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Correspondence

Correspondents should note that space is limited and shorter letters have a greater chance of publication. The Editors reserve the right to cut letters and also to eliminate multitudinous references. Please try to be concise, strictly relevant and interesting to the reader, and check the accuracy of all **references in Journal style.**

PHAEOCHROMOCYTOMA WITH SCHIZOPHRENIFORM PSYCHOSIS

DEAR SIR,

A 35 year old Kenyan woman was admitted to Kenyatta National Hospital, Nairobi, a week before Christmas 1978 with a four week history of intermittent palpitations, sweating, dizziness and headache. These symptoms would last for about an hour at a time. They were not associated with chest pain and were not related to exercise.

Her previous health was normal. Her premorbid personality and behaviour were, according to her husband, normal. She had been a quiet, uncomplaining and generally a happy person all her life. There was no family history of neurological or psychiatric disorder. She was not taking any drugs before admission, neither was she given any for several weeks as an inpatient while investigations were being done.

On examination she looked well. The only abnormalities were a blood pressure of 240/140 mm Hg and a pulse rate of 120 per minute. The subsequent blood pressure and pulse charts demonstrated intermittent high levels, the highest blood pressure recording at any one time being 280/170 mm Hg. Headache, sweating, palpitations and apprehension coincided with the high blood pressure recordings. These preliminary observations strongly suggested the diagnosis of phaeochromocytoma.

The urine analysis on several occasions showed that she had intermittent glycosuria but the glucose tolerance test was normal.

It was observed that she was often withdrawn, detached and would be talking to herself. In between these episodes she seemed friendly and cooperative. On the Christmas Eve, she became aggressive and complained that the ward decorations were meant to be used for hanging her and that the nurses were trying to poison her food. Examination at that time showed that she was correctly orientated and was alert. She recognized everyone around her and knew that it was a time of celebrations. There were no neurological or any other signs, she was apyrexial and there was nothing clinically to suggest any infection anywhere. This episode lasted about one and a half hours. During this time, the blood pressure was normal on the two occasions it was measured.

In the subsequent weeks, these episodes became more frequent. On two occasions she complained that the music that was regularly played to the ward through the loud speaker was the voices of angels calling her to her death. However, whenever the episodes were over she would join in the activities of the ward and would apologize when reminded of her previous behaviour. The most dramatic incident was when she attacked her neighbour in the ward, whom she accused of talking ill of her. Complaints of everyone hating her were reported to the ward sister frequently, but in spite of her allegations, she was not unduly perturbed or violent most of the time. However, she at times talked about several disconnected subjects in quick succession.

On several occasions she complained that she was hearing voices of her enemies plotting her demise and would sometimes point at 'them'. She became annoyed when the nurses and house doctors could not see what she was pointing at. She always remembered afterwards that all her accusations and complaints were unfounded. At no time was she confused or drowsy. Several electroencephalograms done when she became cooperative within a few minutes of such episodes were normal. A trial of carbamazepine 300 mg daily for two weeks on the remote possibility that these episodes were temporal lobe epileptic attacks was not effective. Frequently, laboratory and radiological investigations were postponed at the last moment because of her poor cooperation. However, eventually a trial of thioridazine 25 mg three times daily for six weeks reduced her psychiatric symptoms sufficiently to make investigations possible. Cerebrospinal fluid (CSF) and blood Wassermann and Kahn reactions were normal and the CSF protein and gammaglobulins were normal as was the white cell count. The blood and CSF cultures were negative.

An aortogram showed a tumour blush on the right side in the region of the adrenal gland. At laparotomy an encapsulated tumour was found tucked between the

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