DEAR SIR,

I refer to the letter on lung cancer in long-stay patients, in the January 1979 issue of the *Journal*, **134**, 128. Jancar and Jancar reviewed 1,125 deaths over a 40 year period in Stoke Park Hospital, Bristol. Of these 81 were caused by cancer and 3 of these were of the respiratory system. In a 25 year review (Primrose, 1966) of over 4,000 long-stay patients in Lennox Castle Hospital, Glasgow, out of 764 deaths 79 had cancer. Fifteen of these were of the lungs and a further 3 of the upper respiratory system, (mouth, pharynx, larynx). Both these hospitals are for mental defectives and so only a small proportion of the patients have schizophrenia.

With regard to smoking, it is only recently that spending-money for patients has increased significantly in mental deficiency hospitals, and 10 years ago in this hospital, which then had over 1,300 patients (now 1,200) the pocket money from hospital funds was sufficient for only 20 cigarettes per week.

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Reference

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RED CELL AND SALIVARY LITHIUM LEVELS

DEAR SIR,

The substitution of salivary lithium measurements for the accepted serum assays in the monitoring of lithium therapy has been a topic of recent interest (4, 5). However, the individual variability in the saliva:serum relation is too high for safe clinical use, but it occurred to us that the saliva:erythrocyte relation might be better (1, 3).

Thirty synchronous samples of blood and saliva were taken before the morning dose of drug from nine subjects at varying stages of lithium therapy. The patients rinsed their mouths out four times with tap water and chewed a piece of paraffin wax for three to four minutes; they were instructed to swallow the saliva produced during this period. They then spat into a container several times until 2-3/ml of colourless froth-free saliva were collected. Specimens were diluted 1:10 with deionized water and measured in an atomic absorption spectrophotometer by the method of Hisayasu *et al* (2).

The lithium concentration within the erythrocyte was calculated from values in whole blood, and in plasma with the hematocrit. A high degree of scatter was seen when RBC lithium was plotted against salivary lithium (see figure). We found a stronger correlation between the plasma and salivary lithium values, but like Sims *et al* an unacceptably high individual variation. So, even if one were to accept the viewpoint that RBC lithium levels are a better index than plasma levels for the monitoring of lithium therapy, salivary lithium assessments would still be of little clinical use.

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NALOXONE IN AMYLOBARBITONE-RESPONSIVE CATATONIA

DEAR SIR,

Dysken and Davies (*Journal*, November 1978, **133**, 476) reported a single case where the intravenous injection of naloxone failed to modify a catatonic state in man. They interpreted this finding as evidence that endogenous B-endorphin was not involved in producing the catatonic symptoms. This may be so, but it seems worth making the general point that the inference that opioid peptides are, or are not, involved in behavioural states in man from the response to a 'pure' opiate antagonist is not necessarily straightforward.

Opiate receptors were said to differ in affinity for opiate peptide ligands and in degree of stereospecificity and susceptibility to antagonists (Lord *et al*, 1977; Jacquet *et al*, 1977). Other workers have reported that the behavioural and 'neuroleptic-like' effects of endorphins can be dissociated from their

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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