Anesthesia for ECT

Advanced, Clinical - Neurologic

Electroconvulsive therapy (ECT) is a procedure where a generalized epileptic seizure is purposely induced for the treatment of psychiatric disorders (including acute and chronic depression/mania) that are resistant to medical management. The initial reaction following application of the electric current is a parasympathetic response resulting in bradydysrhythmias and possibly sinus pause (1,2). The parasympathetic response is followed by a sympathetic response associated with tachycardia and hypertension. During the sympathetic response, systolic blood pressure may increase by 30-40% and heart rate may increase by 20% (or more). The typical effective seizure should have a duration of 20 to 50 seconds. The treatment includes a series of ECT performed 3 times a week for a period of 6 to 8 weeks.

Anesthesia for ECT requires both general anesthesia and paralysis and placement of a bite block . Induction is intravenous, followed by complete paralysis usually using a short acting muscle relaxant such as succinylcholine to minimize the amount of muscle contraction that occurs with the seizure.

The Selection of the induction agent or other medications used during the procedure should consider its effect on the seizure duration. **Pretreatment with glycopyrrolate** may reduce the incidence of bradycardia and decrease oral secretions following the initial parasympathetic response.

ECT: anesthetic agents and seizure duration

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According to Miller, "*Methohexital (0.75 to 1.0 mg/kg) is the most commonly used drug for ECT anesthesia and is considered the "gold standard"* ," although there are data to suggest that outcomes are no different between methohexital and propofol, despite the decreased seizure duration with propofol.

Paralysis does *not* have to be complete (intubation is rarely performed) – often 0.5 mg/kg SCh is used. An adequate seizure in ECT is defined as one which lasts greater than 30 seconds.

Other important interactions: **indirectly acting sympathomimetics can cause a hypertensive crisis** in patients receiving MAOIs; **lithium prolongs the action of NMBDs**

ECT: Anesthetic Agents and Seizure Duration

- Increased Duration: Etomidate
- No Effect: Methohexital, ketamine, remifentanil, alfentanil
- Shortened Duration: Propofol, midazolam, lorazepam, thiopental, thiamylal, lidocaine

Many different medications, including lidocaine, have been studied in hopes of attenuating the sympathetic response to ECT. **Lidocaine** , an amide local anesthetic, has been shown to be **ineffective in ameliorating the robust sympathetic response associated with ECT** . In addition, pre-treatment with **lidocaine is also associated with decreased seizure duration and a higher likelihood of patients requiring multiple applications of electric current during a single ECT session to achieve a therapeutic seizure.**

CARDIOVASCULAR DRUGS

Increases duration:

- Aminophylline
- Caffeine (often given pre-procedure to increase duration)

NO change in duration:

- Clonidine
- Esmolol
- Labetalol
- Dexmedetomidine
- Nifedipine
- Nicardipine
- Nitroglycerin
- Nitroprusside

Decreases duration:

- Diltiazem
- **Lidocaine (While lidocaine has been shown to reduce the HR changes associated with ECT, lidocaine significantly reduces seizure duration.)**

Methohexital – gold standard for ECT, no change of seizure duration but better tolerated than etomidate secondary to ability to blunt hemodynamic response and better than propofol and thiopental because it does not decrease seizure duration.

Etomidate – it is also associated with myoclonus, a longer time to wake than methohexital and also does not block the hypertension and tachycardia often associated with ECT. Increases seizure duration.

Propofol and thiopental decrease seizure duration but blunt hemodynamic response.

Opioids are useful in the fact that they allow a lower dose of the anesthestic agent to be used, thus allowing faster wakeup and if using propofol or thiopental it will decrease the amount so the dose related decrease will help lengthen the seizure (i.e. you don't have to give as much propofol so it won't shorten the seizure as much).

Hyperventilation (hypocarbia) will lengthen seizure duration and thus effectiveness of ECT.

ECT: side effects

Clinical - Neurologic

The stimulus leads to **increases in cerebral blood flow and increased ICP. Initially, the seizure activity causes a large parasympathetic discharge** with bradycardia, atrial or ventricular premature beats, and occasional asystole. This is **followed by sympathetic discharge** causing tachycardia (maximal at two minutes), hypertension, ST-segment depression and T-wave inversion not associated with myocardial enzyme changes, and rarely ventricular tachycardia. According to Miller ,"ECT has been found to be relatively safe even in high-risk cardiac patients, if careful management is provided."

Glucose homeostasis is affected variably with both improved and worse control – interestingly, patients with NIDDM generally experience better control, whereas those with IDDM experience worse control.

ECT: Side Effects

- Initial parasympathetic response
- Sympathetic discharge (maximal at two minutes)
- EKG changes unaccompanied by enzyme leak
- Alterations in glucose control

ECT: Contraindications

Advanced, Generic Clinical Sciences

Before discussing contraindications, it is important to first understand the physiologic effects of ECT. These include :

- Large increases in cerebral blood flow and intracranial pressure
- Initial parasympathetic discharge manifested by bradycardia, occasional asystole, premature atrial and ventricular contraction, hypotension and salivation
- Following parasympathetic reaction is a sympathetic discharge associated with tachycardia, hypertension, premature ventricular contractions, and rarely, ventricular tachycardia and ECG changes, including ST-segment depression and T-wave inversion, may also be seen.
- Glucose homeostasis is also affected. Hyperglycemia seen in insulin dependent patients

Absolute contraindications :

- Known pheochromocytoma

Relative contraindications : The risk of the patient's psychiatric illness, side effects of antidepressant medications must be weighed against the risk of ECT and anesthesia. These conditions include :

- Increased intracranial pressure, ok if there is not a mass effect
- Brain tumors, same recommendation as above
- Recent stroke- ECT has been performed successfully
- Cardiovascular conduction defects. Pacemaker is not a contraindication to ECT- AICD function can be deactivated and magnet should be available if needed
- High-risk pregnancy- OB consult and fetal monitoring is recommended
- Aortic and cerebral aneurysms
- Asthma/COPD- some suggest that you should discontinue theophyline because of its potential to cause status epileptics

Recommendations:

- Delay ECT for patients with unstable angina, decompensated heart failure, or severe symptomatic valvular disease until these conditions are stabilized or optimized. Cardiology consultation may be of benefit
- For high-risk neurosurgical lesions including recent stroke and brain tumor, neurosurgical consultation is recommended
- Diabetic patients should hold oral hypoglycemic, short acting insulin and halve their long acting dose with fasting
- Warfarin can be continued in high risk patients with INR <3.5
- In severe GERD antacids can be taken or intubation considered

Postoperative Visual Loss: Risk Factors

Advanced, Organ-Based and Clinical Sciences

Postoperative visual loss after non-ocular procedures is a rare but devastating complication after general anesthesia. Visual loss may or may not be permanent and can be unilateral or bilateral. Risk factors vary depending on the etiology of post-operative visual loss.

Posterior ischemic optic neuritis(ION) is the most common cause of post-operative visual loss followed by anterior ION. Posterior ION is classically seen after spine surgery while anterior ION is more commonly implicated in cardiac surgery.1

Posterior ION (PION): prone position, especially if Wilson frame is used, surgery duration(particularly if >6hrs), age > 50yrs, male gender(estrogen is thought to be protective), anemia(particularly if blood loss is >1 L), hypotension(MAPs <70mmHg), and excessive fluid resuscitation(colloid is thought to be less likely to cause edema than crystalloid but data is unclear). Obesity is increasingly recognized as a risk factor as increased intraabdominal pressure can lead to increased central venous pressure and compression of optic nerves. Use of vasopressors is also considered to be a risk factor, particularly if they are used in lieu of blood transfusion for hypotension and hypovolemia.2

Anterior ION (AION): cardiac surgery but can also be seen in spine surgery, position(particularly Trendelenburg), surgery duration, hypotension, anemia and male gender also seem to be risk factors.3

Central retinal artery occlusion (CRAO): cardiac surgery where atherosclerotic plaque can be dislodged by catheters into retinal circulation, Giant Cell Arteritis, prior transient ischemic attacks, carotid artery stenosis, embolic stroke and hypercoagulable states.4

Sources

Lee A, Golnik K. Perioperative Ischemic Optic neuropathy. American Academy of Ophthalmology.; Jan. 2007

Wolpaw J, Hughes J. Anesthesia and Critical Care Reviews and Commentary. Episode 22: Postoperative Visual Loss

Doran, M. Perioperative Retinal Artery Occlusion: Risk Factors in Cardiac Surgery. Ophthalmology; 2017

Ho VT, Newman NJ, Song S, Ksiazek S, Roth S. Ischemic optic neuropathy following spine surgery. J Neurosurg Anesthesiol. 2005;17(1):38-44.

Rubin DS, Parakati I, Lee LA, Moss HE, Joslin CE, Roth S. Perioperative Visual Loss in Spine Fusion Surgery: Ischemic Optic Neuropathy in the United States From 1998 to 2012 in the Nationwide Inpatient Sample. Anesthesiology. 2016 Sep;125(3):457-64

Unilateral blindness: etiology

Physiology - Neurologic

Post-operative blindess is a devasating and fortunately rare complication of general anesthesia. The incidence of post-opertiave blindness is low (Ref. 1). In one retrospective study, only one out of 60965 patients suffered permanent loss of vision. (Ref. 2)

Post-operative visual loss typically occurs as an ischemic sequelae of external compression of the globe, hypotension, anemia or embolism (Miller 2010). The causes of post-operative unilateral blindness and their possible mechanisms include :

1. Ischemic optic neuritis (ION): anterior ION usually occurs after cardiac surgery and posterior ION typically occurs after spine surgery. This is the most common cause of perioperative vision loss. (Ref. 2). It can be unilateral although most cases are bilateral. ION results from hypoperfusion or decreased oxygen delivery to the optic nerve either in the anterior portion (AION) or the posterior portion (PION). Clinical findings of ION include painless visual loss, and impaired color vision. Fundoscopic exam findings include optic disc edema in AION, however in PION the optic disc may appear normal initially. Visual defects tend to be altitudinal, or loss of vision occurs with respect to the horizontal meridian. (Miller 2010)

2. Central Retinal Artery Occlusion (CRAO): may occur as a result of external compression on the eye causing raised intraocular pressure that blocks blood flow through the central retinal artery, retrobulbar hemorrhage from vasuclar injury or from an emboism. Clinical findings include “painless blindness,” “cherry red macula” and narrowed retinal arteries on fundoscopic exam. Causes of CRAO include 1) raised intraocular pressure, 2) embolism, 3) decreased venous outflow. (Miller 2010)

3. Branch retinal artery occlusion (BRAO): occurs most commonly from an ebolism in one branch of the central retinal artery. BRAO presents as loss of vision in a definied distribution of the arterial branch affected. Clinical presentation may be silent if the vision loss is peripheral. Embolism may be identified and characterized by fundoscopic examination. (Miller 2010)

4. Acute angle glaucoma: results in raised intraocular pressure from blockade of the aqueous humor outflow tract from obliteration of the space between the iris and lens. Clinical findings include a painful and red globe, blurry vision, headache, nausea and vomiting. (Miller 2010) Although a cause of unilateral blindness, case reports have described bilateral acute angle closure glaucoma after spine surgery. (Ref. 3, Ref. 4)