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Conclusion

This development has provided the opportunity for primary and secondary care services to work together in producing a guideline that will facilitate good practice. It is likely to improve patient care, minimise risk and underlines the move towards 'partnership-working' across this interface. Liaison with our information technology departments will ensure our document templates can be integrated into local systems. Audit and review of the use of these guidelines are both essential to ensure this endeavour has any clinical impact. In response to this development the Lothian lithium audit was initiated in July 2001 by the Primary Care Clinical Governance Support Team. The aim of the audit is to identify current management of patients on lithium and support practices to implement any changes in line with the Lothian lithium guidelines. Results from the first phase baseline audit have been collated and an interim report disseminated. The full report (which will include the re-audit data) will be available for dissemination in September 2002 and will inform the review of these guidelines.

Such 'joined-up thinking' is indeed fashionable at present but there will be some who are sceptical about the value of such exercises. At the very heart of this is often an ambivalence to guidelines in general. There can be worries that overly simplified or prescriptive approaches are inappropriate and unhelpful. Furthermore, there are concerns that failure to adhere to guidelines may increase clinicians' and trusts' liability to allegations of negligence. However, current thinking suggests that failure to produce and consult guidelines would be a far greater omission of care. We are certainly hopeful that this guideline in conjunction with local audits and registers of patients on lithium in general practice will make a valuable contribution to improving the health care provision for these patients. Ultimately it may also reduce costs within NHS psychiatry too.

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Declaration of interest

None.

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***Joy Nicholson** Principal Pharmacist, Lothian Primary Care NHS Trust
Brian Fitzmaurice Clinical Lecturer in Psychiatry, Jonathan Swift Clinic, James's Street, Dublin 8

For correspondence: The Clinical Guidelines Team, Lothian Primary Care NHS Trust, Stevenson House, 555 Gorgie Road, Edinburgh EH11 3LG

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SUSAN M. BENBOW, PRITI SHAH AND JOE CRENTSIL

Anaesthesia for electroconvulsive therapy: a role for etomidate

AIMS AND METHOD
Three cases are described to illustrate the elective use of etomidate in electroconvulsive therapy (ECT) anaesthesia.

RESULTS
Use of etomidate is described in an individual who was treated with an electrical stimulus at the maximum level for the ECT machine in use; in a person who had severe side-effects with an alternative induction agent; and in a person with severe cardiac disease.

CLINICAL IMPLICATIONS
The anaesthetic drug should be tailored to the individual needs of the person being treated with ECT. Clinics should involve local anaesthetic departments in reviewing their anaesthetic practice.

In the UK the absence of methohexitone has led to changes in electroconvulsive therapy (ECT) anaesthesia because it was previously the anaesthetic drug of choice, and Kellner (2001) stated that it still continues as such in the USA. Advice from the College's ECT Committee concluded that there was no single alternative drug (Freeman, 1999), so practice now varies from clinic to clinic. Our anaesthetist chose to use thiopental but, for selected patients, we have found etomidate useful. We have been aware of concern about using etomidate because of its association with adrenocortical insufficiency. We describe three cases to illustrate its use. Case

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one demonstrates the use of etomidate for a man whose seizure threshold was steadily rising across the course of treatment until his treatment dose had reached the maximum output of the ECT machine, case two demonstrates its substitution for thiopental in a person complaining of adverse effects post-ECT and case three demonstrates its elective use for a woman with severe concurrent physical illness.

Case histories

Case one

Mr J., a man aged 77 years, was referred for bilateral ECT. Seizure threshold was determined at his first and second treatments using a Thymatron DGx (Somatics Inc., Lake Bluff, Illinois) according to the standard protocol in use in the clinic (Lock, 1995) and was 353 millicoulombs (mC) (treatment dose 504 mC). Thiopental anaesthesia was employed. Over treatments 3–6 electroencephalogram (EEG) seizure length shortened gradually from 34 to 15 seconds with little sign of clinical improvement, and at treatment 7 his treatment dose was increased to 756 mC with EEG seizure length timed at 53 seconds. Over treatments 7–10 EEG seizure length again steadily shortened, and the treatment dose was increased at treatment 12 to 1008 mC, maximum output for the machine in use. Feedback from the clinical team indicated that Mr J. had shown some slow but gradual improvement, and would need to continue with several more treatments. After discussion it was decided to change the anaesthetic to etomidate, which was then used for three more treatments (12–14). EEG seizure length for these three treatments was between 55 and 48 seconds. During these last few treatments Mr J. himself started to report on feedback forms that he felt better, and at the end of the course he rated himself as 'a lot better'.

Case two

Mr P., aged 62 years, was referred for ECT because of a previous good response to ECT, previous failure to respond to antidepressant drugs and severity of current depressive illness. He received 11 treatments employing thiopental anaesthesia with some improvement, but was complaining of feelings of confusion and memory difficulties after ECT. A change from bilateral to unilateral ECT was of minimal benefit. From treatment 12 his anaesthetic was changed to etomidate, with improvement in subjective side-effects, and he continued to a total of 16 treatments, by which time he was reported to be fully recovered.

Case 3

Mrs H., aged 72 years, was referred for ECT. She had known severe ischaemic heart disease, hypertension, atrial fibrillation and hyperlipidaemia, and was on warfarin following a series of transient ischaemic attacks. She had failed to respond to several courses of antidepressant drugs but had a history of successful treatment with ECT. After discussion we opted to use etomidate anaesthesia in preference to thiopental because of her major medical illness, and likely susceptibility to confusion during treatment. She went on to receive a course of eight ECT with partial recovery.

Discussion

Etomidate can lead to adrenocortical suppression and other endocrine effects. Long-term sedation has led to serious adrenocortical insufficiency in people being treated with high dose infusions over long periods, with an associated increase in mortality (Preziosi & Vacca, 1988). Wagner *et al* (1984) reported that a patient in intensive care who received a 20-hour infusion of etomidate (1.3–1.5 mg/kg body weight/hour) developed adrenocortical suppression that persisted for 4 days, whereas surgical patients receiving single dose induction were adrenocortically suppressed at 4 hours postoperation but normal at 24 hours postoperation. Crozier *et al* (1987) found that the cortisol response to adrenocorticotropic hormone stimulation was blunted postoperatively in healthy young men undergoing orthopaedic surgery at 6 hours, but normal at 20 hours. Duthie *et al* (1985), using 0.3 mg/kg etomidate, showed no suppression of cortisol at 15 minutes and 1, 4 and 24 hours postoperation and concluded that a single dose of 0.3 mg/kg etomidate causes no significant adrenocortical suppression. Wagner & White (1984) concluded that etomidate-induced adrenocortical suppression was a direct effect on the adrenal gland. For these reasons various authors have suggested that etomidate should only be used for single dose induction (Preziosi & Vacca, 1988) or short-lasting anaesthesia in minor surgery (Allolio *et al*, 1984).

Interest in etomidate as a possible induction agent for ECT anaesthesia has, however, continued because alternative intravenous anaesthetics commonly used for ECT possess dose-dependent anticonvulsant properties. Avramov *et al* (1995) described 10 people treated with maintenance bilateral ECT who underwent a prospective randomised crossover study that compared methohexitone, propofol and etomidate at low, intermediate and high doses. EEG and motor seizure durations were longest after etomidate induction and shortest after propofol. There were no significant dose related differences using etomidate, whereas methohexitone and propofol both produced dose-dependent decreases in EEG and motor seizure duration. Kovac and Pardo (1992) found no difference in seizure duration using methohexitone (1 mg/kg) and etomidate (0.3 mg/kg) in a prospective randomised crossover study, but more of their etomidate-treated patients experienced pain on injection: the incidence of pain decreased when 35% propylene glycol was added to etomidate as a solvent. Gran *et al* (1984) did not find any difference in mean seizure duration using etomidate (0.3 mg/kg) and methohexitone (1 mg/kg) alternately in eight people having unilateral ECT, but reported that pain at the injection site and

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thrombophlebitis occurred frequently using methohexitone, and did not occur using etomidate dissolved in a soy bean oil emulsion. In a retrospective chart review, Saffer and Berk (1998) compared etomidate with thiopental and found that etomidate was associated with a significantly longer seizure duration. A similar study (Trzepacz *et al*, 1993) on a smaller group of patients reported the same finding and the authors noted the possibility (that they had not investigated) that longer seizure durations might enhance the effectiveness of ECT.

Ilivicky *et al* (1995) described four elderly people who became increasingly refractory to seizure induction during ECT induced with methohexitone. When the seizure duration fell below 25 seconds, etomidate was substituted for methohexitone and mean seizure duration increased by 245%. All four people completed treatment successfully.

What then is the role of etomidate in ECT anaesthesia? Avramov *et al* (1995) regarded it as a useful alternative to methohexitone and propofol for people 'achieving suboptimal therapeutic responses'. We have used it for selected patients for one of three reasons: (1) stimulus dose with alternative anaesthetic drugs maximal for the machine used in the clinic; (2) adverse reactions to thiopental; or (3) concern about the cardiac status of the patient.

Since the withdrawal of methohexitone there has been debate about optimal ECT anaesthetic practice. We believe that, as for electrical dosing, our anaesthetic practice should become more individualised and that there is a useful role for etomidate in ECT anaesthesia. We recommend that clinics currently using thiopental as their preferred anaesthetic agent should consider whether etomidate would be preferable, since it can probably be used for treatment with lower electrical doses. In addition, we recommend that all ECT clinics should review their anaesthetic practice in discussion with their anaesthetic departments and agree local protocols covering choice of anaesthetic drug.

Declaration of interest

None.

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***Susan M. Benbow** Consultant Psychiatrist with responsibility for ECT, **Priti Shah** ECT Anaesthetist, **Joe Crentsil** ECT Manager, ECT Department, Edale Building, Manchester Royal Infirmary, Oxford Road, Manchester M13 9WL

For correspondence: Penn Hospital, Penn Road, Wolverhampton, West Midlands WV4 5HN

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