British Journal of Psychiatry (1985), **146,** 99–104

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.**

DEPRESSION AND AFFECT-LADEN WORDS

DEAR SIR,

Dr Firestone (*Journal,* October 1984, **145,** 447) points out a potential flaw in our experimental procedure whereby we found differences between depressed and control subjects in the influence the hedonic tone of material brought to bear on the accuracy of its subsequent recognition (*Journal.* April 1984, **144,** 376–382). He suggests, in effect, that the "intensity of affect" attaching to the unpleasant material was greater than that of the pleasant material, and that this explained its readier recognition.

We fully accept that the intensity of affect was greater in the unpleasant material. If the original sematic differential groupings are considered (Broadbent D. E. and Gregory M., 1967, *Nature* **215,** 581–584) then the following values are seen:– bad words – class boundaries 3.09–1.00, mean score 1.54; good words – class boundaries 7.00–4.80, mean score 5.89

Thus, the unpleasant material was associated with more intense affect than the pleasant material, but only to a small degree.

However, we do not accept that this explains our findings. The depressed subjects established stronger memory traces for the unpleasant material whereas the non-depressed controls showed precisely the reverse (Table V of our paper). If, as Dr Firestone postulates, intensity of affect is the sole explanation, one would then be in the curious situation that intensity of affects operates in the expected direction for depressives, but in the reverse direction for healthy controls. This would surely be unlikely.

A more parsimonious explanation is made in our paper. Thus, recognition performance is dependent on both the type/intensity of affect associated with the material and the mood state of the subject. If a congruence exists between the two, recognition is enhanced, when compared with a situation where this congruence is lacking.

W. A. LISHMAN
G. C. DUNBAR

Institute of Psychiatry,
London SE5

THE USE OF LITHIUM IN SEVERELY DEMENTED PATIENTS WITH BEHAVIOURAL DISTURBANCE

DEAR SIR,

The problem of aggressive, overactive and agitated behaviour in the severely demented is common and difficult to manage. The usual approach is that of chemotherapy with neuroleptic medication which is not ideal for these behaviour problems. They have undesirable side-effects, especially in the elderly who, when brain damaged, are also prone to tardive dyskinesia.

Lithium has been used in mentally retarded patients with hyperactive and aggressive behaviour, (Souver & Hurley, 1981; Goetzl *et al,* 1977). While the response may be because the behaviour stems from accompanying affective illness, it may be that lithium has a specific anti-aggressive effect (Sheard *et al,* 1976). In the elderly, lithium has been reported to be dramatically and rapidly effective in eight out of ten patients with organic brain syndrome, none of whom had been diagnosed as manic depressive (William & Goldstein 1979). We carried out a pilot study on the feasibility of using low dosage lithium in patients suffering from severe chronic brain syndrome with aggressive, overactive behaviour or agitation normally requiring a neuroleptic to control.

Ten patients were selected, with an age range of 72–85 years. Each patient had severe dementia with difficult behaviour. Seven patients completed the study. The other three became physically unwell during the trial and the lithium was therefore discontinued, although in none was lithium thought to be responsible. Neuroleptic medication was

stopped but hypnotics permitted. Assessments were made using a dementia scale, the Crichton Behaviour Rating Scale and the Plutchik Geriatric Scale. (Plutchik *et al*, 1970). Lithium carbonate was started at a dose of 250 mgs. at night. Serum levels were checked weekly. At the end of the four week trial period the patients were reassessed on the three rating scales.

In our study, lithium was not found to be effective. This could be ascribed to the low dose used or the limited length of the study. To investigate the usefulness of lithium in this group more thoroughly, it would be necessary to carry out a double blind study with higher doses over a longer period of time. However, we encountered a number of problems which would make such a study difficult to carry out. Many patients with severe dementia have co-existing physical problems, and are on drugs (for example, diuretics) which may complicate lithium administration. Lithium tablets are rather large and difficult to administer, regular blood samples must be obtained, and co-operation in such patients can be difficult to obtain.

Another factor which could account for our failure to detect any change could be the lack of specificity of the rating scales employed.

ANTHONY HOLTON
KURUVILLA GEORGE

St. Nicholas Hospital,
Newcastle-upon-Tyne. NE3 3XT

References

GOETZL, U., GRUNBERG, F. & BERKOWITZ, B. (1977) Lithium carbonate in the management of hyperactive aggressive behaviour of the mentally retarded. *Comprehensive Psychiatry*, **18**, 599–606.

PLUTCHIK, R., CONTE, H., BAKUR, M., GROSSMAN, J. & LEHMAN, N. (1970) Reliability and validity of a scale for assessing the function of geriatric patients. *The Journal of American Geriatric Society*, **18**, 491–500.

SHEARD, M. H., MARINE, J. L., BRIDGES, C. I. & WAGURE, E. (1976) The effect of lithium on impulsive aggressive behaviour in man. *American Journal of Psychiatry*, **133**, 1409–1413.

SOUVER, R. & HURLEY, A. (1981) The management of chronic behaviour disorders in mentally retarded adults with lithium carbonate. *The Journal of Nervous and Mental Diseases*, **169**, 191–195.

WILLIAM, K. H. & GOLDSTEIN, G. (1979) Cognitive and Affective responses to Lithium in patients with organic brain syndrome. *American Journal of Psychiatry*, **136**, 800–803.

SPONTANEOUS SEIZURES AFTER ECT

DEAR SIR,

James and Simpson (*Journal*, September 1984, **145**, 337–338) questioned how unusual it is for another tonic-clonic seizure to occur within a minute of ECT, and whether this seizure is best considered a spontaneous seizure or continuation of the original electrically induced seizure. It is known that ECT-induced epileptiform EEG activity can persist more than one minute following cessation of clinically observed tonic-clonic movements (e.g. Christensen & Koldbaek, 1982; Maletzky, 1978). Without EEG monitoring (Sørensen *et al*, 1981; Staton *et al*, 1981), it is therefore difficult to tell whether the clinical seizure James and Simpson observed beginning one minute post-ECT was continual ECT-induced seizure activity or a separately occurring spontaneous seizure.

Continual ECT-induced seizure activity in a patient was reported by Weiner *et al*, (1980). In this patient, ECT produced a clinical seizure lasting less than 60 seconds. However, EEG monitoring demonstrated that sustained EEG epileptiform activity persisted for several minutes, after which a 45 second period of clonic movements involving the rostral half of the body appeared. Diazepam I.V. was used to terminate the seizure. Weiner *et al*, speculate that the reappearance of clinical seizure activity was concurrent with metabolism of the succinylcholine administered before ECT. The times given in the Weiner *et al*, case are similar to those given in the James and Simpson case; continual ECT-induced seizure activity could therefore easily have been occurring in the James and Simpson case.

The other possibility that James and Simpson mention (i.e., that a spontaneous tonic-clonic seizure occurred after termination of the ECT-induced seizure) is also plausible. Such a case was reported by Weiner (1981), who states (p. 1237): "The ensuing (ECT-induced) seizure, monitored by EEG, initially appeared to terminate after 1.5 minutes, but it was followed by intermittent bursts of 1-2 Hz paroxysmal slowing superimposed on faster background activity . . . 10 minutes after stimulation, while Mr. A remained unconscious, the paroxysmal activity became continual. It rapidly progressed into a 30-minute period of status epilepticus, which included two tonic-clonic clinical convulsions and a third seizure that was present only electrographically. The seizure activity eventually aborted after a total of 10 mg of diazepam i.v."

Regarding the question of how unusual it is for a second clinical seizure to occur shortly after ECT, the answer from existing literature (a survey asking this question would be useful) has to be that such an occurrence is rare (Strain & Bidder, 1971; Blachly, 1976; Blackwood *et al*, 1980; Weiner *et al*, 1980; Weiner, 1981). It has been suggested that the occurrence of prolonged seizures (i.e., 5-10 minutes