



Challenges during Electroconvulsive Therapy—A Review

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J Neuroanaesthesiol Crit Care 2021;8:173–179.

Abstract

Electroconvulsive therapy (ECT) is one of the most successful treatment techniques employed in psychiatric practice. ECT is usually administered as a last resort to a patient who fails to respond to medical management or on an urgent basis as a life-saving procedure when immediate response is desired. It is performed under general anesthesia and is often associated with autonomic changes. All attempts should be made to minimize the resulting hemodynamic disturbances in all the patients using various pharmacological methods. Anesthesiologists providing anesthesia for ECT frequently encounter patients with diverse risk factors. Concurrent cardiovascular, neurological, respiratory, and endocrine disorders may require modification of anesthetic technique. It is ideal to optimize patients before ECT. In this review, the authors discuss the optimization, management, and modification of anesthesia care for patients with various cardiac, neurological, respiratory, and endocrine disorders presenting for ECT to improve the safety of the procedure. It is not infrequent that an anesthesiologist also plays an important role in inducing a seizure. Proconvulsants such as caffeine, adjuvants like opioids, hyperventilation, and appropriate choice of anesthetic agent for induction such as etomidate or ketamine can help. The use of BIS monitoring to guide the timing of electric stimulation is also elaborated in this review.

Keywords

- ▶ anesthesia
- ▶ electroconvulsive therapy
- ▶ challenges
- ▶ concurrent diseases
- ▶ proconvulsant
- ▶ systemic diseases

Introduction

Electroconvulsive therapy (ECT) is one of the most effective treatment modalities in psychiatric practice. ECT was first used in 1938 by Ugo Cerletti.¹ In 1944-45, it became “modified ECT,” with initial anesthesia consisting of only a hypnotic agent. The first muscle relaxant was tubocurarine; later, suxamethonium (Sch) was added in 1951.²

Anesthesiologists providing anesthesia for ECT frequently encounter patients with diverse risk factors. Concurrent cardiovascular, neurological, respiratory, and endocrine disorders may require modification of anesthetic technique. It is ideal to optimize patients before ECT; however, this may not be feasible if ECT is administered on an urgent basis as a life-saving procedure. We, in this

narrative review, highlight the physiological and pharmacological initiatives that can be undertaken to optimize these patients and improve the safety of the procedure. An anesthesiologist’s role in facilitating seizures for patients who are seizure resistant is noteworthy; proconvulsant techniques are therefore elaborated.

The information is collated from the published literature on ECT and has been discussed under the following headings, focusing on each system involvement.

Implications for anesthesia and ECT will be discussed under the following headings:

1. Cardiovascular system considerations.
2. Central nervous system considerations.
3. Considerations in respiratory illnesses.

published online
August 2, 2021

DOI <https://doi.org/10.1055/s-0041-1731627>
ISSN 2348-0548

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4. Endocrine diseases.
5. Eliciting seizures in patient with missed seizure.

Cardiovascular System Considerations

ECT has a biphasic effect on the cardiovascular system (CVS), with initial marked parasympathetic discharge, manifesting as profound bradycardia, hypotension and, rarely, sinus arrest or asystole, followed by sympathetic hyperactivity with the onset of convulsion.

The initial vagal effect can be attenuated with intravenous (IV) atropine or glycopyrrolate. Since this phase is transient and is followed by sympathetic discharge, routine use of anticholinergics (A/ch) is not recommended.³ However, sub-convulsive stimuli may precipitate severe life-threatening bradyarrhythmia and hypotension due to absence of seizure-induced sympathetic activation. A/ch are therefore indicated in patients in whom a seizure threshold is yet to be established, when missed seizures are likely (e.g., first ECT), and in patients receiving sympathetic blockers.

The phase of sympathetic discharge lasts for several minutes. There is increase in cardiac output (CO), systemic vascular resistance (SVR), blood pressure (BP), and heart rate (HR), with tachyarrhythmia and increasing myocardial oxygen demand. Although this phase is also transient, it may lead to myocardial infarction (MI) or intracerebral hemorrhage (ICH) in vulnerable population. It can also lead to transient left ventricular (LV) systolic dysfunction, acute coronary syndrome (ACS), or cardiac failure, even in patients with no previous cardiac history.⁴⁻⁶ Takotsubo cardiomyopathy has been reported in postmenopausal women.⁷

In view of these adverse effects, pharmacological attenuation of sympathetic effect is desirable. Esmolol reduces tachycardia but at higher dosages can shorten the seizure duration.⁸ The dose of esmolol ranges from 500 mcg/kg to 1 mg/kg IV bolus, followed by infusion of 100 to 300 mcg/kg/min. Dexmedetomidine (Dex) 1 mcg/kg IV over 10 minutes just prior to induction (with propofol) fared better than esmolol or placebo in blunting the hemodynamic response to ECT, without prolonging recovery time or duration of motor seizure.^{9,10} Delayed recovery is reported with the use of Dex, which can be mitigated by reducing the dose of anesthetic.^{11,12} Dex can reduce post-ECT headache and agitation.¹³ Remifentanyl at a dose of 1 mcg/kg bolus has been found to be a beneficial adjunct to 8% sevoflurane, without affecting seizure duration or recovery profile.¹⁴ Lignocaine (1 mg/kg) conferred no benefit in blunting the hemodynamic effects of ECT.¹⁵

American Heart Association (AHA)/American College of Cardiology (ACC) does not specify ECT into a specific risk category. A few shelves ECT into a high-risk procedure, due to the acute and severe physiologic changes it produces in a brief period.¹⁶

American Psychiatry Association (2001) does not declare any absolute contraindication for ECT. However, coexisting diseases like pheochromocytoma, aortic/cerebral aneurysms, decompensated heart failure, cardiac rhythm disturbances, and major orthopedic fractures present grave risks and challenges.¹⁷

Specific Cardiac Pathologies

In general, hemodynamic fluctuations should be prevented/treated aggressively. Stimulus dose titration and subthreshold stimulus should be avoided.

Congestive cardiac failure: Medical therapy should be continued, and cardiac failure should be well-compensated. ECT resulted in only minor complications (benign arrhythmias) without increasing mortality risk in patients with reduced LV systolic function.¹⁸ Echocardiogram is recommended when baseline exercise capacity is < 4 METs.

Ischemic heart disease: Pre-ECT cardiac consultation and stress testing/angiography are advisable. All antihypertensive medications including angiotensin-converting enzyme-inhibitors (ACE-I) and angiotensin-receptor blockers (ARBs) can be continued. The continuation of diuretics has not been found to increase the risk of hypotension.¹⁹ A/ch is usually withheld to avoid tachycardia. A postprocedure ECG should be considered for assessing any new ECG changes.

Valvular heart disease: AHA/ACC places patients with severe valvular heart disease in high-risk category for morbid, high risk procedures, and ECT may increase the burden on an already compromised cardiac function. Tight control of hemodynamics and an overnight stay in high-dependency unit (HDU)/ICU unit would be a wise approach.

Severe aortic stenosis: Hypotension, tachycardia, and decrease in preload/SVR/cardiac contractility are poorly tolerated.²⁰ ECT can precipitate cardiac failure. A normally functioning prosthetic valve poses no additional risk.

Mitral stenosis (MS): No published literature exists with regard to ECT treatment in patients with severe MS. In severe lesions, surgical replacement or repair should be considered prior to ECT.

Regurgitant lesions: Data is lacking about the management of patients with severe valvular regurgitation lesions.

Patients with cardiac conduction blocks (uncomplicated): Corrected QT (QTc) prolongation is not a contraindication to ECT, barring the risk of ventricular tachycardia. Attempts should be made to correct the QTc by adjusting medications known to prolong QTc (psychotropic medications, antibiotics, antiarrhythmics).²¹ ECT itself can cause temporary prolongation of QTc dispersion.²²

Thromboembolic disease/atrial fibrillation (AF): Risk of pulmonary embolism exists, so location of deep venous thrombosis (DVT) should be determined, and anticoagulation should be continued.²³ Increasing the dose of neuromuscular relaxant is advised. Rate control and anticoagulation are advised for patients in AF to prevent embolic stroke.²⁴

Patient with pacemaker: ECT usually does not cause electromagnetic interference, as electrical stimulation is far away from the device (> 15 cm). The seizure myopotentials or Sch-induced fasciculations may inhibit the pacemaker, causing cardiac arrest in a pacemaker-dependent patient. Therefore, nondepolarizing muscle relaxant (NDMR) would be a better choice. For the same reason, ketamine, etomidate, and shivering should be avoided. Pacemaker should be converted into VOO mode.²⁵

With automatic implantable cardioverter defibrillator (AICD): Device should be reprogrammed, and antitachyarrhythmia functions should be disabled.²⁶ External defibrillator, temporary pacemaker, and full-resuscitation equipment should be ready. Adequate grounding, continuous ECG (in diagnostic mode), and pulse plethysmography should be present. A trained programmer or cardiac electrophysiologist should preferably be available. Before and after each ECT treatment, battery status should be checked and reprogramming should be done.

Brugada syndrome is classically identified on ECG as right-bundle branch block (RBBB) and ST elevation on right heart leads (V1 to V3), with no underlying structural heart disease. Other manifestations are ventricular arrhythmias, syncope, and sudden death. Class I anti-arrhythmic agents, β -blockers, neostigmine, and α -agonists are contraindicated.²⁷ Vagotonic agents like Sch can induce ST-segment elevation without coronary spasm.²⁸ Konishi et al have used rocuronium and sugammadex for ECT.²⁸ Caution is advised with regard to the use of propofol and bupivacaine (www.brugadadrugs.org).

Abdominal aortic aneurysm (AAA): Common sense approach would suggest that ECT should increase the risk of rupture for any weak-walled, distorted blood vessel like aneurysm. However, Mueller et al have found safe conduct of ECT in 8 patients with unruptured AAA.²⁹ They urge caution and recommend consultation with vascular experts in AAAs measuring > 5.5 cm diameter in men, > 5.0 cm in women, and in those with expansion of > 1.0 cm diameter in 1 year. Strict control of hemodynamics in the peri-ECT period is advised.

CVS complications are the most common form of complications associated with ECT. In a large retrospective study of 17,394 ECT treatments, the overall incidence of complications was 0.92%, with cardiac complications being the most common. Among cardiac, self-limiting arrhythmias were the most common.³⁰

Depression is independently associated with increased cardiovascular morbidity and mortality, so ECT becomes inevitable.³¹ If time permits, BP control should be attempted in patients with uncontrolled hypertension (mild-to-moderate elevation should not delay ECT). If possible, ECT should wait for 6 weeks after recent acute MI to prevent major adverse cardiac events.³²

Hemodynamic changes may continue in the immediate post-ECT period, so monitoring should continue in the postanesthesia care unit (PACU). At all the stages of the procedure, oxygen supplementation is mandatory. If seizure duration exceeds 60 seconds, it should be aborted to prevent myocardial stress. Maintenance ECT (MECT) is administered once in 2 or 3 weeks for many years to prevent relapse in refractory psychiatric disorders. During this time, patients might develop new comorbidities, or the severity of existing disease increases. Accordingly, the patient's family needs to be explained about the associated increased risk with the subsequent ECT treatment compared with the previous ones.³³

Central Nervous System Considerations

The sympathetic surge associated with ECT leads to sudden and significant increase in cerebral blood flow (CBF) and CBF velocity (CBFV).³⁴

Specific CNS Pathologies

Patients with intracranial tumor: ECT can lead to increase in intracranial pressure (ICP) in patients with compromised intracranial compliance. ECT causes hypertensive surge, and breakdown of blood-brain barrier, leading to mild cerebral edema.³⁵ Up to the late 1960s, ECT was considered an absolute contraindication in patients with intracranial space-occupying lesions (ICSOL) because of rapid neurological deterioration after ECT.³⁶ Now, ECT has been safely used in patients with primary (mostly meningiomas) and metastatic brain tumors.³⁷

A small, solitary ICSOL, which is not associated with mass effect or focal neurological deficits, does not pose any additional risk for ECT. However, lesions causing increased ICP should be assessed for risk-benefit ratio. Neurosurgical consultation should be obtained and hemodynamic changes of ECT be minimized. Antiedema measures like steroids, diuretics, and hyperventilation may be useful, although there are no supporting trials. If a patient is receiving MECT, serial imaging should be done to monitor for increase in the size of lesion.

Intracranial aneurysm: There is increased risk of ICH in larger aneurysms, posterior circulation aneurysms, and in smokers. Reducing ICP can increase transmural pressure and thus the risk of rupture. BP should be maintained within 15 to 20% of baseline with invasive BP monitoring. ECT has been administered safely in patients with unruptured, ruptured, and treated intracranial aneurysms, although peri-ECT aneurysmal rupture has been reported.³⁸

Active cerebral hemorrhage, recent cerebrovascular accident and active central nervous system (CNS) infections are only relative contraindications to ECT. In recent ICH, imaging may be done to confirm that the episode has resolved, and it may be prudent to wait for at least 30 days before administering the ECT.

There is no increased risk in patients with normal-pressure hydrocephalus or ventriculoperitoneal shunt.

In patients with previous craniotomy, skull defect, or in the presence of intracranial metal on one side, unilateral ECT with electrode placement on the opposite is advised.³⁹ The tissue resistance is low at the skull defect site, leading to lower current requirement.⁴⁰

Idiopathic intracranial hypertension is a relative contraindication to ECT, due to risk of herniation. In these patients, ECT may be necessary, because of risk of depression related to the disease per se. Before ECT, normalization of cerebrospinal fluid (CSF) opening pressure (using medications like acetazolamide, furosemide or topiramate) should be ensured. ECT may also help in reducing CSF pressure.⁴¹

Epilepsy: ECT may predispose to seizures.⁴² On the contrary, in refractory epilepsy, ECT may terminate or decrease

the frequency of seizures, provided anticonvulsants are continued. Discontinuation of medications can precipitate status epilepticus (SE). The mechanism of anticonvulsant action of ECT is probably by facilitation of gamma-aminobutyric acid (GABA)-ergic transmission, promotion of neurotrophic factors, and effects on synaptic plasticity.⁴³

In organic dementia and pseudo-dementia, ECT is safe and effective in treating the associated behavioral symptoms, with cognitive improvement by way of alleviating negative cognitive effects of depression. However, there is higher incidence of post-ECT confusion and agitation for which spacing the ECT treatments, administering unilateral ECT, and lowering the stimulus dose may help.⁴⁴

Parkinson's disease: ECT increases dopaminergic function, and helps to improve depression and motor symptoms.⁴⁵

Traumatic brain injury: It is advisable to wait for the resolution of acute phase. The electrodes should be placed equidistantly as far as possible from the lesion to avoid an excessive concentration of electrical stimulation.⁴⁶

Deep brain stimulators are at risk of damage and should be switched off prior to ECT. ECT is safe in patients with intracranial metallic objects and do not pose a contraindication.⁴⁷

Sch should be avoided in patients with neurological disorders causing motor weakness such as poststroke or polio and in other etiologies associated with prolonged immobilization.

Considerations in Respiratory Illnesses

Chronic obstructive lung disease (COPD): Optimization of patients with COPD with regard to medications (β -2-agonists, A/ch, corticosteroids, cessation of smoking, antibiotics) is advised, unless ECT is emergent. Theophylline and aminophylline use can cause prolonged seizures and SE.⁴⁸

Bronchial asthma: In a retrospective review involving 34 patients, 4 patients developed post-ECT exacerbations of asthma, all of which were treatable. Augmenting bronchodilator therapy in patients with moderate-to-severe asthma along with appropriate antibiotics made ECT course possible in all patients.⁴⁹ Methylxanthines are omitted 24 hours prior to ECT due to risk of arrhythmias and SE.

Endocrine Diseases

Altered cortisol status: Depression is associated with both hypocortisolism and hypercortisolism. Hypocortisolism is more prevalent in major depression. In Addison's disease, glucocorticoid supplementation improves depressive symptoms. In some patients, depressive symptoms persist despite seemingly adequate treatment. Heijnen et al opine that in some forms of Addison's disease, persistent dysregulation of hypothalamic-pituitary-adrenal (HPA) axis unresponsive to exogenous steroids caused treatment-resistant depression (TRD).⁵⁰ A patient with TRD later turned out to be a case of adrenal adenoma, causing Cushing's disease.⁵¹ Usually, patients on chronic steroids are administered additional doses of steroids during preoperative period to cope with the added stress of surgery and recovery. Rasmussen et al concluded that there was no need for "stress doses" of steroids, and regular morning dose of steroid prior to ECT would suffice.⁵²

Eliciting Seizures in Patient with Missed Seizure

Generalized motor seizure of at least 20 seconds is regarded as the minimum criteria for therapeutic response to ECT. Other parameters of seizure quality like postictal suppression index (PSI), interhemispheric coherence, peak heart rate (PHR), midictal amplitude, and extent to which stimulus intensity exceeds the seizure threshold are key determinants of the efficacy and speed of response to ECT.⁵³ When a clinician is not able to elicit a generalized seizure during ECT, it is called missed or abortive seizures. If increasing the stimulus intensity fails to elicit convulsion, proconvulsants should be tried.

Caffeine, IV or oral, increases the seizure duration in a dose-dependent manner without affecting seizure threshold. During the second ECT session, pretreatment with IV caffeine at a dose of 242 mg over 30 seconds, 5 minutes later followed by stimulus, decreased the stimulus intensity in contrast to placebo.⁵⁴ Dose of caffeine was further increased by 242 mg in the subsequent ECT session if the seizure duration remained at 20 to 30 seconds. Caffeine is associated with faster rate of recovery from depression and minimizes the need for higher stimulus dose to elicit seizure.⁵⁵ However, caffeine dose may have to be increased with progressive ECT sessions.

Theophylline (2 mg/kg) or aminophylline (3–5 mg/kg) IV, 10 minutes before ECT, has been shown to lengthen the seizure duration with a risk of precipitating SE.^{56,57}

Most of the anesthetics are anticonvulsants. The magnitude of anticonvulsant action varies between drugs and their dosage. Seizure duration is reduced with propofol compared with barbiturates. In a retrospective study comparing ketamine, etomidate, thiopentone, and propofol, the seizure quality and duration was best with ketamine and etomidate, and worst with propofol.⁵⁸ Etomidate has been shown to increase seizure duration and lower stimulus intensity compared with thiopentone.⁵⁹

Ketamine has a significant antidepressant effect of its own. There is significantly higher treatment effect with ketamine as add-on anesthetic.⁶⁰ It can lower the seizure threshold when added to propofol.⁶¹ The quality of seizure with respect to concordance between EEG and motor seizure and PSI is the best with ketamine. However, cardiovascular side effects, hallucinations, and longer recovery time can be expected. Moreover, bispectral index (BIS) monitoring is not reliable under ketamine anesthesia.

Opioids have a dose-sparing effect on anesthetic agents. Alfentanil (250–1000 mcg), combined with anesthetics, increases seizure duration.⁶² Concurrent administration of remifentanil (0.5–1 mcg/kg) improves seizure duration, provided the dose of anesthetic is reduced.⁶³ Remifentanil, when added to propofol, may also improve some indices of seizure quality.⁶⁴ Studies comparing propofol versus sevoflurane with respect to seizure duration have reported mixed results.^{65,66}

The anesthetic doses are usually kept to minimum during ECT in view of their anticonvulsant attribute and to allow early recovery. Nonetheless, without further reducing the dose of the anesthetic, if the peak anesthetic action is allowed to wear off before giving ECT stimulation, seizure

prolongation can be achieved. In this context, BIS may help to optimize the timing of ECT stimulus. A linear positive correlation between BIS value just before stimulation and the duration of seizure has been reported.⁶⁷ Waiting for BIS to increase by 10 to 20% effectively prolonged seizure duration without adverse effects.⁶⁸ Similar results have been reported with methohexital.⁶⁹ With thiopentone, a BIS of 55, and with propofol, a BIS of 47 has been suggested as the lower limit for inducing seizures.^{70,71} Lemmens et al, however, could not demonstrate a relation between increasing BIS and seizure duration.⁷²

Longer anesthesia-stimulation time interval (ASTI) is also associated with higher PHR, which is an indicator of spread of seizure to diencephalon, the site of therapeutic effect of ECT. BIS has also been found to positively correlate with seizure markers (PSI, concordance, coherence, and PHR).⁷³ High preictal BIS also translated into number of ECT treatments required.⁷⁴ However, this strategy places the patient at risk of awareness and violent seizures.⁷⁵

Hyperventilation, prior to stimulation positively, impacts seizure duration. Further, there is significant and synergistic influence of transcutaneous tissue partial pressures of oxygen and carbon dioxide ($tcpO_2/tcpCO_2$) on seizure quality. Higher $tcpO_2/tcpCO_2$ was associated with lower stimulation energy and better seizure quality.⁷⁶ Thus, preoxygenation is not only a safety measure but also helps in improving seizure quality by causing hyperoxia.

Other adjunctive strategies to counter seizure resistance are as follows: changing to unilateral stimulation, decreasing the frequency of treatments (from thrice weekly to twice weekly), decreasing pulse width, and withholding anticonvulsant medications if any.

The techniques to prolong seizures may not necessarily translate into improved outcomes. However, they may allow for lower stimulus energy, low levels of absolute electrical dosage, and fewer treatments, thereby enhancing efficacy and reducing cognitive side effects.

Conclusion

Providing anesthesia for ECT sometimes becomes arduous in the presence of coexisting diseases, particularly when ECT emerges indispensable. In this situation, the risk to the patient can be curtailed, using techniques described above. In other situations, where time permits, maximization of treatment strategies and involvement of specialists can be done to scale down the complication rate. Overall, the risk seems to be low, provided adequate measures are taken. There is a need for patient-specific individualized approach to ECT with effective communication between psychiatrist and anesthesiologist.

Conflict of Interest

None declared.

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