

# Anesthesia for ECT

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## Abstract

*Successful electroconvulsive therapy (ECT) requires close collaboration between the psychiatrist and the anaesthetist. During the past decades, anaesthetic techniques have evolved to improve the comfort and safety of administration of modern ECT. We review the literature and discuss the selection, preparation, and management. Specifically, the general principles and the pre-ECT evaluation are discussed; a review of induction agents, muscle relaxants, anticholinergics, and antihypertensives is included. Particular focus is given to interference with a seizure by these medications (German J Psychiatry 2001;4:33-39).*

*Key words: Electroconvulsive therapy (ECT), pre-ECT evaluation, anaesthesia, ECT, anticholinergics, anti-hypertensives, seizure inhibition*

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## Introduction

**E**lectroconvulsive therapy (ECT) has changed substantially during the past decades. It has become more complex, more precise, and is always regarded as a highly sophisticated medical procedure (American Psychiatric Association [APA], 1990). The ECT practitioner must have skills not only in patient selection and the optimal use of medications accompanying ECT, but must also be knowledgeable about cardiovascular physiology, anesthesia, and interpretation of the ictal EEG. The practitioner is required to make decisions about electrode placement, energy dosing, joint use of psychotropic and systemic medications, and continuation treatment with either medication or ECT (APA, 1990; Beyer et al., 1998; Fink & Kellner, 1998). Furthermore, in order to achieve informed consent the practitioner must be able to explain all aspects of the treatment and to answer questions from patients and their relatives in an accurate and understandable way (Fink & Kellner, 1998).

These changes in technology have stimulated the repeated calls for better training (Fink, 1986; NIH, 1985). Unfortunately, surveys indicate that adequate training in

this area is lacking (Chanpattana, 1999; Duffett & Lelliott, 1997, 1998; Halliday & Johnson, 1995; Pippard, 1992; Pippard & Ellam, 1981). In addition, a survey of ECT-related psychiatric knowledge among 261 British anesthetists revealed that two thirds of respondents favored anesthetic training including more information about psychiatric aspects of ECT (Haddad & Benbow, 1993). Since successful ECT treatment requires close collaboration between the psychiatrist and the anesthetist, and the casual approach to either the psychiatric or the anesthetic management of a patient scheduled for ECT is unacceptable; therefore, proper ECT training syllabus and adequate supervision on modern ECT for both the psychiatric and anesthetic residents are sorely needed (APA, 2001; Duffett & Lelliott, 1997, 1998; Fink, 1998; Folk et al, 2000; Haddad & Benbow, 1993; Halliday & Johnson, 1995; Pippard, 1992; Pippard & Ellam, 1981).

The anesthetist must have an in-depth knowledge of the physiologic effects of ECT and the pharmacology of drugs given (Folk et al, 2000). At the present time, a number of medications have been used during ECT including pretreatment sedation, anesthetic agents, muscle relaxants, anticholinergics, and drugs to attenuate parasympathetic and sympathetic responses (APA, 2001). The authors review the literature and discuss the selection, preparation, and management.

Specifically, the general principle and the pre-ECT evaluation are discussed; a review of medications used during ECT is included. Particular focus is given to interference with a seizure by these medications.

## General principle

Inhibition of the ECT seizure reduces its efficacy, as seen when lidocaine diminished the efficacy of ECT as it decreased seizure length, seizure intensity, and EEG postictal suppression (Cronholm & Ottosson, 1960). Absence of an effect on seizure length does not imply the absence of inhibition of seizure, because seizure intensity and generalization through the brain greatly affect therapeutic impact (Chanpattana et al, 2000; Nobler et al, 1993, 2000). Although the seizure promoting and inhibiting effects of CNS stimulants and depressants are generally recognized, the clinician can be surprised, as for example by the potent anticonvulsant effects of the cough suppressant dextromethorphan; therefore, minimization of medications for ECT patients is generally desirable (APA, 1990, 2001).

Because psychotropic medications (e.g. antidepressants, neuroleptics, benzodiazepines, lithium, ect.) might have interactions with the drugs used in anesthesia or might influence seizure threshold. The APA Task Force Report on ECT (1990), all psychotropic medications should be stopped before the beginning of ECT without recommending about a definite time for the wash-out period. In research, we usually require about 7 days. In the 2001 Edition, they add that neuroleptics should be used concomitantly with ECT in treating schizophrenic patients.

## Pre-ECT evaluation

The pre-ECT evaluation serves several functions. The anesthetist's goal is to ensure the patient's safety by identifying risks of anesthesia and recommending appropriate modifications, tests, and consultations (Beyer et al, 1998; Haddad & Benbow, 1993; Kellner et al, 1997). Patients scheduled for ECT may be delusional, paranoid, or uncommunicative. A thorough explanation may alleviate fears and anxiety. The evaluation should include the medical history and physical examination. A review of systems, particularly the neurologic and cardiovascular systems, may identify potential problems. A complete examination, including airway examination, must be performed. The cardiovascular, pulmonary, and neurologic examinations are particularly important for the possible physiologic changes during ECT. Previous anesthetic history, allergies, and current medications used are

integral parts of the evaluation (Beyer et al, 1998; Kellner et al, 1997).

Minimum pre-treatment laboratory data guidelines have not been suggested for preparing patients for ECT. Nevertheless, young, medically healthy patients may not require any laboratory tests (APA, 2001). In general, complete blood count, serum electrolytes and electrocardiogram may be sufficient (APA, 1990). If there is any coexisting medical illness and hence an increase in peri-ECT risk, then further testing or consultation is appropriate (Beyer et al, 1998; Kellner et al, 1997).

Potential modifications of ECT technique include the use of medications before, during, and after the ECT treatment, changes in technical aspects of the electrical stimulation, and the use of additional types of physiologic monitoring (APA, 1990, 2001; Beyer et al, 1998). In certain high-risk situations, the presence of specialty medical consultants at the time of treatment may be indicated (APA, 2001, Kellner et al, 1997).

## Pretreatment sedation

The goal of pre-ECT sedation is to facilitate cooperation with the procedures (APA, 1990). Fearful patients are helped in their decision for ECT by offering sedation prior to the first ECT, to decrease awareness of the procedures and to calm them. The sedative of choice is given intramuscularly, because of the prohibition on oral intake; it should avoid interference with the ECT seizure or with the determination of response to ECT, as by obscuring clinical signs (APA, 1990, 2001). Drugs with anticonvulsant properties, specifically benzodiazepines and barbiturates (Greenberg & Pettinati, 1993), are counterproductive. Potent neuroleptics may obscure clinical signs in patients with psychotic depression or mania, and thus impairing the ability to judge when a course of ECT is sufficient. Therefore, hydroxyzine or promethazine 25-50 mg IM, or droperidol 2.5-5 mg IM, are helpful because these sedatives promote seizure activity or do not affect it (APA, 1990).

## Anesthetic agents

*Objective:* To leave the patient unaware of potentially frightening sensations, particularly muscle paralysis and feelings of suffocation and the image of a light flash that may accompany the beginning of the stimulus, without obstructing the seizure (McCleave & Blackmore, 1975).

*Principle:* To promote ultra-brief, light general anesthesia (APA, 1990, 2001). Excessive anesthetic dosage may prolong unconsciousness and apnea, have more anticonvulsant effect, increase the risk of cardiovascular complications, and intensify amnesia (Boylan et al, 2000; Miller et al, 1985).

An ideal induction agent for ECT would ensure rapid unconsciousness, be painless on injection, have no hemodynamic effects, have no anticonvulsant properties, provide rapid recovery, and be inexpensive (APA 1990, 2001; Folk et al, 2000). No drug has all these characteristics. Nevertheless, methohexital has many of them; thiopental, ketamine, propofol, and etomidate have also been used successfully in ECT.

## Commonly used induction agents

1. *Methohexital* has a rapid action, short duration of action, low cardiac toxicity (Mokriski et al, 1992), minimal anticonvulsant effects (dose-related), and is associated with pain on injection. Other possible side effects include hypotension, shivering, hiccoughing, and necrosis of soft tissue at the injection site. The APA Task Force on ECT recommends its use as an induction agent of choice (APA, 1990). The typical dose is 0.5-1 mg/kg.

2. *Thiopental* has greater anticonvulsant effects and longer duration of action than methohexital (APA, 1990). Patients with cardiovascular disease whom induced with thiopental may have a greater incidence of postictal electrocardiographic abnormalities, compared with methohexital (Pitts, 1982). As with methohexital, thiopental can induce hypotension and cause possible necrosis at the injection site. Typical dose is 2-4 mg/kg (APA, 1990).

3. *Ketamine* is a derivative of phencyclidine, which inhibits the N-methyl-D-aspartate (NMDA) subtype of glutamate receptor (Huettner & Bean, 1988). Compared with methohexital, ketamine had slower onset, delayed recovery, and increased incidence of nausea, hypersalivation, 'bad trips', and ataxia during recovery (McInnes & James, 1972). It is recommended for patients with increased seizure threshold so that seizure elicitation is difficult, and typical dose is 0.5-2 mg/kg (APA, 1990, 2001).

4. *Propofol* has rapid onset, short duration of action, and is commonly associated with pain on injection. It has potent anticonvulsant properties (APA, 1990), as evidenced by a number of studies. Propofol (dose 0.75-1.5 mg/kg) resulted in: 1) markedly decreased the intensity and the duration of seizure (Avramov et al, 1995; Boy & Lai, 1990; Chanpattana, 2000; Kirkby et al, 1995; Ramp-ton et al, 1989; Rouse, 1988), 2) a need for more numbers of treatment (Mitchell et al, 1991), 3) attenuation of ECT-induced hypertension and tachycardia (Villalonga et al, 1993), 4) a decrease in seizure-induced prolactin and ACTH release (Mitchell et al, 1990), and 5) successful use in the treatment of status epilepticus (Chilvers & Laurie, 1990; Wood et al, 1988). Nevertheless, randomized trials between propofol and either methohexital or thiopental do not demonstrate a difference in the therapeutic outcome or the speed of postictal recovery (Martensson et al, 1994; Matters et al, 1995). Other side effects include hypotension, apnoea, bradycardia, etc.

5. *Etomidate* is associated with pain on injection, as with methohexital, and causes prominent myoclonic activity during induction. Etomidate offers the advantage of having minimal effects on myocardial contractility and cardiac output (Morgan & Mikhail, 1996). It is recommended in patients with decreased cardiac output as well as patients with increased seizure threshold (APA, 1990). Typical dose is 0.15-0.3 mg/kg.

## Muscle relaxants

*Objective:* To prevent injuries to the musculoskeletal system and to improve airway management (APA, 1990).

*Principle:* To provide the moderate degree of muscular relaxation (APA, 1990, 2001). In general, complete paralysis is neither necessary nor desirable since it may be associated with prolonged apnea. In addition, the intensity and the duration of ictal motor movements should be observed and monitored (Beyer et al, 1998). Muscle paralysis not only facilitates oxygenation, but also decreases oxygen utilization by muscles during the seizure (APA, 1990). Consideration should be given to higher dosage of muscle relaxants for patients with Harrington rods, or at risk for developing pathologic bone fracture (APA, 1990; Coffey et al, 1986; Milstein et al, 1992).

The adequacy of muscular relaxation should be ascertained before applying the ECT stimulus. This process is done by testing for a reduction in deep tendon reflexes and muscle tone (APA, 1990). In patients receiving high dose of succinylcholine, a peripheral nerve stimulator should be used (APA, 1990; Beyer et al, 1998; Kellner et al, 1997).

## Commonly used agents

Common side effects of succinylcholine include arrhythmia, increased intraocular or intraabdominal pressure. Since succinylcholine has been associated with malignant hyperthermia and hyperkalemia, nondepolarizing muscle relaxants have been developed. The dosage of these agents should be determined individually and clinically. Typically, the succinylcholine dose is 0.5-1 mg/kg. Atracurium, 0.3-0.5 mg/kg (Hickey et al, 1987); mivacurium, 0.15-0.2 mg/kg (Kelly & Brull, 1994); rocuronium, 0.45-0.6 mg/kg (Motamed et al, 1997); and rapacurium, 1-2 mg/kg (Szenohradzky et al, 1999) are alternatives to succinylcholine (Savarese et al, 2000). These nondepolarizing muscle relaxants produce more prolonged paralysis, and both the onset and duration of action should be monitored with a nerve stimulator (APA, 2001).

## Anticholinergics

*Objective:* To protect against the parasympathetic-induced bradycardia or asystole (Altschule, 1950; Bankhead et al, 1950).

*Principle:* To decrease the effects of ECT-induced vagal stimulation (APA, 1990). Vagal reflex occurs immediately following the ECT stimulus regardless of the amount of electrical charge, and may be associated with transient bradycardia or asystole. If the electrical charge is near or above the seizure threshold, tonic-clonic motor seizures may occur with accompanying sympathetic stimulation. This sympathetic surge counteracts the effects from vagal stimulation. But if the electrical stimulus fails to elicit the seizure (subconvulsive stimulation), the bradycardia immediately following the stimulus is of graver concern, since the protection afforded by the ictal tachycardia is absent (Bellet et al, 1941).

### Indications

1. Patients undergoing an estimation of seizure threshold by dose-titration method, especially at the first ECT session (APA, 1990; Bouckoms et al, 1989).
2. Patients receiving sympathetic blocking agents (APA, 1990).
3. Situations when the occurrence of vagal bradycardia should be prevented: e.g., pre-existing cardiac disease, hypodynamic cardiac functions (APA, 2001; Bouckoms et al, 1989).

### Commonly used agents

The most commonly used anticholinergics are atropine (0.4-0.8 mg iv or 0.3-0.6 mg im) and glycopyrrolate (0.2-0.4 mg iv or im). Atropine has a more potent effect on heart rate (APA, 1990). Glycopyrrolate does not cross the blood-brain barrier, and has more potent antisialagogue effects (Morgan & Mikhail, 1996).

## Agents modifying cardiovascular response

*Objective:* To attenuate the ECT-induced cardiovascular responses (Gravenstein et al, 1965; Perrin, 1961).

*Principle:* The risks of ECT are well recognized. The peritreatment mortality rate is about 0.002% (or 1: 80,000 [APA, 2001]). Cardiovascular complications, arrhythmias,

myocardial infarction, congestive heart failure, and cardiac arrest, are among the most common causes of death (APA, 1990). At the present time there is no consensus on the indications for use of these agents. The APA Task Force on ECT suggests that indiscriminant use should be avoided (APA, 2001).

During ECT-induced seizures, cerebral blood flow increases up to 300%, oxygen use and glucose metabolism increase up to 200% (Ackerman et al, 1986). Therefore, the peripheral hemodynamic surge appears to be necessary to sustain this demand, and provides adequate supply of oxygen and carbohydrates to the brain. Given these concerns, judgment is needed about when to use these agents (APA, 2001).

## Recommendations

1. Over-treatment is more dangerous than under-treatment. Thus, routine prophylactic antihypertensive treatment of all patients is not recommended (APA, 1990, 2001).
2. Initial efforts should be directed to obtain control of blood pressure and heart rate with daily administration of an oral agent, before beginning ECT (APA, 2001; Beyer et al, 1998).
3. In patients who are at risk for cardiovascular complications, such as those with unstable aneurysms, a complete block of ECT-induced hemodynamic changes is recommended (APA, 1990, 2001; Beyer et al, 1998; Kellner et al, 1997).
4. Sustained hypertension or significant arrhythmia after seizure is then treated acutely, and prophylaxis is considered for subsequent treatments (APA, 1990, 2001).

### Commonly used agents

Several antihypertensive agents have the potential to limit the hemodynamic effects of ECT. They differ in onset and duration of action, relative impact on blood pressure versus heart rate, effects on myocardial work, and on seizure duration (APA, 2001; Folk et al, 2000; Perrin, 1961).

1. *Beta blockers.* The literature on beta blockers is extensive. The major side effect is their anticonvulsant properties. Labetalol is the most commonly used  $\beta$ -blockers at present. It selectively blocks  $\alpha_1$  and nonselectively blocks  $\beta_1$  and  $\beta_2$  adrenergic receptors. The starting dose is 5-10 mg given intravenously. Its onset of action is 2-5 minutes with duration of action about 4-6 hours. Esmolol has a faster onset (30-90 seconds) and much shorter duration of action (~ 10 minutes), has more effect on blood pressure than heart rate, and decrease more cardiac work load, compared to labetalol (APA, 2001; Castelli et al, 1995).

2. *Nitroglycerine* dilates the venous system with little effect in myocardial contractility (Villalonga et al, 1989). There are

many preparations, such as spray, injection, and ointment. In practice, nitroglycerine administered as sublingual spray (0.4 mg/spray) several minutes before ECT attenuates hypertension.

3. *Trimethaphan*, a ganglionic blocking agent, is among the first intravenous antihypertensive recommended for ECT (APA, 1990). It inhibits both sympathetic and parasympathetic nervous systems with effect on arterioles, and promotes peripheral vasodilation without inducing reflex tachycardia. It is only available in parenteral form and has onset of action within minutes. Trimethaphan boluses attenuated blood pressure and heart rate during ECT without rebound hypertension, prolonged hypotension, arrhythmias, or effects on seizure duration (Petrides et al, 1996).

4. *Nicardipine* provides adequate control of mean arterial pressure, but heart rate usually increases before, during, and after ECT (Avramov et al, 1996). There was no effect on seizure duration.

5. *Nitroprusside* can also be used to control blood pressure but has a greater incidence of post-ECT hypotension (Ciraulo et al, 1978). It is a potent vasodilator affecting both arterioles and venous system, and may produce reflex tachycardia. Some anaesthetists consider intra-arterial monitoring while using this agent (Beyer et al, 1998; Kellner et al, 1997).

## Conclusions

ECT is a safe and effective treatment modality in psychiatric patients. Understanding the basic knowledge of ECT and the pharmacology of the agents used during the treatment are extremely important for providing optimum anesthetic care. Anesthetic management, when performed with proper theoretical background, can enhance the efficacy and safety of ECT. Successful ECT treatment requires close collaboration between the psychiatrist, anesthetist, and the nursing staff.

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