Anaesthesia for electroconvulsive therapy

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Electroconvulsive therapy (ECT) is most commonly used to treat severe or medication-resistant depression, although it can also be beneficial in mania and catatonia. ECT induces a generalized, tonic–clonic epileptic seizure, yet despite being first described in 1938 the exact mechanism of action remains elusive. For almost 30 years it was performed without an anaesthetic. Now general anaesthesia is used, and although the number carried out has been decreasing thousands are still undertaken each year in the UK, often on remote sites.

This review focuses primarily on the physiological and physical responses to ECT and the rationale behind the choice of anaesthetic technique. The physical and psychological needs of patients and obtaining consent for ECT are also considered.

Administration of ECT

During ECT, an electrical current is applied transcutaneously to the brain via two electrodes positioned either bilaterally or unilaterally (Fig. 1). Bilateral ECT is used more commonly and is preferred when speed of clinical recovery takes priority. Unilateral ECT is performed on the non-dominant hemisphere and has the advantage of minimizing cognitive adverse effects. The overall aim of both techniques is to induce a generalized seizure with characteristic EEG changes. The optimal seizure duration remains unclear. Too short (<10 s) or too long (>120 s) may reduce clinical efficacy but other research suggests that the amount of current delivered is more important than length of seizure. Typically, ECT is performed twice weekly until there is a lack of further improvement (on average, 3–4 weeks). Maintenance ECT thereafter is not generally recommended.

Physiological and physical responses to ECT

Cardiovascular effects

The cardiovascular response is secondary to activation of the autonomic nervous system. Beginning with the electrical stimulus, there is an initial parasympathetic discharge lasting 10–15 s. This can result in bradycardia, hypotension, or even asystole. A more prominent sympathetic response follows during which time cardiac arrhythmias occasionally occur. Systolic arterial pressure may increase by 30–40% and heart rate may increase by 20% or more, generally peaking at 3–5 min. Myocardial oxygen consumption, as determined by the rate–pressure product (RPP), therefore increases. RPP increases are more marked with bilateral ECT, in older patients and during hyperventilation-induced hypocapnia. Simultaneously, seizure activity increases tissue oxygen consumption, potentially reducing myocardial oxygen supply. Myocardial ischaemia and infarction can therefore occur, particularly with pre-existing disease. Left ventricular systolic and diastolic function can remain decreased up to 6 h after ECT. Cardiac rupture has also been described.

Cerebral effects

Cerebral oxygen consumption, blood flow, and intracranial pressure all increase. There is conflicting evidence as to whether neuronal cell death occurs and if so whether this is clinically relevant. Transient ischaemic deficits, intracranial haemorrhage, and cortical blindness have all been reported. Prolonged seizures or status epilepticus are other complications.

More commonly, cognitive adverse effects occur. These may be greater in patients with pre-existing dementia. Disorientation, impaired attention, and memory problems are frequent post-ictally, and short-term memory impairment lasting several weeks occurs in more than
50% of the patients. Although this usually resolves within 6 months, permanent memory loss is possible. Both retrograde and anterograde amnesia can occur. Non-memory cognitive functions (e.g. intelligence and judgement) are unaffected. Altering the stimulus intensity or waveform, using unilateral electrode placement, and lengthening the inter-ECT interval can reduce cognitive adverse effects.

Other physiological effects
Intraocular and intragastric pressure increases but this latter effect does not appear to be clinically significant.

General physical effects
During unmodified fits, there was a high incidence of fractures and dislocations but these are now rare. Headaches, myalgia (either from the seizure or succinylcholine), drowsiness, weakness, nausea, and anorexia may all occur but symptoms are usually mild. Increased salivation can occur, as can dental damage and oral cavity lacerations.

Mortality
Despite the physiological effects, and its frequent use in elderly people with significant co-morbidity, ECT is a low-risk procedure. The mortality rate of ~1 per 10 000 patients (1 per 80 000 treatments) is similar to that of anaesthesia for minor surgical procedures. Cardiovascular (arrhythmia and myocardial infarction) and, to a lesser extent, pulmonary (laryngospasm and aspiration) complications are the most common causes of death and serious morbidity.

Anaesthetic technique and drugs used for ECT
Anaesthesia should be provided by an experienced anaesthetist. The Royal College of Anaesthetists’ guidelines for ECT provision at remote sites recommend that appropriate resuscitation equipment and drugs are immediately available and that the Association of Anaesthetists of Great Britain and Ireland’s standards for monitoring, trained assistance, and recovery facilities are met.

For ASA III or higher patients, discussion should precede any decision to provide ECT at a remote site, taking into account the individual patient needs, hazards of transfer, and contingency plans for transfer to a critical care facility if needed.

Preoperative assessment
Many patients are elderly with a variety of co-morbidities and can be poor historians if depression is severe. Although problems such as oesophageal reflux must be sought, particular conditions which may be a contraindication to ECT should be identified. None is absolute, but the physiological responses to ECT make it particularly hazardous within 3 months of either a myocardial infarction or cerebrovascular accident, or where raised intracranial pressure exists. Further relative contraindications include uncontrolled cardiac failure, deep venous thrombosis (until anticoagulated), untreated cerebral aneurysm, unstable major fracture, or severe osteoporosis, phaeochromocytoma, retinal detachment or glaucoma. Cochlear implants are also a contraindication, although unilateral ECT has successfully been used. In all cases, risks of untreated depression must be balanced against risks of anaesthesia and ECT, remembering that ECT is quicker in onset than other treatment modalities and potentially life-saving in states of catatonia when, for example, there is no oral intake.

Many psychiatric drugs have either a variety of side-effects or potential to interact with anaesthetic drugs. Selective serotonin reuptake inhibitors (SSRIs) may cause syndrome of inappropriate secretion of antidiuretic hormone, whereas nephrogenic diabetes insipidus can occur in lithium use. Fortunately, interactions with anaesthetic drugs (e.g. indirect sympathomimetics causing hypertensive crises with either tricyclic antidepressants or monoamine oxidase inhibitors; meperidine or tramadol causing serotonin syndrome with SSRIs) are uncommon as the anaesthetic drugs in question are not generally required during ECT.

Physical examination should seek evidence of cardiac failure, severe valvular disease, dysrhythmia, uncontrolled hypertension, poor dentition, or dehydration requiring fluid therapy. Blood tests, ECG, and other investigations should be performed as clinically indicated. The time taken to optimize problems has to be balanced
against the urgency of ECT. ‘Emergency’ ECT, however, usually begins within days not hours.

**Conduct of anaesthesia**

Consent should be checked, patients fasted, and regular medications administered before ECT. Sedative premedications delay emergence and interfere with seizure generation and should be avoided. Patients should be encouraged to empty their bladder as incontinence is common.

The objective of anaesthesia is to provide a rapid onset and offset of both unconsciousness and muscle relaxation for the duration of the electrical stimulus and subsequent seizure, while minimizing the aforementioned physiological and physical effects. Many anaesthetic agents however have anticonvulsant properties. The choice of drugs is therefore a balance between providing adequate anaesthesia without adversely affecting the efficacy of ECT.

**Induction agents**

The dose of agent is initially titrated to patient weight but modified thereafter as necessary depending on previous response to ECT and any changing seizure thresholds. Methohexital, which had minimal anticonvulsant properties compared with other barbiturates, was the original gold standard, but due initially to lack of availability, other hypnotic drugs became more widely used. The main advantages and disadvantages of each are described in Table 1. A recent systematic review concluded that all currently available induction agents are suitable for ECT and the small variations in emergence and recovery times should not govern drug choice. Ketamine however was not included. Using etomidate results in the longest seizure duration, and it is the only i.v. induction agent that may reduce the seizure threshold. Combining opioids such as remifentanil (1 μg kg⁻¹ over 30–60 s) or alfentanil (10–25 μg kg⁻¹) with agents other than etomidate can produce similar effects on seizure duration by an induction agent dose-sparing effect. Whichever drug is used, it is preferable to utilize the same one throughout a course of treatment to avoid interfering with the seizure threshold (which generally increases over a course of ECT).

**Neuromuscular blocking agents**

Neuromuscular blocking agents reduce muscular convulsions and decrease the risk of serious injury. Visible muscle activity and, more accurately, EEG monitoring are used to monitor seizure activity. Succinylcholine (0.5 mg kg⁻¹) is most commonly used. Larger doses up to 1.5 mg kg⁻¹ may be required, particularly in cases of severe cachexia, osteoporosis, or pre-existing skeletal injury. If contraindicated, a non-depolarizing agent can be used. Mivacurium is short acting, but seizure modification is reportedly inadequate with low doses (0.08 mg kg⁻¹) and at least 0.15 mg kg⁻¹ should be used. Rocuronium and vecuronium were generally only administered as precurarizing agents but these could theoretically be used with the advent of sugammadex.

**Adjuncts**

Several drugs have been utilized attempting to either reduce the dose of induction agent (described above) or more commonly to control the cardiovascular responses in high-risk patients. Adverse parasympathetic effects may be controlled with atropine or glycopyrrolate. Glycopyrrolate has superior anti-sialogogue effects, no adverse central nervous system effects, and results in less post-ECT tachycardia. Routine atropine premedication is not recommended due to detrimental effects on myocardial work and oxygen demand. Deleterious sympathetic effects may be controlled with β-blockers either pre- (atenolol) or intra-procedurally (labetalol and esmolol). There is controversy however regarding whether labetalol and esmolol reduce seizure duration. Calcium-channel blockers can also effectively control arterial pressure. Sublingual nifedipine and i.v. nicardipine have been used (but reflex tachycardia occurs), as has diltiazem, although it also may reduce seizure duration. Preoperative α-2 agonists such as dexmedetomidine also blunt the hyperdynamic response as does glyceryl trinitrate, which

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**Table 1** Advantages and disadvantages of commonly used induction agents for ECT. For seizure quality, methohexital is the reference, unless otherwise stated. CVS, cardiovascular; PONV, postoperative nausea and vomiting; ICP, intracranial pressure

<table>
<thead>
<tr>
<th>Induction agent</th>
<th>Seizure quality</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Propofol 0.75–2.5 mg kg⁻¹</td>
<td>Shortest duration (weighted mean difference however only 9 s)⁵</td>
<td>Improved CVS stability; less PONV; quicker emergence⁶</td>
<td>Pain on injection</td>
</tr>
<tr>
<td>Etomidate⁷ 0.15–0.3 mg kg⁻¹</td>
<td>Duration prolonged; may reduce seizure threshold¹ⁱ</td>
<td>Resistant seizures</td>
<td>Hyperdynamic response more pronounced compared with propofol; increased PONV longer emergence time</td>
</tr>
<tr>
<td>Methohexital 0.5–1.5 mg kg⁻¹ (supplemented with narcotic vs single agent)</td>
<td>‘Gold standard’</td>
<td>Long history of use</td>
<td>Reduced availability; increasing lack of familiarity with use</td>
</tr>
<tr>
<td>Thiopental 2–5 mg kg⁻¹</td>
<td>Duration reduced; better than propofol¹²</td>
<td></td>
<td>Need to reconstitute; increased dysrhythmias</td>
</tr>
<tr>
<td>Ketamine 0.7–2.8 mg kg⁻¹</td>
<td>Unclear—reduced³ and prolonged in different studies</td>
<td>Resistant seizures</td>
<td>Emergence phenomena; reduced CVS stability, and increased ICP</td>
</tr>
<tr>
<td>Sevoflurane 6–8% inspired concentration for induction. Thereafter 1–2 MAC</td>
<td>Comparable with thiopental;¹⁴ reduced seizure duration compared with methohexital¹⁵</td>
<td>Difficult venous access; reduces uterine contraction in pregnancy</td>
<td>Extra equipment required; more time-consuming</td>
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</tbody>
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should be considered in patients at high risk of myocardial ischae-

mia. Intravenous lidocaine is not effective.

**Airway management**

The patient should be pre-oxygenated. Intubation of the trachea is not routinely required, unless there are specific risk factors for reflux. After induction, ventilation can be gently assisted with a face mask. Hyperventilation lowers the seizure threshold and can prolong seizure duration. Inserting a bite block protects the patient’s teeth, lips, and tongue. After the electrical stimulus and during the seizure, the patient can be gently hand-ventilated until breathing resumes. During initial treatments, the stimulus magni-
tude may be titrated until an adequate seizure is generated. In such circumstances, further boluses of induction agent are required to maintain anaesthesia.

**Postoperative considerations**

Standard monitoring should be applied during recovery and oxygen supplementation continued until oxygen saturations are adequate on air. Most patients recover quickly and can be discharged according to the standard criteria. The presence of a trained escort familiar to the patient can be reassuring. The commonest side-effects are confusion, agitation, violent behaviour, amnesia, headache, myalgia, and nausea and vomiting. Cardiovascular complications may also still occur at this stage. Emergence agitation can be the most challenging problem to treat. Small doses of midazolam may be useful if simple measures such as a secluded, calm recovery environment do not help.

**Physical and psychological needs of the patient**

Regular review during treatment is required to assess both response to ECT and to detect any of the adverse physical events described above in order to avoid, minimize, or treat the problem. Adverse psychological effects are rare, but some patients develop an intense fear of treatment. Support and information are important in reducing this side-effect of therapy.

**Special patient populations**

**Cerebral aneurysm**

An increase in aneurysm wall stress can lead to enlargement or rupture. The increase in cerebral blood flow velocity during ECT is generally less with propofol than thiopental. Nitroprusside and β-blockers can partially inhibit this increase.

**Intracranial mass lesion**

Intracranial pressure may be reduced by pretreatment with steroids and diuretics and by hyperventilation before applying the electrical stimulus.

**Pacemakers or implantable defibrillators**

The risks are low because only small amounts of electricity reach the device due to high tissue resistance. Skeletal muscle potentials during the seizure may trigger pacemaker activity. A temporary conversion to fixed-rate pacing before ECT is recommended. Internal cardioverter-defibrillators should be deactivated before-hand and reactivated in the early recovery period.

**Neuroleptic malignant syndrome**

Neuroleptic malignant syndrome is a serious side-effect produced by some antipsychotic drugs with some clinical similarities to malignant hyperthermia (MH). Presenting features include muscle rigidity, fever, elevated creatine kinase, delirium, and autonomic instability. Agents such as succinylcholine known to trigger MH should be avoided.

**Pregnancy**

A large number of reports suggest that ECT is safe and effective throughout pregnancy and may be preferable to some forms of drug therapy. Potential complications for both the mother (e.g. aspiration) and the fetus (e.g. premature labour and spontaneous abortion) exist. Prophylactic tocolytic therapy is useful if there is a history of premature labour. In the later stages of pregnancy, using sevoflurane for maintenance may reduce the risk of uterine constrictions.

**Consent and the mental health act**

Illnesses requiring ECT can feature significant impairment of mental capacity, which may preclude obtaining informed consent. When prescribing ECT, capacity must therefore be first assessed. An adult has capacity if he/she can understand and retain the information relevant to the decision in question, believe that information, and weigh that information in the balance to arrive at a choice.\(^8\) When a patient can understand the nature, purpose, and likely risks and benefits, ECT cannot be given without consent. Even when a patient is capable, NICE recommends that doctors encourage the involvement of either an independent person who speaks on behalf of the patient (an ‘advocate’) or the person’s carer. When a patient is unable to consent, approval from the patient’s family should ideally be sought but this is not legally required. In these circumstances, care must be taken to ensure that it is legal to deliver ECT. Throughout the UK, a variety of forms and legislations exist, and although the names vary between the different countries, the principles in practice are generally similar, as described below.

When a patient is not detained in hospital and not resisting treatment but unable to give informed consent, then Incapacity legislation may be used. Other patients may be detained in hospital under Mental Health legislation for the assessment and supervision of a mental illness. Within this cohort, some patients still have
capacity to consent. If, however, a detained patient is incapable of consenting, then a formal, independent second opinion from the appropriate governing body, for example, the Care Quality Commission in England or the Mental Welfare Commission in Scotland must be arranged. When a patient is resisting or objecting to treatment or unable to express consent, the second opinion is essentially to confirm that ECT should only be administered to save the patient’s life or to prevent serious deterioration or suffering.

Emergency treatment (where the rate of deterioration dictates that time is of the essence) may be administered without consent or a formal second opinion, although it is good practice to obtain an informal second opinion before proceeding.

Finally, where a patient has an Advance Directive or Statement indicating that they would not wish to receive ECT, then this would normally be overridden only in an emergency.

Summary

Appropriately administered ECT is safe and effective. Anaesthetists must be aware of not only the physiological responses to ECT and how to modify these, but also understand the anaesthetic factors that may influence the efficacy of ECT.

Conflict of interest

None declared.

References


Please see multiple choice questions 20–23.