (Burns *et al*, 1990*a*). Post-mortem rates of 100% would be misleading as people not consenting to the procedure would inevitably be excluded. To imagine that the epidemiological method should or could be uncritically applied to a procedure comparatively rarely undertaken seems incorrect. Also, in epidemiological studies the number of demented patients is in the minority, and so an enormous amount of work and normal post-mortems would be required to get a so-called 'representative sample' of those with dementia. As stated in the lively correspondence following the publication of our study, we would have sorely liked to have included more detail of the sampling frame, but could not due to lack of space.

The finding of particular clinical features in a sample does depend from where the sample is drawn and it is to be accepted that those samples with a psychiatric bias may include more with behavioural disturbance or psychiatric symptoms. The case that Lewy body dementia is peculiarly associated with such disturbances is unproven, as is Dr Brayne's assumption that this may have led to the relatively high proportion of Lewy body cases in our series – in fact, our series does not contain a larger number than others (Perry *et al*, 1989).

The introduction of clinical criteria for the dementias has a number of aims and should not be seen as the exclusive purview of the epidemiologist. To achieve an accurate diagnosis in an individual case is an important clinical objective. This was one of the main aims of our study and although we were rightly chastised for the use of the term 'sensitivity', the title of our paper '*Accuracy*' should not have misled even the most naïve reader (Burns *et al*, 1990*b*). We do not agree that the reason for the different positive predictive values in our own study and that of Homer *et al* (1988) was due to a differing prevalence of Alzheimer's disease. Homer *et al* did not employ any recognised clinical criteria, indeed it was as a reaction to that paper and the associated widespread diagnostic nihilism which prompted us to report our own study in the same journal. Diagnostic criteria *are* being developed for vascular dementia. In fact, followers of that diagnostic group now have two to choose from! (Chiu *et al*, 1992; Roman *et al*, 1993.)

Dr Brayne is to be congratulated for producing such a timely review of this important issue. Clearly, dementia is such a hard nut to crack that contributions from a number of different perspectives are

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