narcotic activity by structural modification (Kastin *et al*, 1976; DeWied *et al*, 1978). Although Jacquet has stated that in rats both the analgesic and catatonic effects of intraventricular B-endorphin were mediated by naloxone-sensitive receptors it remains possible therefore, that endorphins could be involved in psychotic states even where naloxone effects appear to be absent. In this respect, it may be significant that in one study which claimed naloxone induced changes in other psychotic symptoms, the characteristics of the effect differed from the acute blockade of exogenous opiates and was obtained only in a subpopulation of subjects (Watson *et al*, 1977).

It would seem prudent, therefore, not to consider failure of naloxone antagonism as sufficient cause to exclude an association between endorphin activity and psychotic symptoms, including catatonic ones, until other types of evidence are available.

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ERRATUM

It is regretted that in the letter by Maurice W. Dysken and John M. Davis (*Journal*, November 1978, **133**, 476) naloxone was misspelt naxolone.

STRESS AND HYPERCALCAEMIA

DEAR SIR,

I would like to report the case of Mrs X, 45-years-old, who was admitted in a state of organic confusional psychosis on the day of the funeral of her mother. She had no history of past psychiatric illnesses, had uncomplicated rheumatic fever when 12 years old and toxaemia of pregnancy when pregnant with her second and youngest son (9 years old). There is a positive family history of depressive psychosis amongst first and second degree relatives. Her physical examination showed no abnormality. Her initial investigations, including a urine analysis, VDRL and RPCFT, blood count, fasting blood glucose and ECG, were normal. Vickers series revealed hypercalcaemia and hypophosphataemia. There was no history of vitamin D or alkali ingestion. Further investigations were done, including Bence Jones protein analysis, urine electrophoresis, thyroid function tests, plasma protein electrophoresis, thyroid stimulating hormone estimation, skull, chest and hands X-rays, IVP proved to be normal. The blood parathyroid hormone estimation was 3.7 ng/ml (N = up to 1 ng/ml). From this overall picture a diagnosis of primary hyperparathyroidism was confirmed. Her mental state which was fluctuant in course improved on prescribing chlorpromazine, which was changed to haloperidol due to apparent oversedation. Complete recovery in the fourth week of admission was concurrent with the return of serum calcium concentration to normal (see table I over).

Surgical exploration after discharge proved the presence of parathyroid adenoma which was then removed.

Anderson and Lindholm (1967) reported cases of hyperparathyroidism who presented with clear mental symptoms due to hypercalcaemic crises. In the case of Mrs X her mental dysfunction was associated with only mild hypercalcaemia of less than 3.75 mmol/l (Zilva and Pannall, 1975) on top of a quiescent parathyroid adenoma; and was triggered off by the stress of bereavement. Stress can bring changes in internal milieu with resultant symptoms or disorder (Rees, 1976). The disorder in Mrs X's case was that of organic confusional state, its content was coloured by the bereavement nature of the stress.

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TABLE I

Day of blood sample intake after admission	Serum Ca. (N = 2.2–2.7 mmol/l)	Serum Albumin (N = 35–47 mmol/l)	Serum Phosphate (N = 1.0–1.5 mmol/l)
Second Day	3.07 mmol/l	46 mmol/l	0.82 mmol/l
Ninth Day	3.08 mmol/l	41 mmol/l	—
Third Week	2.99 mmol/l	44 mmol/l	0.95 mmol/l
Fourth Week	2.66 mmol/l	—	0.6 mmol/l

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