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