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.

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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.

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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