Review Article

Electroconvulsive therapy: physiological and anaesthetic considerations

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Electroconvulsive (ECT) or electroshock therapy remains controversial, and its indications are still the subject of discussion.^{1,2} Despite both medical and legal opposition, it is still widely practiced. In Britain, 200,000 treatments are given per year,³ and American studies show that about four per cent of psychiatric admissions are given ECT.^{4,5}

The aim of ECT is to produce a grand mal seizure – it is the seizure rather than the electrical stimulus which is responsible for the therapeutic effect.² This causes widespread physiological changes, particularly affecting the cardiovascular and nervous systems.

The cardiovascular changes may be altered by anaesthetic and other drugs, and the violent muscular contractions occurring during the convulsion can be reduced by muscle relaxants.

Before discussing the use of drugs, the physiological changes must be described.

Physiological changes

Activation of the autonomic system is responsible for profound cardiovascular changes during ECT.⁶ At the same time, venous return to the heart is

reduced by the raised intrathoracic pressure caused by the convulsion and by intermittent positive pressure ventilation (IPPV). Other physiological changes may relate to muscular work and anoxia.

The autonomic disturbance consists of a parasympathetic-sympathetic sequence during the early part of the procedure, followed by a similar but less marked sequence, starting in the clonic phase of the seizure and persisting into recovery. Parasympathetic discharge occurs immediately the current is applied, and the first sympathetic surge follows within seconds. The second sequence is less rapid and less severe.

These changes were investigated in dogs by Anton.⁷ During a five-second shock the dog was asystolic, with a dramatic fall in blood pressure (Figure). Immediately following the shock, cardiac rhythm returned, and the animal exhibited bradycardia and hypertension. The initial asystole could be abolished by atropine or the ganglion-blocker ecdolid. The post-shock rise in blood pressure could be reduced by alpha-adrenergic blockade, using phenoxybenzamine or high spinal block.

The same study showed a rise in circulating catecholamines. This also occurs in man,⁸ and is reduced by anaesthesia.⁹

Sympathetic-induced dysrhythmias and hypertension after treatment may occasionally be severe. In some patients the blood pressure reaches a peak within 60 seconds and then returns to normal, although in some it continues to rise for three minutes.¹⁰ Patients with untreated essential hypertension do not show a greater hypertensive response.¹⁰

Severe hypertension following ECT should be controlled with beta-adrenergic blockers, and pro-

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FIGURE Effects of ECT without atropinisation on the ECG and blood pressure in dogs. The arrow indicates passage of current for five seconds. Redrawn from the data of Anton.⁴

pranolol is effective.¹² It may be given prior to treatment.¹³ Although propranolol may exaggerate the muscarinic effects of succinylcholine,¹⁴ it gives better control than nitroprusside or diazoxide, which cause persistent hypotension.^{15,16}

Monitoring the heart rate and blood pressure regularly during treatment and recovery is therefore mandatory. Readily available facilities and expertise for performing cardiopulmonary resuscitation are essential.

Modification by anaesthetic drugs

Anticholinergics

Atropine has been routinely administered before ECT to attenuate excess vagal stimulation of the heart. It may be given subcutaneously an hour before treatment or intravenously immediately before induction. The intravenous route is more effective in preventing bradycardia, and spares the patient the discomfort of a dry mouth¹⁷ – it is widely used.^{3,4}

This current routine is now being questioned. French¹⁸ argues that most patients have a tachycardia preceding treatment, possibly due to tricyclic antidepressant therapy, and that if bradycardia or asystole occurs this reflects inadequate ventilation.

Succinylcholine may cause a bradycardia, but pretreatment with atropine does not offer significant protection.¹⁹

The practical benefits of intravenous atropine appeared small in two published series. Wyant^{20,21} performed a within-patient crossover trial, on 37

patients. He gave 1.0 mg or 0.5 mg, or none, ten seconds or two minutes before induction. Tachycardia occurred in all groups before treatment, and intravenous atropine conferred no particular benefit or protection. In particular, the antisialogogue effects were not clinically beneficial.

Verheke²² confirms this contention. In a series of 6000 ECT's, over a period of 24 years, he claims severe bradycardia (50 beats per minute) was not detected clinically, and sialorrhoea was not troublesome.

A well-publicised death after ECT was blamed on inadequate atropinisation.²³ However, cardiorespiratory arrest occurred ten minutes after the shock. No ECG recordings were available at the time, and the initial rapid response to resuscitation suggests hypoventilation may have been responsible.

On the basis of the available evidence, the routine administration of atropine does not seem necessary. It should, however, be readily available whenever ECT is performed.

Induction agents

Anaesthetic requirements include smooth induction, autonomic stability, safety with repeated administration and rapid recovery. The recovery period is significantly affected by the patient's concomitant psychotropic medication.

For the past twenty years, methohexitone has been the most popular anaesthetic agent.²⁴ After a series of within-patient crossover trials, Allen and co-workers²⁵ showed it to be the drug of choice, and it remains the most widely used.³

Thiopentone, another barbiturate, has also been used. However, it offers no significant advantages over methohexitone and may prolong the recovery time.^{26,28}

Propranolol, a eugenol derivative, provides rapid recovery,^{29,30} but prolongs the action of succinylcholine.³¹ It has a high incidence of anaphylactoid reactions.³² However, it is widely used in Russia.³³

Alfathesin (alfadolone/alfaxalone) also provides fast recovery, though slower than methohexitone,³⁴ with no prolongation of the apnoea time. However, the incidence of anaphylactic reactions, particularly on repeated administration,³⁵ led to its withdrawal by the manufacturers in 1984.

Ketamine was claimed to be a safer drug³⁶ with little evidence. It has sympathomimetic properties

which are undesirable, recovery is slower than after methohexitone, and there is a high incidence of nausea and ataxia.³⁷ Emergence hallucinations are rare, due either to some aspect of the treatment or to concomitant medication. Intramuscular ketamine may rarely be justified if intravenous access is impossible.

Etomidate was studied in an uncontrolled trial³⁸ and the authors expressed some enthusiasm, although noting the high incidence of pain on injection. A more objective trial³⁹ found recovery was no faster than after Alfathesin, and induction was more stormy.

The incidence of post-treatment anxiety, agitation, headaches and giddiness may be reduced by pretreatment with intravenous diazepam or haloperidol. Although neither drug prolongs the recovery period,⁴⁰ there is evidence that administration of benzodiazepines shortens seizure duration,²⁷ and should not be routinely given.

Methohexitone remains the drug of choice for most patients undergoing ECT. Other drugs offer little advantage in the majority of patients.

Muscle relaxants

The violence of the convulsions led to the use of muscle relaxants during ECT, preceeding their use in anaesthesia. Curare was the first used by Bennett,⁴¹ but succinylcholine is now the most popular.

When neuromuscular blockers are used, it may be difficult for the therapist to ascertain whether a convulsion has actually occurred. Dilatation of the pupils, "goose-flesh," flickering of the eyelids or jerking of the toes have been used as signs of cerebral seizure. These are not dependable,²⁶ as they can result from direct stimulation of peripheral pathways.

The surest way to record a seizure is electroencephalography, and some modern ECT instruments include a recorder for the direct measurement of EEG activity. An alternative method is to inflate a tourniquet around an arm, above arterial pressure, before giving the relaxant. This "isolated forearm" will then exhibit the tonic and clonic phases. When unilateral ECT is given, the cuff should be placed on the homolateral arm. Arm movements persisting for 25 seconds or more is regarded as adequate evidence that a convulsion has occurred – this correlates well with EEG recordings⁴² and is an acceptable alternative.

Muscle relaxants have reduced but not abolished traumatic complications. During application of the current, the jaws clench, and tooth damage resulting from force on a Guedel airway has occurred.⁴³ To prevent this, a rubber or plastic "bite block" should be inserted prior to stimulation. Other reported injuries include spontaneous rupture of the spleen.⁴⁴

There is a period of apnoea lasting about two minutes after the shock, during which artificial ventilation is performed with oxygen. Joshi's claim that this is unnecessary⁴⁵ seems extremely dangerous – 90 seconds of apnoea without preoxygenation results in a substantial fall in arterial PO_2 to a level which threatens the subject with loss of consciousness.⁴⁶

Succinylcholine dosage should be carefully judged. Pitts⁴⁷ found that $0.3 \text{ mg}\cdot\text{kg}^{-1}$ gave little modification (softening of convulsions), $0.4 \text{ mg}\cdot\text{kg}^{-1}$ reduced the number of vigorous seizures and $0.5 \text{ mg}\cdot\text{kg}^{-1}$ prevented them. $0.6 \text{ mg}\cdot\text{kg}^{-1}$ caused a prolonged period of apnoea, and he concluded $0.5 \text{ mg}\cdot\text{kg}^{-1}$ was the optimal dose. The dose given and the clinical effects should be recorded after each administration, and subsequent doses adjusted accordingly.

The duration of apnoea after a given dose of succinylcholine is reduced by ECT.⁴⁸ It is prolonged by lithium, although this interaction is probably not clinically significant.^{49,50}

It is fortunate that muscle pains are remarkably uncommon after succinylcholine administration for ECT.^{26,29,51} This is in spite of the fact that these patients are ambulant and show normal fasciculations.

Contraindications to succinylcholine are rare, though in some patients, it must be considered an unacceptable drug. Allergy is not uncommon,³² and these patients should be given an alternative relaxant. Succinylcholine is also undesirable in patients with cholinesterase deficiency – the most extreme case being homozygotes for atypical genes who may only require 1–3 mg.⁵² This dose would be too low for heterozygotes or patients with acquired cholinesterase deficiency.

Succinylcholine may also produce an unwanted rise in serum potassium.^{48,53} As an alternative, gallamine (80 mg) has been advocated by Brunton, who reported the duration of apnoea post-shock (using neostigmine reversal) was only one min-

ute.⁵⁴ The role of the newer short-acting non-depolarising relaxants remains to be seen.

Whenever neuromuscular blockers are used, there is a risk that the patient may be aware whilst paralysed. This may occur if succinylcholine is given together with propranolol^{26,31} or monoamine oxidase inhibitors (see below) or to patients with abnormal pseudocholinesterase. Although the amnesia induced by ECT may prevent the patient from recalling this experience, this is not always reliable,⁵⁵ and such patients are unlikely to consent to a further treatment.

Assessment of patients

All patients subjected to ECT should undergo a thorough physical examination, and a detailed medical history should be taken.⁵⁶ Particular attention should be paid to cardiorespiratory function, symptoms of oesophageal reflux, allergies, and previous anaesthetic experience, and a full neurological assessment should be made. The presence of loose or missing teeth should be recorded. Suggested minimum investigations are given in Table I. The patient should give written consent to the procedure.

TABLE I Suggested investigations to be performed on patients undergoing ECT

Full blood count and serum B ₁₂
Electrolytes, BUN and glucose
Thyroid function tests
Sickledex (where appropriate)
Urinalysis and urinary porphyrins
Chest x-ray
Electrocardiograph

This list should be regarded as a minimum, and other tests should be performed where clinically indicated.

Records should be kept of each administration. This should include details of the drugs given and their effects, the nature of the convulsion, the heart rate and blood pressure during the treatment and subsequent 60 minutes and any side effects or complications encountered.

Contraindications and precautions (see Table II)

Myocardial infarction within the preceding three months constitutes an absolute contraindication, although at least one patient has inadvertently undergone ECT whilst during the acute phase.⁵⁷

TABLE II Contraindications to ECT (after Perrin³) (See text for discussion)

Cardiovascular	Myocardial infarction within preceding three months. Severe angina or congestive heart failure. Aneurysm of major vessel. Thrombophlebitis.
Orthopaedic	Severe osteoporosis Major fractures.
Pulmonary	Acute or severe chronic disease.
Neurological	Cerebrovascular accident in past 3 months. Cerebral tumour.
Other	Remediable conditions, e.g., thyrotoxicosis, pernicious anemia, pregnancy.

The presence of severe ischaemic heart disease is also a contraindication, in view of the possible cardiovascular stress imposed by ECT (see above).

Patients with cardiac pacemakers present little problem since, providing the pacemaker is correctly sited, virtually no current passes the heart. During the convulsion, skeletal muscle potentials may fall within the range of the pacemaker's sensing circuits, and inhibit demand pacemakers. The magnet-operated switch, fitted in most devices, should then be activated. The patient should be isolated from ground, and the electrocardiogram should be monitored during and after treatment.^{58,59}

Cerebral tumours, particularly when associated with raised intracranial pressure, are usually considered a contraindication to ECT. Maltbie⁶⁰ reports a 28 per cent one-month mortality in these patients, although Dressler⁶¹ considers it justifiable in depression associated with terminal disseminated malignancy. A rise in cerebral blood flow, lasting for some hours, occurs after ECT,⁶² which may be responsible for a further rise in intracranial pressure.

Thrombophlebitis was originally considered a contraindication because of the risk of dislodging an embolus—nowadays these patients would be treated with anticoagulants. Although there are risks from intracranial bleeding after ECT, there have been no reported cases in anticoagulated patients, and Alexopolous *et al.*⁶³ report two uneventful cases. They suggest that warfarin is changed to heparin before a course of treatment, to allow better control, and reduce the possibility of drug interactions.

ECT causes significant elevation of intraocular

pressure after the convulsion,⁶⁴ which may be undesirable in patients with glaucoma.

Malignant hyperpyrexia, commonly precipitated by succinylcholine, has not been reported after ECT in the past 16 years.⁶⁵ It is unlikely that this would have been undetected – perhaps the short exposure terminates attacks. A patient with a history of malignant hyperpyrexia has been successfully managed by Franks,⁶⁶ using pretreatment with dantrolene, premedication with diazepam, meperidine and droperidol, and induction with thiamylal without a relaxant.

Operators of ECT equipment have received electric shocks. The electrolyte solution, used to ensure good electrical contact between electrode and patient, may drip along the legs of the headset, producing a conducting path. The operator usually holds the headset in one hand and supports the chin with the other, and is at risk of electrocution. It is wise practice to wear rubber gloves, and to minimise the amount of electrolyte used.⁶⁷

Antidepressant therapy

Antidepressant therapy may cause cardiovascular disturbances. However, depressive illness itself is associated with an autonomic disturbance, characterised by a decrease in central sympathetic outflow, coupled with increases in alpha-adrenergic and decreases in beta activity. This profile varies with the severity of the depression.⁶⁸ The anticholinergic side effects complained of during antidepressant therapy may be a feature of the disease rather than the drug.^{69,70}

Antidepressant drugs commonly used include tricyclics and monoamine oxidase inhibitors. Tricyclics, as a group, are all similar although some are more sedative than others. Cardiotoxicity may occur in therapeutic doses, causing tachycardia and conduction abnormalities, and they enhance the action of epinephrine and norepinephrine. Mianserin, a tetracyclic, is relatively free of these effects.⁷¹

Monoamine oxidase inhibitors (MAOI's) have great potential toxicity, and there is a tendency to employ them only in cases which fail to respond to other drugs or ECT. They form a stable complex with the enzyme, and regeneration of new enzyme takes 10–14 days.

Postural hypotension and an exaggerated hypotensive response to anaesthetic agents are common. In addition, hypertensive crises can occur if patients

who have taken these drugs are exposed to certain foods or drugs. A number of therapeutic agents not metabolised by monoamine oxidase are potentiated, especially sympathomimetic amines.^{72,73} These pressor effects are particularly common with pargyline and iproniazid.

Cardiac arrest has occurred after both modified and unmodified ECT in patients taking these drugs, probably due to uncontrolled sympathetic activity.⁷⁴ Such a crisis may be managed with alpha-adrenergic blockers.

In addition, phenelzine may (reversibly) reduce serum pseudocholinesterase activity, and patients taking it have remained paralysed for up to an hour after a dose of succinylcholine.⁷⁵

ECT in patients taking MAOI's presents considerable risks, and most psychiatrists agree should not be performed.³

Mortality

It is noteworthy that the mortality amongst depressed patients is increased compared to the age-corrected population, even excluding deaths from suicide. Those having ECT have the lowest mortality.⁷⁶

In the 1940's, the mortality of ECT was 0.06 per cent, and in the five years up to 1953, 67 deaths occurred in Britain. This compared favourably with leucotomy but was higher than after insulin therapy. The majority of these deaths were related to the cardiovascular system, although only two were due to primary cardiac arrest. Nineteen related to the respiratory system, including three cases of pulmonary embolus. At least two were caused by anaesthesia.⁷⁷

Recently, a series was reported in which one death occurred in 42 patients.⁵ In another American series, three deaths were recorded in 1,765 patients,⁴ while a series from Britain quotes four deaths in 2,594 patients³ and an Australian series²⁶ reports no deaths in 3,500 administrations. The overall mortality in these series is 0.16 per cent, but it is unclear why there is such large variation between hospitals.

The risks of ECT have to be balanced against the toxicity of antidepressants – many consider that ECT is the safest and most effective treatment for severe depression. This particularly applies in elderly patients,⁷⁸ in whom ECT is particularly widely used.³

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