

in bone, but there still remains the possibility that the rapidly exchanging pool was expanded independently of the other fractions of sodium in bone, and fell by the calculated 180 mEq on recovery.

(3) The changes in behaviour of the two isotopes could be due to alteration in the characteristics or amounts of macromolecules with ion 'binding' capacities in the extracellular space. If this were so the relative masses of sodium and possibly chloride (or bromide) in the tissues held in part in association with these molecules (Manery 1966) might differ as between ill and well phases.

(4) The observations could indicate an alteration in the concentration of sodium in the cells.

Of the above explanations, it is obvious that either of the latter two, if valid, could have aetiological significance, and this might hold even if the central nervous system were not involved in similar processes.

However, even allowing for the recent work of Naylor *et al* (1971), there is no direct evidence to indicate which of the above (or other) interpretations is correct, so that until the situation is clarified the question must remain open.

One further point in the evaluation of studies of electrolytes and water metabolism in affective disorders needs clarification.

The report *Biochemical Research in Psychiatry* refers to the 'conceptual difficulties in the way of ascribing a causal role to this type of electrolyte change', and to the fact that 'patients with cardiac failure or hepatic cirrhosis have much larger distortions of the electrolyte household than any described in depressed patients; yet these are not associated with any consistent disturbances of behaviour'. The report goes on to mention the lack of firm data on electrolyte content of the brain in psychiatric patients, but it could be pointed out that such data are also lacking in the physically ill individuals. Until this information is available in both groups it is too soon to speak of a 'conceptual difficulty' in ascribing a causal role to the electrolyte findings in depression.

It is likely that the affective disorders are a complex syndrome the susceptibility to which is determined by genetic endowment. As yet the events leading up to the manifestation of these periodic illnesses have not been defined. The electrolyte changes, whatever they are, could be parallel manifestations of a common cause. They could be secondary yet significant in that they could play a part in the perpetuation of the illness. They could be secondary in every way, or could be a link in the aetiological chain of events leading up to these illnesses. Any of these possibilities may be correct, and in the meantime those who are working in the field will continue to regard the electrolyte changes, not in terms of a crude unitary

causal hypothesis but as coexisting with other possible aetiological factors operating in the various forms of affective illness. If electrolyte changes are eventually shown to have aetiological significance these will have to be integrated at some level with other data and in particular with amino acid and amine metabolism.

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REFERENCES

- COPPEN, A. and SHAW, D. M. (1963). 'Mineral metabolism in melancholia.' *Brit. med. J.*, *ii*, 1439-44.
- — —, MALLESON, A., and COSTAIN, R. (1966). 'Mineral metabolism in mania.' *Brit. med. J.*, *i*, 71-5.
- — —, and MANGONI, A. (1962). 'Total exchangeable sodium in depressive illness.' *Brit. med. J.*, *ii*, 295-8.
- GIBBONS, J. L. (1960). 'Total body sodium and potassium in depressive illness.' *Clin. Sci.*, *19*, 133-8.
- HOWE, C. T. and EKINS, R. P. (1963). 'The bromide space after the intravenous administration of ^{82}Br .' *J. nucl. Med.*, *4*, 469-79.
- MANERY, J. F. (1966). 'Connective tissue electrolytes.' *Fed. Proc.*, *25*, 1799-1803.
- MEDICAL RESEARCH COUNCIL (1970). *Biochemical Research in Psychiatry: Survey and Proposals*. London. H.M.S.O.
- NAYLOR, G. J., McNAMEE, H. B., and MOODY, J. P. (1971). 'Changes in erythrocyte sodium and potassium on recovery from a depressive illness.' *Brit. J. Psychiat.*, *118*, 219-28.
- SHAW, D. M. and COPPEN, A. (1966). 'Potassium and water distribution in depression.' *Brit. J. Psychiat.*, *113*, 269-76.
- VEALL, N. and VETTER, H. (1958). *Radioisotope Techniques in Clinical Research and Diagnosis*. London. Butterworth & Co. (Publishers) Ltd.

LITHIUM TREATMENT: PROPHYLACTIC OR COMPENSATORY?

DEAR SIR,

The methodological problems of evaluating the claims for lithium in recurrent affective disorders may be resolved if we substitute the concept of 'compensatory therapy' for prophylaxis. The term prophylaxis connotes 'guarding from or preventing disease'. Neither our own studies with lithium/nor evidence in the literature demonstrate prevention of recurrent affective disorders.

After the introduction of neuroleptic and anti-depressant drugs, I proposed the term 'compensatory therapy' (2) because the psychoactive drugs indicated that once improvement of symptoms of affective and schizophrenic disorders had been achieved it could be maintained by continuous medication. This put psychiatric drug treatment in the realm of compensatory methods of treatment comparable to those

used in endocrine, cardiovascular and many other disorders, where drug treatment aims to restore and preserve the patient's functional adequacy.

Lithium does not abolish the clinical and psychosocial evidences of recurrent affective disorders. It diminishes the severity of symptoms to varying degrees and reduces the amplitude of mood swings to a point where hospitalization can be avoided. Actually, most of the reported statistics concerned with prophylactic effects concern reduction of frequency or length of hospitalization. While hospitalization may be taken as a global measurement of the severity of affective episodes, clinical evaluation must also take into account the less severe mood fluctuations which continue during lithium therapy.

It is well known that lithium is frequently combined with antidepressant drugs during depressive cycles to maintain patients' ambulatory status. Similarly, neuroleptic drugs may have to be added to control rapidly emerging manic disturbances to maintain the patients' functional balance. Neither clinical nor psychosocial patterns indicate that lithium prevents affective disorders to the point where evidence of illness disappears.

Whatever the pharmacological action of lithium may turn out to be, it appears to interact effectively with an ongoing biochemical disorder, counteracting its socially and clinically disruptive manifestations. I believe that this is best conceived of as compensatory therapy rather than as prophylaxis.

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REFERENCES

1. FREYHAN, F. A., MAYO, J. A., and O'CONNELL, R. 'Clinical evaluation of the treatment of recurrent affective disorders with lithium carbonate.' *International Pharmacopsychiatry*. Karger, Basel. In press.
2. — F. A. 'The evolution of compensatory therapy with drugs in modern psychiatric practice.' (In *Neuropsychopharmacology*. Elsevier Publishing Company, Amsterdam, 1959.)

CRIMINALITY AND VIOLENCE IN EPILEPTIC PRISONERS

DEAR SIR,

Once again, by use of a biased sample, a paper has been published purporting to show that, 'it is clearly incorrect to think of epileptic prisoners as being especially violent' (*Journal*, March 1971, p. 337). Although reference is made to the possibility of the sample being unrepresentative in not including patients in Special Hospitals, broad conclusions have

been drawn which, while strictly true for the sample chosen, do nothing to elucidate the problems of epilepsy and violence.

The definition of the Hospital Order (M.H.A., Section 60) includes the terms 'A patient convicted . . . of an offence punishable on summary conviction with imprisonment . . . etc.' Thus the Mental and Subnormality Hospitals, as well as the Special Hospitals, must contain many individuals who, but for the Hospital Order, would be in prisons.

In Rampton in 1968 there were 138 known male epileptics, representing 20% of the males in that institution. Of these, 11 committed property offences chiefly and 127 were violently aggressive and assaultive, leading to deaths on four occasions. It was apparent in reading the records that in many cases deaths had only narrowly been averted.

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CLASSIFICATION OF DEPRESSED PATIENTS: A CLUSTER-ANALYSIS-DERIVED GROUPING

DEAR SIR,

In his paper (*Journal*, March 1971, page 275) describing cluster-analysis-derived groups of depressed patients, Dr. Paykel rightly observes that few studies using factor analysis have seriously explored the possibility of more than two groups. He also comments that previous factor-analytic studies suggested a simple division of depressives into two polar types rather than more complex classifications.

Since the implications for methodology could be considerable, I wish to point out that at least one previous factor-analytic study went further than a simple dichotomy and proposed a multiple-group classification. In a factor-analytic study of 126 depressed patients seen in general practice (1), two clear-cut groups of patients, one endogenous and one non-endogenous, were found when the patients were distributed on one factor; on another, virtually independent, factor there were *three* patient groups: phobic-anxious, (non-phobic) anxious, and non-anxious. Two other factors, identifying reactive depression and general severity respectively, did not serve to distinguish patient groups. These results acknowledge a diversity of neurotic sub-groups independent of a primary division of patients into endogenous (or psychotic) and non-endogenous.

This multiple-group classification was obtained by factor-analytic methods (including calculation of factor scores) only when the number of variables