right renal vein and artery. Complete removal was achieved. The histology of the tumour was compatible with phaeochromocytoma. Total urinary catecholamines in three 24-hour specimens pre-operatively were 769–900 μg or 2.38–2.75 μmol (normal, up to 180 μg or 0.55 μmol), and vanillyl mandelic acid 29–40 mg (normal less than 7 μg). After operation these values fell to 97–112 μg total catecholamines and 4.7–7.9 mg VMA, respectively.

In the immediate post-operative period she was treated with propranolol 60 mg twice daily and phenoxybenzamine 10 mg twice daily. Those drugs were gradually reduced and stopped in ten days by which time her blood pressure and pulse were consistently normal. She was observed for a further six weeks during which time her mental state was normal on no treatment at all. During the three years of follow-up, no abnormal behaviour or mood has been reported by her relatives or observed by the outpatient staff.

The most common presenting features of phaecochromocytoma whatever its site may be are intermittent sweating, headache, palpitations, and arterial hypertension, and this diagnosis can be made confidently in at least 85 per cent of cases on clinical grounds alone (Gifford *et al*, 1964).

This patient had episodes of auditory and visual hallucinations, paranoid ideas and delusional perception as other major features of her illness, at times when she was alert and correctly orientated. That these symptoms remitted after surgery and have not recurred in spite of no medication for three years suggests that they were causally related to the tumour and its pathological secretions. What particular catecholamine metabolites were present in the secretion, and whether they could precipitate psychotic symptoms we do not know, but theories relate dopamine neuronal supersensitivity to schizophrenia (Owen *et al*, 1978) and noradrenaline receptor weakness to severe depression (Schildkraut, 1965), and therefore interference with brain function by catecholamine substances in abnormally large amounts is a plausible explanation of this woman's psychosis. So far as I know this is the first report of a schizophreniform psychosis in a case of phaeochromocytoma.

M. Bahemuka
College of Medicine and King Khalid Hospital,
Riyadh,
Saudi Arabia

References

Gifford, R. W., Kvale, S. F., Maher, F. T., Roth, G. M. & Priestley, J. T. (1964) Clinical features, diagnosis and treatment of phaeochromocytoma: a review of 76 cases. *Mayo Clinic Proceedings*, **30**, 281–302.

Owen, F., Crow, T. J., Poulter, M., Cross, A. J., Longden, A. & Riley, G. J. (1978) Increased dopamine-receptor sensitivity in schizophrenia. *Lancet, ii*, 223–5.

Schildkraut, J. J. (1965) The catecholamine hypothesis of affective disorders: a review of supporting evidence. *American Journal of Psychiatry*, **122**, 509–22.

RATE OF BLINKING MAY PREDICT NEUROLEPTIC-INDUCED PARKINSONISM

Dear Sir,

There are suggestions that neuroleptic-induced parkinsonism is mediated by dopaminergic blockade, and recent studies indicate that the rate of blinking is a centrally regulated phenomenon related to dopamine turnover as well as the integrity of the basal ganglia. We studied the rate of blinking in 26 consecutive schizophrenics, diagnosed according to the Research Diagnostic Criteria of Spitzer *et al* (1975) and treated with a neuroleptic (trifluoperazine 15 mg daily) for the first time. We found a negative correlation between pretreatment blink rates and parkinsonism scores during treatment, estimated using the Simpson-Angus scale ($\chi_2 = 7.58$ $P < 0.01$). Compare Karson *et al's* 1981 finding that neuroleptics decrease blinking in schizophrenic patients.

If this observation is confirmed, routine bedside estimation of the blink rate may provide a useful pointer to patients for whom antiparkinsonian medication should be prescribed.

M. S. Keshavan
I. V. L. Narasimha Rao
H. S. Narayanan
645 Studentenheim Akademikerhilfe,
3A Pfeilgasse, 1080 Wien,
Austria

References

Karson, C. N., Freed, W. K., Kleinman, J. E., Bigelow, L. B. & Wyatt, R. J. (1981) Neuroleptics decrease blinking in schizophrenic patients. *Biological Psychiatry*, **7**, 679–82.

Simpson, G. M. & Angus, T. W. (1970) Drug induced extrapyramidal disorder. *Acta Psychiatrica Scandinavica*, Supp. 212.

Spitzer, R. C., Endicott, J. & Robbins, E. (1975) Research Diagnostic Criteria—Rationale and reliability. *Archives of General Psychiatry*, **34**, 136.

A COMPARISON OF DEPRESSION RATING SCALES

Dear Sir,

Kearns *et al* (*Journal*, July 1982, **141**, 45–9) boldly suggest that the Beck Depression Inventory, its subscale, and the Wakefield Inventory "should now be abandoned in research", (p 45). In my opinion this is a

premature and unjustified conclusion untenable on the basis of their research design and use of inappropriate statistical methods. More specifically:

1. The criterion against which the rating scales were compared was a set of ratings of severity of depression made by two psychiatrists and a nurse. The authors use a weak non-parametric test (rank-order correlation) to test their own scale, yet use a relatively stringent parametric test to evaluate the rating scales. And as the authors compare rating scale scores on adjacent severity levels based on their own 'scale', then they should have presented reliability figures for *each adjacent pair of severity levels*. For all the reader knows, the pairs of items on which the BI and the WI are 'failed' could be of very low reliability: certainly a crude measure such as the rank-order correlations tells one little about the reliability of discrimination between two items.

2. Related to this first point, the scales scrutinized by the authors are a result of extensive piloting and validation studies. This is not the case for the *ad-hoc* scale devised by the authors. Why is this weak instrument used as a criterion by which to judge properly designed and tested instruments?

3. Finally, in many cases, self-rating inventories as opposed to interviewer-rating scales are necessary in research. The two self-rating scales which 'pass the test' do no better in my opinion, than do the Wakefield and Beck Inventories by the criteria of the authors' flawed design. Taking the author's 6 grade scale for instance, the Leeds Scales fail to discriminate between two levels at the 0.05 level of significance, and the Irritability-Depression-Anxiety Scale fails to discriminate on three levels. The Beck Scale, on the other hand, fails to discriminate on three comparisons, and the Wakefield on two. Why do the authors conclude that the two latter instruments are unsatisfactory, and the former ones satisfactory?

In conclusion, the authors fall short on scientific caution and experimental design, and their recommendation to abandon two instruments should be rejected until more adequate studies are conducted.

IAN ROBERTSON

The Andrew Duncan Clinic,
Morningside Terrace,
Edinburgh EH10 5HF

DEAR SIR,

I welcome the opportunity to reply, on behalf of my colleagues, to Dr Robertson's criticisms of our study. Dr Robertson seems to take great exception to our choice of criterion which he refers to as 'a weak instrument'. He does not say what criterion he would have chosen; clearly it would have been futile to compare all the scales with yet another scale. We could have chosen some weak measure of severity such as in-patient, day-patient or out-patient status or videotaped assessments of some characteristic such as retardation. However we considered that our choice of a criterion, derived from an interview by an independent psychiatrist and a nurse who knew the patient well, was the best that can be obtained; we still maintain this and believe it is one of the major strengths of our study.

I do not follow Dr Robertson's objection to our use of a non-parametric rank correlation which is generally accepted for ordinal data; we were not 'testing our own scale' but providing information that we were justified in combining the psychiatrist's and the nurses' ratings into a single measure. Nor have we stated anywhere that we used 'rank order correlations to test the reliability of discrimination between items'; we used the parametric Student's t-test to distinguish between scale scores at different degrees of severity and still consider that we are justified in doing so. The further objection is made that we failed to provide information concerning the differences between the various grades in our criterion; again, I do not understand this since the information is all supplied in our first figure and the critical reader can soon assure himself that the differences between all successive grades are in fact statistically significant.

Our advice to abandon the use of the Beck and the Wakefield scales rests not only on the number of non-significant differences between successive grades but also on the finding that, in those two scales, higher scores were achieved at a lower compared with a higher grade. The advice also has another source which is that of economy of time; the Beck Depression Inventory consists of 21 items and the user is advised to read all these aloud to the patients. The expenditure of this amount of time would be justified if it resulted in a more accurate assessment but our study has shown this not to be the case.

Finally Dr Robertson appears to have a rather naive belief that certain well-known scales are better than others because they have been subjected to 'extensive piloting and validation studies': What in fact happens is that a scale is frequently devised, often after a minimum of preliminary work, and is subsequently found to work in a number of situations; this need not cause much surprise or prove that the scale is particularly good. At another place (Snaith, 1981) I submitted that one of the major impediments to progress in psychiatry was the primitive state of measuring instruments. Progress will continue to be hindered if research workers continue, perhaps out of some form of misguided loyalty, to adhere to the use of the scales provided by the earlier pioneers in psychometrics. The lack of comparative studies of various