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.

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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

duration) has been under-reported due to the lack of EEG monitoring of seizures (Weiner *et al*, 1980). Nevertheless, the occurrence of either a prolonged seizure or spontaneously occurring seizures should be treated expeditiously, perhaps with anti-convulsant medication and immediate consultation with a neurologist. (c.f., Strain & Bidder, 1971; Weiner *et al*, 1980; Weiner, 1981).

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SUICIDE IN HOSPITAL

DEAR SIR,

I read with great interest your recent Symposium on Suicide in Hospital (*Journal*, November 1984, **145**, 459–476). I recently carried out a small retrospective survey on all known in-patient suicides at Fulbourn Hospital over the five year period 1979-84. There were fourteen such suicides in a 455 bed mental hospital serving a catchment area of 570,000, giving a rate of 0.49 per 100,000 per year of the general population which seems similar to that found by Langley and Bayatti (*Journal*, November 1984, **145**, 463–467).

I would like to raise two points from the study. Firstly, I found that ten out of the fourteen had made previous attempts and that in seven cases this involved the use of a violent method. In seven cases (six using a violent method) the attempt was just prior to or during admission. This suggests that attempted suicide by a violent method is an important risk factor in the assessment of in-patient suicides where death by overdosage is more difficult to accomplish. Secondly, I found that in twelve of the fourteen cases the medical notes contained as their last entry little more than a note of the patient's death or of details of attempted resuscitation (the nursing notes were far more complete). This may have resulted from a wish to deny the patient's death or perhaps more simply because the doctor becomes too busy at the time, forgets or thinks it of no value.

Regular audit would improve this practice which, if it is widespread, would make retrospective surveys more difficult.

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DIAGNOSTIC ISSUES IN THE HYPERVENTILATION SYNDROME

DEAR SIR,

We would like to take issue with the methods used by Kraft and Hoogduin (*Journal*, **145**, 538–542) to establish the diagnosis of hyperventilation syndrome (HVS). They remark that a patient had to be suffering from "at least 18 out of 45 complaints commonly associated with the hyperventilation syndrome" to qualify for inclusion in their study. The symptoms in their checklist are so non-specific that conclusions based on patient groups satisfying these criteria are bound to be tentative. Grossman and de Swart (1984) have already demonstrated that reported complaints are an unreliable guide to the diagnosis of HVS.

No clinician would diagnose diabetes without measurement of blood sugar: similar objective measures should be used to establish a diagnosis of HVS. Hyperventilation implies arterial hypocapnia. In patients with normal lung function it has long been accepted that end-tidal (equivalent to alveolar) pCO_2 (or $PACO_2$) is very close to arterial pCO_2 ($PaCO_2$) (Bannister *et al*, 1954). We believe that objective measurement of $PACO_2$ is essential before establishing a diagnosis of hyperventilation.

Diagnostic issues are important for two reasons. Firstly, the array of symptoms in HVS and panic disorder is similar (Bass & Gardner, 1983). Diagnostic criteria for panic disorder have been established (American Psychiatric Association,