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ECT anaesthetics

SIR: I read with interest the audit of ECT in two NHS regions by Pippard (*Journal*, May 1992, **160**, 621–638) which was a fascinating follow-up to his earlier work. As an anaesthetist with responsibility for provision of anaesthesia to an ECT treatment unit, I would like to comment on one small point. Dr Pippard states quite correctly that the use of propofol is probably contraindicated for ECT, because it reduces seizure duration. Therefore methohexitone remains the agent of choice, particularly in view of its proconvulsant potential. Unfortunately, the injection of methohexitone is often painful, and repeated anaesthesia with this agent can be distressing for some patients. Dr Pippard stated that the use of 10 mg lignocaine with anaesthetic induction agents is not recommended because of the potential anticonvulsant activity of this local anaesthetic. The use of lignocaine to modify the pain of injection of methohexitone during anaesthesia for electroconvulsive therapy has been studied (Simpson *et al*, 1989). We showed that pain on injection of methohexitone occurred in nearly half of the patients, and in a quarter of them pain was reported as moderate or severe. The use of 10 mg lignocaine before or mixed with the methohexitone reduced this significantly. We measured seizure duration using an isolated arm technique and demonstrated that the use of 10 mg lignocaine did not significantly affect seizure duration. In view of these findings I would urge that lignocaine continue to be used routinely when injecting methohexitone into a vein on the dorsum of the hand, particularly when patients are to undergo repeated anaesthesia.

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Out-patient ECT for depression in a man with moderate learning disability

There is little written on the place of ECT in the treatment of mental illness in those with learning disabilities (Lazarus *et al*, 1990). We would like to report the case of a 69-year-old man with moderate learning disability (IQ of 40).

Case report. Mr X presented with a six-week history of weepiness, lethargy, social withdrawal, weight loss, and poor sleep following an uneventful inguinal hernia repair. On examination he looked sad and described his mood as such. He spoke in monosyllables and complained of aches and pains in his feet (for which no organic cause was found). He had a past history of depression, following a previous inguinal hernia repair at age 61 years, which responded to tricyclic antidepressants. Otherwise he had always been in good health. A diagnosis of depression was made. Dothiepin, 50 mg daily, was started, increasing to 150 mg daily. However, he became more agitated and distressed. He was preoccupied with being unable to swallow, believed he had no throat and ate very little. He lost over 13 kg (two stones) in weight. His self-care skills deteriorated and he became doubly incontinent. The dothiepin was discontinued after two months and replaced with fluoxetine, 20 mg daily, increasing to 40 mg daily with no improvement after one month. At this stage his permission to commence ECT was sought and given. He received 11 bilateral treatments. After the first treatment a marked improvement in his appetite was noted with a reduction in his agitation. This continued to improve, as did his sleep; his bodily preoccupations and delusions then faded, followed lastly by a reduction of his social withdrawal. Three months later he is considered to be his 'old self' and is on fluoxetine, 40 mg daily.

This case adds to the small literature on the use of ECT in those with learning disability and mental illness. The majority of the cases described are, as is this one, of major depression. Goldstein & Jensvold's (1989) case bears particular similarities in that their patient's depression was seemingly precipitated by a surgical procedure. Kearns (1987) described a case of a man with learning disabilities with Cotard's syndrome who responded to ECT. In the present case the patient similarly denied the existence of part of his body (his throat). Our patient had ECT as an outpatient. Lazarus *et al* (1990) advocate the use of this,

emphasising the financial savings it allows. Outpatient ECT was chosen in our case because given that the physical examination, routine blood tests, chest X-ray and electrocardiogram were normal, it was felt beneficial for the patient to be spending most of his day among those he knew, particularly as hospital admission had contributed to his present mental state.

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Are polioviruses a cause of schizophrenia?

SIR: The thought-provoking commentary by Eagles on a possible role of polioviruses in the development of schizophrenia (*Journal*, May 1992, **160**, 598–600) raises the possibility that two other closely related groups of picorna enteroviruses (Coxsackie and ECHO) may also be involved in the genesis of some instances of schizophrenia. Like polioviruses, the incidences of Coxsackie and ECHO virus infections peak during the summer, both are neurotropic (Sells *et al*, 1975), and can be passed from mother to foetus (Modlin *et al*, 1981). Poliovirus sensitivity genes may encode several subunits of the membrane-bound picornavirus-receptor complexes that are members of the immunoglobulin super family, some of which may be identical to intercellular adhesion molecules (see Selinka *et al*, 1991). The picornavirus receptor subunits might be combined in various ways to yield multimeric receptor complexes that are more or less specific for certain viruses, but with some overlap. For example, Siddique *et al* (1988, cited by Eagles) found that several rodent–human hybrid cell lines, infected with appropriate fragments of human chromosome 19, could subsequently be infected by both poliovirus 1 and ECHO virus 11. If later reinfection is required for the full development of schizophrenia, as Dr Eagles suggests, the reinfection might therefore also be by one of the Coxsackie or ECHO viruses, in previously sensitised and immunocompromised individuals. The idea of required reinfection receives support from the epidemiological findings of Hare & Walter (1978) on the seasonal variations in the admissions of schizophrenic and manic patients with peaks in July and August, corresponding roughly to the peaks in the incidence of all three enterovirus infections. The somewhat earlier peaks in psychiatric admissions may be due to pre-existing sensitisation of these patients, with most non-sensitised individuals presenting with other symptoms of viral infection 1 to 3 months later. The peaks of enterovirus infections are followed, approximately six months later, by peaks in excess births of individuals who will, 25 to 30 years later, develop schizophrenia or mania. These findings suggest that infected mothers may pass a psychotogenic enterovirus to their foetuses during the first or second trimester of pregnancy when the foetal brain is most sensitive to infective damage, as Dr Eagles points out. Such early viral infection may result in selective destruction of excitatory glutamatergic and cholinergic neurons in the foetal brain (Squires & Saederup, 1991) as well as causing the foetus' immune system to treat the invading viral antigen as non-foreign, leading to permanent sensitivity to the virus.

The hypothesis of *in utero* sensitisation and immunocompromise by an enterovirus predicts greatly lowered levels of antibodies to this virus in schizophrenic adults and can be easily tested by determining the relative abilities of blood samples, taken from schizophrenic and non-schizophrenic adults, to neutralize various enteroviruses *in vitro*.

Perhaps repeated reinfections of sensitised individuals, over a lifetime, may be a cause of the progressive deterioration seen in schizophrenia.

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