disappeared after the fourth treatment. A bilateral electrode position had been resumed after the second diagonal ECT.

The patient's subjective impressions were as follows:

1. She experienced slight periods of 'confusion' after coming round from her treatments. This was worse after the second (diagonal) treatment, when she felt 'totally confused for thirty six hours'. The third (bilateral) ECT was described as a 'milestone', by which she meant that she felt considerably improved.
2. After the second treatment she became 'almost completely deaf', her vision became 'very peculiar' and she felt that objects 'jumped' and 'swam'. She described feeling 'extremely frightened'.

The auditory and visual symptoms were not associated with any objective signs on clinical testing; e.g. reduced hearing or nystagmus. These disturbances together with the memory impairment had subjectively resolved within three weeks of the last diagonal ECT.

It is tempting to speculate that the visual dysfunction was related to the close proximity of one electrode to the occipital cortex. We report these incomplete observations as providing probable evidence of the adverse effects of this form of ECT.

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ECT AND CEREBRAL DAMAGE

DEAR SIR,

We feel that Professor Kendell, in his review of the present status of electroconvulsive therapy, overstates the case when he says that there is no evidence from animal studies that electrically induced convulsions produce cerebral damage.

The results from two animal studies (Ferraro *et al*, 1946; Hartelius, 1952) indicate that there are structural changes in neurones and glial cells, especially in the frontal area, following electrically induced convulsions, and that the degree of damage is proportional to the number of convulsions received.

Computerised tomography has now provided a non-invasive method of examining structural changes in vivo. Weinberger *et al* (1979) performed CT scans on 75 chronic schizophrenics and found that in 17 ECT-treated patients there was significantly more cortical atrophy than in 58 patients who had not received ECT. In 41 elderly depressives who had CT scans as part of an earlier study (Jacoby and Levy, 1980) we found a statistically significant association between frontal lobe atrophy and previous treatment with ECT (Calloway *et al*, 1981).

In view of these findings, a comprehensive study to investigate the association between ECT and cortical atrophy is being undertaken in the Academic Department of Psychiatry, the Royal Free Hospital.

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POST OPERATIVE DEPRESSIVE STUPOR REQUIRING ECT

DEAR SIR,

It is well known that minor psychiatric complications may follow surgery, but occasionally a severe psychiatric illness can occur. Knox (1961) estimated that 1 in every 1600 surgical procedures was followed by a severe disturbance of which one third were due to depression.

We would like to report what we believe is a unique case—of a severe depressive stupor occurring within a few days of surgery where it was necessary to administer electroconvulsive therapy (ECT).

Case report

A 71-year-old man, who had no previous psychiatric illnesses, presented with dysphagia due to carcinoma of the oesophagus. He was admitted to hospital and an oesophagectomy was carried out. For the first three days postoperatively he appeared quiet and withdrawn but fully orientated. This state rapidly worsened and by the fifth day after surgery he was mute and unresponsive, although

obviously conscious. He began to deteriorate to such a degree that the lack of coughing and movement rendered pneumonia likely and his life was thus very seriously at risk. At this stage it was difficult to determine the aetiology of his stupor. During an injection of intravenous diazepam a brief conversation elicited that he was well orientated and that he had delusions of worthlessness and auditory hallucinations where the other patients discussed his sins. All this was consistent with a severe psychotic, depressive stupor.

Intravenous antidepressants were started but as by the 10th post-operative day he still appeared to be deteriorating he was given ECT. Following this he was more responsive and spoke a few words. After the second treatment, although still depressed, he began to talk spontaneously and was able to sit out of bed and to eat. Oral antidepressants were substituted and he had one further ECT following which he was able to converse normally and participate in his rehabilitation. At the time of his discharge four weeks after surgery he had made a complete psychological and an almost complete physical recovery.

We believe this case is unusual in that there has been no report of a case of depressive stupor arising at such an early stage after surgery. The necessity to use ECT so soon after a major surgical procedure is also unique. A recent case study (Ries and Bokan, 1979) reported using ECT 30 days after pituitary surgery in a patient with depressive stupor where the risk of physical deteroiration was high. In some ways this case was similar to our own, but the patient had in fact suffered from a psychotic depression related to her Cushing's disease prior to surgery, unlike our patient.

Although ECT has been used for many years now in the treatment of psychiatric disorders its exact place as a therapeutic agent is still debated (Palmer, 1981). In this case, however, although the risks of ECT were probably higher than is usually the case, it does appear that it was effective and almost certainly life saving.

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ANXIETY IN EGYPT

DEAR SIR,

In their study of anxiety in Egypt (*Journal*, July 1981, **139**, 70–3) Okasha and Ashour claim significant differences in some presentations, but their methodology allows for questioning the validity of their findings.

The cluster of symptoms used to diagnose anxiety was stretched beyond their definition of an 'anxiety state'. The inclusion of fugues and amnesia, dissociative and conversion symptoms, as well as specific phobias, hypochondriasis, obsessional checking and depersonalisation suggests the possibility of other neurotic disorders being present. Could it not be the case that they were studying other neurotic disorders, some of which are known to be significantly different from each other but could each include anxiety as a symptom?

Both the ICD-9 and PSE are clear about their definitions of various neurotic disorders which are often mutually exclusive. However, patients can still present with mixed neuroses but then the most prominent features will favour a specific diagnosis. Specific phobias, fugues and amnesia, dissociative and conversion symptoms are more expected to point to a diagnosis other than anxiety.

To diagnose anxiety, I understand, the authors applied 52 out of the 140 symptoms included in the PSE symptom list. They assessed symptoms like organic impairment of memory, subjective euphoria, ideomotor pressure, and grandiose ideas and actions. Some of these latter symptoms do not appear under any of the PSE syndromes of neurotic disorder.

Anxiety may present as a symptom, trait or state when the implications for treatment and outcome may differ. A comment on management and prognosis might have clarified the situation further.

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ABNORMAL INVOLUNTARY MOVEMENTS IN THE ELDERLY

DEAR SIR,

A. J. Blowers and colleagues (*Journal*, October 1981, **139**, 363–4) claim their study "has shown that in a group of elderly subjects . . . antipsychotic drugs . . . increase the risk of developing dyskinesias". Surely not. What their study has shown is that those elderly subjects who had been selected for treatment with antipsychotic drugs had a significantly higher prevalence of abnormal involuntary movements than those who had not been so selected.

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