
REVIEW ARTICLES

UPDATE ON ANESTHESIA CONSIDERATIONS FOR ELECTROCONVULSIVE THERAPY

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Abstract

Depression is diagnosed in 14 million Americans every year, and pharmacotherapy is the standard treatment. However, in approximately 50% of patients, pharmacology intervention does not resolve depression. Electroconvulsive therapy (ECT) has been a mainstay as a treatment option for treatment-resistant major depression since its inception in the 1930s. It has also been shown to be effective in treatment-resistant mania and catatonic schizophrenia. The complication rate of ECT has improved from 50% in the 1960's to almost anecdotal adverse events, similar to the morbidity and mortality seen in minor surgery and childbirth. Although anesthetic agents are administered briefly, many patients experience significant fluctuations in physiologic parameters. The clinical anesthesiologist must be aware of these changes as well as have an understanding of perioperative pharmacological interventions. ECT is a proven therapy for select psychiatric patients, and appropriate anesthesia is a critical part of successful ECT. Careful review of the patient's medical history may reveal pertinent anesthetic considerations.

Introduction

Electroconvulsive therapy has earned an evidence based niche in modern day psychiatry as a treatment for refractory major depression, mania and catatonic schizophrenia^{1,2}. Traditionally, it was reserved primarily for the most gravely disabled patients who had failed numerous treatment options. However, ECT is a popular treatment option, and it is estimated that over 50,000 procedures take place annually³.

Depression is diagnosed in 14 million Americans every year, and pharmacotherapy is the standard treatment. However, up to 50% of patients do not respond to the initial round of pharmacologic treatment⁴. Unfortunately, studies have shown that subsequent medication trials have decreasing rates of successful remission⁵. A meta analysis has shown ECT to be more effective than single or combination pharmacotherapy in achieving remission of major depression⁶. Elderly patients are also known to be more refractory to pharmacotherapy, but it is estimated that up to 50% will improve with ECT⁷.

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Some patients may notice an improvement in their symptoms after one or two sessions of ECT. However, the current practice in the USA is to administer three treatments weekly for two to four weeks. In Europe treatments are administered twice weekly and remission rates are lower². The benefits of each individual treatment are theorized to build on one another, and response is usually attained between treatment 6 and 12. Some patients may require further treatment, or begin bi-monthly or monthly maintenance treatment if still symptomatic. To date, the exact mechanism of the effectiveness of ECT is unknown.

In the earliest days of ECT, prior to proper anesthetic and paralytic control, injuries such as vertebral fractures and chipped teeth were common in up to 50% of patients¹. The challenge for the anesthesiologist involves the complex effects, electrical and chemical, converging on the patient's central nervous system (CNS) at the time of therapy. The anesthesiologist must find the balance between under-medicating, which risks physical injury from convulsion, and over-sedation, which can raise the seizure threshold, preventing a sufficient therapeutic seizure. The complication rate of ECT has improved from 50% in the 1960's to almost anecdotal adverse events, similar to the morbidity and mortality seen in minor surgery and childbirth².

Physiologic Changes

ECT involves placing electrodes on the head and applying a current of electricity for 2-8 seconds sufficient enough to induce a seizure lasting at least 30 seconds, which is considered to be the minimally effective length of time for a therapeutic seizure⁸. Typically, electrode placement is either bitemporal or bifrontal⁹. Most treatment centers begin with right unilateral placement in an effort to reduce the cognitive side effects of treatment. Subjective impairment of memory is the most common disturbing adverse event associated with treatment, which usually fully recovers in the weeks to months following treatment⁸.

Understanding the physiologic changes that occur during the procedure allows the anesthesiologist to anticipate and limit complications. Most ECT morbidity results from adverse cardiovascular events¹⁰. As the electrical current is delivered, the parasympathetic

nervous system is stimulated, primarily through direct neuronal stimulation of the hypothalamus to the vagal nerve⁸ resulting in transient sinus bradycardia, or rarely, asystole. Shortly thereafter, the sympathetic nervous system is stimulated, releasing catecholamines, which usually causes tachycardia, hypertension, and may lead to arrhythmias.

ECT exerts several other effects on the central nervous system. When a seizure is induced, the metabolic demands of the brain are increased, doubling blood flow velocity through the middle cerebral artery in order to provide enough oxygen to the brain¹. There may also be a significant increase in intracranial pressure if the autoregulatory mechanism is overwhelmed by an increase in peripheral blood pressure. Therefore, the presence of cerebral aneurysms and arteriovenous malformations (AVMs) are relative contraindications for ECT. Other relative contraindications to ECT include conditions associated with increased intracerebral pressure, space-occupying cerebral lesions (tumor), recent intracerebral hemorrhage, pheochromocytoma, and recent myocardial infarction⁸.

Preoperative Management

There is little time on the day of ECT to perform a detailed evaluation and, therefore, patients are typically seen in a preanesthetic assessment clinic ahead of time and have received medical clearance. Any preoperative evaluation starts with a directed history and physical. Many of these patients can be quite old including over 90 years of age, with poor dentition and body hygiene, and be very apprehensive. Consent may be difficult to obtain. Preoperative fasting status must be determined as patients often do not comprehend the need for withholding food and drink prior to even a short general anesthetic. Special attention should be paid to cardiovascular health, especially a history of myocardial infarction, congestive heart failure (CHF), hypertension (HTN), aneurysms, and arrhythmias, which should result in additional cardiac evaluation. Any focal signs of CNS pathology should result in further neurological assessment¹¹. Documentation of an airway examination, allergies, and current medications should be included as well as informed written consent. Although bag masking is usually all that is required, airway equipment as indicated should be readily

available. Also, standard ASA monitors for blood pressure, heart rate, temperature, oxygen saturation, and capnography should be used. The seizure is monitored via electroencephalogram (EEG), and a bite block is used to protect the patient's dentition and tongue. Blood pressure and heart rate modulating agents need to be prepared if needed, typically including labetalol, esmolol, nitroglycerin and/or nicardipine.

The risk of arrhythmia, ischemia and hypertension can be diminished with oxygenation and pretreatment with appropriate medication, discussed below. In general, a demand pacemaker should be converted to a non-sensing asynchronous mode with a magnet to disable rate responsiveness at the time of treatment; however, different models and brands each have slightly different considerations. Cardiology consultation should be obtained, if warranted, prior to treatment¹². JCAHO requirement of side site documentation applies in all cases.

Drug Interactions

Usually, the condition requiring ECT is also treated pharmacologically, and potential drug interactions are important. Most psychiatric patients may be taking medications with anticonvulsant properties and these medications should be reduced prior to the procedure as tolerated since they increase the seizure threshold². Anticonvulsants should be continued only in epileptic patients, and the medication withheld the morning of treatment to prevent inhibition of an adequate seizure. Lithium is a popular agent used in bipolar disorder and as an augmentation agent in treatment of refractory major depression. Lithium has been associated with post-procedure delirium and can prolong the effects of succinylcholine⁸. Even though benzodiazepines may help reduce anxiety before the procedure, their use increases the seizure threshold². If they cannot be stopped, an option is to exchange long acting benzodiazepines for short-acting ones. Alternatively flumazenil may be used prior to the procedure and has shown no adverse effects on seizure quality¹. Theophylline can significantly lower seizure threshold and prolong seizure duration, and this medication should be minimized or discontinued if possible prior to treatment⁸. Caffeine augments electroconvulsive seizures¹³.

There are very limited studies regarding interference of ECT from continuation of various psychiatric medications but controversy continues in this area.

Intraoperative Considerations

Successful ECT anesthesia should meet several requirements according to the American Psychiatric Association¹⁴. It should be administered by an anesthesiologist who is responsible for both the anesthesia and the cardiopulmonary management¹⁰. In general, the anesthesiologist aims to help provide a procedure with an adequate seizure duration, free of associated side effects and injuries, and a quick recovery. The goal of ECT is to induce a 30-60 seizure². Therefore, only brief anesthesia is required. However, there are many anesthetic options that may be considered. The anesthetic's properties, including half-life, recovery time, effects on the CNS, autonomic nervous system and cardiovascular systems and interaction with other drugs must be considered to tailor an appropriate therapeutic plan.

Induction Agents

Methohexital was for many decades the most commonly used induction agent because it has minimal effect on seizure threshold while providing quick induction and recovery. It is designated as "first choice hypnotic" by the American Psychiatry Association¹⁴. Methohexital has a wide therapeutic dose range of 0.5-1mg/kg and is easy to titrate. Thiopental is another barbiturate, but compared to methohexital, results in shorter seizure duration and is associated with increased hemodynamic changes¹. Propofol has become a more popular choice, and multiple studies have been conducted comparing it to methohexital. Although propofol causes seizures of a shorter duration, the seizures are still of adequate length to provide full therapeutic benefit¹. Propofol has repeatedly been shown to be associated with a milder hemodynamic response than methohexital^{1,15}. Whether the drug provides a faster cognitive recovery is debatable. The most recent study available concluded that propofol is associated with a slightly quicker postictal recovery¹⁵. Because propofol can increase the seizure threshold, it should not be used in doses greater than 1mg/kg¹¹, or

in patients for whom an adequate seizure duration with maximal stimulus is not obtained.

Etomidate is another drug that may be considered. It has been repeatedly studied and is known to cause seizures of a longer duration than either propofol or methohexital. Unfortunately, etomidate is associated with increased confusion after ECT and longer recovery time¹⁶. However, because etomidate does not increase the seizure threshold, studies have shown its use requires significantly lower electrical currents¹⁷. Etomidate has also been associated with both reduced hemodynamic responses but higher rates of nausea. A recent small study comparing etomidate to propofol showed no significant difference in hemodynamic parameters¹⁸. Therapeutic doses in ECT range from 0.15-0.3mg/kg¹.

Volatile anesthetics, in particular sevoflurane, are also being studied for use in ECT. However, it has been concluded that sevoflurane offers no benefit over methohexital, yet is more time consuming to the physician¹⁶. One trial compared sevoflurane to thiopental and concluded sevoflurane lead to a quicker recovery with comparable hemodynamic changes¹⁹.

Muscle Relaxants

In addition to IV anesthetics, muscle relaxants are critical in ECT to prevent convulsions thus protecting the patient from musculoskeletal injury. Succinylcholine has been used in ECT since the 1950s and is still the muscle relaxant of choice³. It is contraindicated in patients with closed angle glaucoma, malignant hyperthermia potential, and malignant hyperthermia For ECT, doses up to 1mg/kg may be used¹¹. The anesthesiologist must be aware of patients with a history of pseudocholinesterase deficiency. When succinylcholine is contraindicated, mivacurium, rocuronium, or cis-atracurium are traditional alternatives (unless the patient has pseudocholinesterase deficiency). These agents are nondepolarizing muscle relaxants with significantly longer half-lives and thus, are not considered superior to succinylcholine in most instances.

Attenuation of Hemodynamic Responses

To attenuate the immediate parasympathetic discharge, which lasts 10-15 sec, pretreatment with IV

atropine or glycopyrrolate is generally recommended. There is some controversy in the literature about the use of atropine on every patient and some prefer glycopyrrolate because it causes less cognitive impairment since it does not cross the blood brain barrier¹⁶. The subsequent sympathetic discharge, which lasts 5-7 min can be attenuated by a wide variety of drugs including beta blockers, calcium channel blockers, nitrates, and the antiquated ganglion blockers.

Typically, the psychiatrist and anesthesiologist communicate as to which agents will be administered during ECT, as there are slight regional variations. For patients with cardiovascular complications, esmolol or labetalol have been shown to be the most effective in controlling heart rate and mean arterial pressure⁵. Labetalol reduces BP spikes and cardiac dysrhythmias, but may also be associated with a shorter seizure duration. Esmolol is slightly preferred by some because it reduces peak systolic BP a little more than labetalol¹. However, it may induce a dose-dependent bradycardia²⁰. Verapamil 0.1mg pre-ECT decreases both heart rate and BP²¹, while nicardipine has only been proven to lower the BP. Nicardipine has not been proven to lower cerebral blood flow and may cause a baroreceptor induced reflex tachycardia²⁰.

Studies have also looked at the effects of adding remifentanyl to a sedative hypnotic. Remifentanyl appears to have no effect on the seizure duration but is effective in lowering the HR and BP for up to 3 min after the procedure²². Some studies have shown a decrease in seizure duration when remifentanyl is added to methohexital, however in these studies, the dose of methohexital was also reduced by 40%.

The myocardium has been another area of study. Many studies have failed to show either new ECG changes or troponin isoenzyme elevations after ECT. Twenty-four percent of the patients in these studies had cardiovascular disease, including conduction abnormalities, recent MIs and existing regional wall motion abnormalities²³. Another study that used echocardiography to evaluate cardiac function during ECT found no evidence of new regional wall motion abnormalities and recommended beta blockers as the drug of choice to minimize heart rate and function changes²⁴.

In recent years research has attempted to correlate Bispectral index (BIS®) of electroencephalography (EEG) to the duration of and recovery from an ECT induced seizure. Entropy technology, from which several brands are now available with similar bioengineering, assesses the hypnotic state of an anesthetized patient. It may assist in indicating appropriate depth of anesthesia prior to seizure. White et al have shown that BIS® readings just after induction positively correlate to the duration of the seizure²⁵. This potentially allows the clinicians to predict relative sufficiency of the induced seizure; however, limited data is available and more studies are warranted.

Postoperative Concerns

After the treatment, the awake and hemodynamically stable patient should be monitored for at least 30 minutes, usually by a psychiatric nurse or assistant known to the patient. Mental status should be noted and the patient observed for post-ictal delirium. A short-acting benzodiazepine such as lorazepam may be needed for agitation. Headache and muscle soreness are the most common complaints and may indicate that an increase in succinylcholine may be warranted at the

next treatment. Any decrease in pulse oxymetry should be evaluated for possible aspiration. Chest auscultation, X-ray and even blood gas analyses may be indicated. If the patient remains asymptomatic, discharge may still be possible. The patient is allowed to resume normal activity as tolerated and any medications that were held prior to the procedure should be restarted. Patients should not work or drive on the day of treatment.

Conclusion

ECT is a proven therapy for select psychiatric patients, and appropriate anesthesia is a critical part of successful ECT. The anesthesiologist aims to provide amnesia and musculoskeletal relaxation without increasing the seizure threshold. First line drugs are propofol and succinylcholine. The hemodynamic changes following the electric current are usually the only ones requiring management. In general, beta-blockers control heart rate and BP. However the anesthesiologist is also responsible for managing cardiopulmonary complications that may be present before the procedure as well as those that arise during ECT. Careful review of the patient's medical history may reveal pertinent anesthetic considerations.

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