CME

Individualized Anesthetic Management for Patients Undergoing Electroconvulsive Therapy: A Review of Current Practice

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Electroconvulsive therapy (ECT) remains an indispensable treatment for severe psychiatric illness. It is practiced extensively in the United States and around the world, yet there is little guidance for anesthesiologists involved with this common practice. Communication between the anesthesiologist and the proceduralist is particularly important for ECT, because the choice of anesthetic and management of physiologic sequelae of the therapeutic seizure can directly impact both the efficacy and safety of the treatment. In this review, we examine the literature on anesthetic management for ECT. A casual or "one-size–fits-all" approach may lead to less-than-optimal outcomes; customizing the anesthetic management for each patient is essential and can significantly increase treatment success rate and patient satisfaction. (Anesth Analg 2017;124:1943–56)

Electroconvulsive therapy (ECT) is administered to an estimated 100,000 patients per year in the United States, and an estimated 1,000,000 patients per year worldwide. ECT is indicated for the treatment of severe and treatment-resistant depression, psychosis, bipolar disorder, and catatonia. Movement disorders such as Parkinson's disease and the self-injurious behavior associated with severe autism may also respond to ECT, but they are considered less standard indications.[1] The procedure involves the induction of a therapeutic seizure to the patient under general anesthesia. The anesthesiologist must be intimately familiar with both the physiologic effects of ECT and the propensity for the administered drugs to interfere with the treatment.[2] Our review of the current literature suggests that a more individualized approach to ECT anesthesia will result in better outcomes, reduced costs, and fewer complications.

PREANESTHETIC ASSESSMENT

ECT generally is considered a low-risk procedure, but many patients referred for therapy present with significant medical comorbidities with the potential to complicate anesthetic management. As with any patient who is to receive an anesthetic, a complete and thorough preanesthetic evaluation is mandated. This evaluation can be complicated in the psychiatric patient, who may be uncommunicative, delusional, or paranoid. Proper evaluation may take more time than usually required for the average surgical patient and may involve contacting multiple treatment providers or family members to corroborate historical information. Given the considerable physiologic response to the catecholamine surge associated with the therapeutic seizure, it is especially important to identify any undiagnosed underlying cardiac disease, because this has been associated with increased complication rates.[3] Preprocedure tests for the patient with diagnosed or suspected cardiac disease presenting for ECT should include a baseline electrocardiogram (ECG) and possibly a consultation with a cardiologist, who may recommend further studies such as an echocardiogram or stress test before treatment. For the young, healthy patient, the pre-ECT workup may consist of only a basic metabolic panel and ECG; other tests may be indicated based on the patient's medical history. Local hospital policy may dictate specific preprocedural testing for patients receiving general anesthesia.

Coexisting Medical Illness

The most common medical illnesses relevant to the anesthetic management of the patient referred for ECT involve perturbations of the cardiac, pulmonary, neurologic, and musculoskeletal systems.

Cardiac Comorbidities. Patients with significant cardiovascular disease, that is, patients with a history of previous myocardial infarction, coronary artery disease (CAD), or previous cardiac intervention such as coronary artery bypass grafting, percutaneous coronary intervention with either angioplasty or stent placement, pacemaker or implantable cardioverter-defibrillator (ICD) placement, should be evaluated by a cardiologist before the treatment to determine the risk for a peritreatment event and to ensure medical optimization.[4] The physiologic stress associated with ECT is tolerated by the vast majority of patients without sequelae, but patients with unstable CAD, decompensated congestive heart failure, preexisting arrhythmias or implantable cardiac devices, and significant valvular disease require further evaluation and medical optimization.[5] Many patients who receive ECT fall into the intermediate or high cardiac

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risk category, but despite this, the mortality rate remains extremely low, most recently estimated to be less than 1 in every 75,000 treatments.[6] Although death during treatment is extremely rare, when it does occur, it is associated most commonly with cardiac arrhythmias, most often asystole resulting from significantly increased parasympathetic tone.[7]

Pulmonary Comorbidities. Patients with preexisting pulmonary disease may be at increased risk for post-treatment sequelae, such as delayed return of spontaneous respiration or rupture of preexisting pulmonary blebs related to aggressive hyperventilation. In patients requiring supplemental oxygen therapy, it may be prudent to obtain preprocedure arterial blood gas measurement before the first treatment to identify patients with a chronically elevated PCO_2 in whom delayed return of spontaneous ventilation due to hyperventilation may affect posttreatment management.[8]

Gastroesophageal Reflux Disease. Gastroesophageal reflux disease (GERD) presents a theoretical concern for the patient who will receive ECT because the airway rarely is protected against aspiration, because intubation (and the airway protection it provides) is not part of routine airway management during ECT. The vast majority of patients with mild or well-controlled GERD are not at increased risk for aspiration during ECT, and intubation is not performed under these circumstances.

Patients with mild, asymptomatic GERD should continue their medications throughout the course of treatment, and no additional considerations are necessary. If the patient is experiencing symptoms despite medical treatment, gastric acid–neutralizing agents such as citric acid/sodium citrate should be administered immediately before ECT.[9] In cases of more severe GERD, histamine (H2) receptor antagonists or promotility agents such as metoclopramide may be administered intravenously (IV) 30 minutes before treatment if the patient is not already receiving one of these medications.[10]

Neurologic Comorbidities. Baseline neurologic function is important to assess so that the patient can be evaluated properly after treatment. Patients with suspected intracranial lesion or elevated intracranial pressure may require an imaging study as part of their pre-ECT evaluation and those with known or suspected brain tumor or other pathology with or without suspected intracranial hypertension should receive clearance by a neurologist and/or neurosurgeon before the institution of ECT. These include patients with a history of cancers known to metastasize to the brain, a history of treated brain cancer, and patients with previous craniotomy. With proper management, some patients with neurologic comorbidities may still be treated safely. One case describes the successful treatment of a patient with a surgically repaired cerebral aneurysm,[11] and 2 reviews suggest that intracranial aneurysms present no contraindication to ECT.[12,13]

Musculoskeletal Comorbidities. Musculoskeletal disorders are common in the elderly population. Osteoporosis is especially common in older patients presenting for ECT, particularly in women. Adequate neuromuscular relaxation is important to prevent the rare complications of bone fractures and joint dislocations.[14–16] For patients who are at increased risk for significant morbidity should the neuromuscular block be incomplete, such as those with cervical spine disease or recent orthopedic surgery, an increased dose of muscle relaxant may be given to ensure full paralysis.

The Contribution of Psychiatric Illness to Medical Morbidity

Psychiatric illness has the potential to significantly adversely affect physical health and has systemic inflammatory effects on multiple organ systems.[17] Recent investigations have presented evidence that depression increases the risk for adverse medical outcomes in patients with CAD experiencing acute coronary syndrome,[18] increases the risk for ischemic stroke,[19] and is an independent risk factor for subclinical inflammation in healthy individuals.[20,21] Because the connection between psychiatric illness and CAD may not be appreciated fully by the majority of physicians, the anesthesiologist evaluating a patient before ECT should maintain an elevated index of suspicion for undiagnosed CAD.

Concurrent Psychiatric Medications

ECT is often recommended after a patient has failed to respond fully to multiple antidepressant medications, and it is not uncommon for these patients to present for treatment while still receiving these medications.[1] Many psychiatric medications have the potential to complicate anesthetic management and may also be unfamiliar to the anesthesiologist. Common antidepressant medications in this population include selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs); less common antidepressants are the tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs). Other commonly encountered psychotropic medications include mood stabilizers (eg, carbamazepine, valproate, and lithium), antipsychotics (eg, chlorpromazine, quetiapine, risperidone, and olanzapine), and benzodiazepines.

Selective Serotonin Reuptake Inhibitors. Many patients undergoing ECT are treated safely while taking full doses of SSRIs or SNRIs. However, some rare and/or potential risks do exist. Patients who are taking SSRIs are at risk for serotonin syndrome (hyperreflexia, agitation, and hyperthermia) if multiple drugs with serotonergic activity are administered. SSRIs also inhibit CYP2D6, which may lead to increased anesthetic requirements.[22] Sertraline has been reported to cause prolonged action of succinylcholine via decreased pseudocholinesterase activity, and patients on this agent may require a reduced dose of succinylcholine.[23]

Tricyclic Antidepressants. Patients maintained on TCAs may have an elevated resting heart rate due to the intrinsic anticholinenergic effects of the drug and are at increased risk for developing QRS prolongation or atrioventricular block leading to ventricular arrhythmias and hypotension.[24] In addition, the sedative effects may

contribute to a prolonged emergence after treatment.[2] The combination of ECT and TCAs, however, generally is considered safe.[25]

Monoamine Oxidase Inhibitors. Patients who are taking MAOIs are at increased risk for hypertensive crisis should direct or indirect acting sympathomimetic drugs or meperidine be administered. These patients are otherwise not at an increased risk for severe adverse hemodynamic events, provided they are on a stable dose. If the decision is made to discontinue therapy, MAOIs should be stopped 2 weeks before anesthesia to allow for MAO activity to return to baseline. If the decision is made to maintain therapy during ECT, it is recommended that the patient be on a stable dose of the MAOI to avoid an increased risk of arrhythmia.[26]

Bupropion. Bupropion is an antidepressant that lowers the seizure threshold and carries the risk of prolonged seizures during ECT. The risk is dose dependent and lower with the extended release formulation. This agent should be held the morning of treatment.[27]

Mood Stabilizers. Mood stabilizers include lithium and the anticonvulsants valproate and carbamazepine. Lithium has a narrow therapeutic index and multiple effects such as interference with antidiuretic hormone, cardiac arrhythmias, gastrointestinal disturbances, thyroid abnormalities, and tremor.[28] Lithium may cause increased confusion or delirium; thus, it is recommended to hold doses 12 hours before the treatment.[29] Lithium also may be held entirely for the course of ECT, although a recent review demonstrates the safety of combined treatment in certain clinical circumstances.[30]

Anticonvulsant mood stabilizers can inhibit the induction of a seizure during ECT.[31] Typically, the evening dose of anticonvulsant is held the day before ECT. Although clinical practices vary on holding anticonvulsants or reducing the dose during a course of ECT, a recent study demonstrated that treating with full dose anticonvulsants during a course of bilateral ECT resulted in a faster recovery, without effects on seizure parameters.[32]

Carbamazepine has effects on intrinsic enzyme activity, is a potent inducer of cytochrome P450 3A4, and may increase anesthetic requirements. Carbamazepine may cause hematologic side effects, as well as hyponatremia and liver function abnormalities. Rarely, it may prolong the action of succinylcholine.[25] Valproic acid may cause thrombocytopenia, abnormalities in liver function tests, and occasionally hyperammonemia. Perioperative exacerbation of valproic acid-related hyperammonemia has been reported.[33]

Antipsychotics. Antipsychotics such as chlorpromazine, quetiapine, and risperidone put patients at increased risk for developing extrapyramidal syndromes (akathisia, Parkinsonian symptoms, and tardive dyskinesia). A much rarer side effect, neuroleptic malignant syndrome, is characterized by hyperthermia, autonomic dysfunction, and muscle rigidity.[34] The combination of ECT and antipsychotics generally is well tolerated.[29] Antipsychotics may enhance the effects of central nervous system depressants and lower doses of anesthetics may be required.[35] Many psychotropic medications affect the activity of cytochrome P450 2D6, the genetically variable enzyme that metabolizes several beta-blockers and can impact their effects in the treatment of tachycardia or hypertension during ECT.[25] The use of ECT with clozapine is a safe and effective treatment option for refractory schizophrenia. Side effects of combining ECT with clozapine treatment typically are mild but may include tachycardia, sedation, confusion, or risk of aspiration due to drooling. The occurrence of cognitive effects or prolonged seizures is rare.[36]

Benzodiazepines. Benzodiazepines are sedating and synergistic with most commonly used anesthetic agents. They also have the potential to interfere with the generation of the therapeutic seizure. In most cases these drugs should be held 12 hours before the treatment. In some cases, if high doses of benzodiazepines are being used or if the patient cannot tolerate discontinuation, reversal with flumazenil (0.4–1.0 mg IV administered immediately before the treatment) may be required to elicit an adequate seizure.[37] In patients with high-seizure thresholds, the use of flumazenil has been reported in the absence of benzodiazepines to facilitate the ECT seizure (Table 1).[38]

Implanted Devices

As the elderly population grows, the number of patients presenting for ECT with cardiac implantable electronic devices (CIEDs) continues to increase. In addition, new treatments for refractory psychiatric disorders, such as obsessive-compulsive disorder, involve the surgical implantation of deep brain stimulators (DBS). Because these patients often have concurrent refractory depression, the number of patients presenting for ECT with these devices also has increased. It is imperative that the anesthesiologist providing care for these patients be familiar with the current recommendations for intraoperative management of these devices.

Pacemakers and ICDs. Periprocedural management of the patient with a CIED should focus on maintaining proper device function. Modern CIEDs are extremely resistant to the effects of electromagnetic interference (EMI) but may still become damaged or reprogrammed under certain circumstances.[39] Although it is theoretically possible that EMI produced by the ECT stimulus itself or the use of a peripheral nerve monitor could interfere with device function, these sources usually are far enough away (greater than 15 cm) from the device so as to have minimal impact. Potential adverse outcomes include physical damage to the device or lead, including lead fracture or repositioning resulting in device failure, changes in device function such as inadvertent reprogramming, inappropriate discharge of an ICD, and inadvertent reset to backup mode.[40] The most likely source of interference during ECT comes from muscle movement due to fasciculations or poor muscle relaxation. Because of this, it is prudent to consider using an increased dose of succinylcholine in patients with CIEDs.

If a pacemaker is present, the anesthesiologist should determine whether the patient is pacemaker dependent. It should be noted that all ICD devices are potentially pacemakers, in that, they all retain the ability to overdrive pace tachyarrhythmias or pace asystole after a defibrillatory shock, but not all

Table 1. Strategy for the Management of Common Psychiatric Medication Administration During the Peri-ECT Period

Class	Effects	Recommendations
TCAs[a]	Tachycardia QRS prolongation Atrioventricular block	• Obtain baseline ECG. • Patient may continue taking medication.
MAOIs[b]	Hypertensive crisis Serotonin syndrome	• Avoid meperidine and all direct and indirect acting sympathomimetic drugs. • Avoid SS precipitants. • Patient may continue taking medication.
SSRIs[c]	Tachycardia Serotonin syndrome	• Obtain baseline ECG. • Avoid SS precipitants. • Patient may continue taking medication.
Bupropion	Prolonged seizures	• Hold the morning of treatment
Mood stabilizers[d]	May reduce or increase anesthetic drug requirements	• Discontinue via taper if possible before treatment or • Hold for 12 hours before treatment.
Antipsychotics[e]	NMS Extrapyramidal effects	• Have MH cart available. • Monitor patient temperature for 60 minutes post-ECT. • Patient may continue taking medication.
Benzodiazepines[f]	Sedative May interfere with seizure efficacy	• Discontinue via taper if possible before treatment or • Hold for 12 hours before treatment.

Abbreviations: ECG, electrocardiogram; MAOI, monoamine oxidase inhibitor; MH, malignant hyperthermia; NMS, neuroleptic malignant syndrome; SS, serotonin syndrome; SSRI, selective serotonin reuptake inhibitor; TCA, tricyclic antidepressant.
[a]Amitriptyline, imipramine, doxepin.
[b]Venlafaxine, fluoxetine.
[c]Phenelzine, moclobemide.
[d]Carbamazepine, valproate, lithium.
[e]Prochlorperazine, chlorpromazine, quetiapine, risperidone.
[f]Lorazepam, temazepam.

patients with an ICD are pacemaker dependent.[41] If the patient is not pacemaker dependent, a magnet should be available in case of device failure, but it does not necessarily need to be placed over the pulse generator during treatment. Placing a magnet over the pulse generator will put the device into an asynchronous mode until the magnet has been removed. If the patient is pacemaker dependent and the risk for interference is high, consideration should be made to having the device reprogrammed to asynchronous mode during treatment.

Management of an ICD is, for the most part, less complicated. Placing a magnet over the ICD renders the therapy function of the device inactive. The ICD will continue to monitor and record the patients' cardiac rhythm but will not deliver a therapeutic shock. Because of this, should a malignant arrhythmia be identified, timely removal of the magnet is essential. It is important to note that if a patient with an ICD is pacemaker dependent, placing a magnet over the pulse generator will only deactivate the ICD, it will not place the pacemaker into an asynchronous mode. In this instance, consultation with a cardiologist is indicated. In any case, it is important to have a backup form of external pacing and defibrillation available whenever a magnet is placed over a CIED (Table 2).

DBS, Cochlear Implants, and Other Metallic Objects. DBS has been used historically to treat essential tremor, dystonia, and Parkinson's disease not adequately controlled with medications, and is now being used as treatment for other neurological disorders such as obsessive-compulsive disorder.[42] The use of DBS for depression is an active area of research and several brain targets are under exploration in clinical trials. These devices are at risk for damage (physical lead damage) or generator reprogramming and interference related to EMI in much the same way that pacemakers and ICDs are. In addition, DBS devices may interfere with monitoring and other devices.[43] Because of these concerns, DBS devices should be switched off by a trained professional prior to ECT. Consultation with the neurosurgeon or device manufacturer can facilitate reprogramming.

Eight cases of ECT in patients with DBS devices have been reported thus far, all without complications.[44] Device management varied across these cases, with the DBS being either stopped for the complete course of treatment, or temporarily disabled before each procedure by setting the voltage to 0 and then resuming DBS stimulation immediately afterward.[45] Some proceduralists choose right unilateral ECT electrode placement to create the greatest distance from the hardware,[46] but bilateral is also effective and does not appear to produce any device-related side effects.[45]

A recently published review of the literature concerning ECT in the presence of intracranial metallic objects (including cerebral clipping systems, cerebral coils, DBS hardware, foreign bodies, and other metallic medical devices) found no complications, although there were modifications to the technique (ie, electrode placement) in several cases.[47] A study modeling the electric field produced by different ECT electrode placements in the presence of the DBS system demonstrated an increase in the electric field strength within the brain due to conduction through burr holes, especially when these were not fitted with nonconductive caps. In the case of typical subthalamic nucleus DBS lead placement, the required burr holes increased the electric field strength and focality the most during bifrontal ECT,

Table 2. Algorithm for CIED and Management During ECT

Verify the Presence or Absence of CIED	
What Type of Device Is It?	
Pacemaker?	**ICD?**
If the patient is not pacemaker dependent	Deactivate the ICD with a magnet or a programmer, have backup external.
Have a magnet available in case of device failure	Defibrillation available.
If the patient pacemaker dependent consider reprogramming to asynchronous mode with either a programmer or a magnet	If the patient is pacemaker dependent a magnet will NOT put the pacemaker into asynchronous mode, consultation with EPS is indicated.

For the purpose of this algorithm, we assume that any EMI produced by the ECT stimulus itself or the use of a peripheral nerve monitor is sufficiently removed (>15 cm) from the device so as to have a minimal chance of interfering with device function, and the most likely source of interference comes from movement due to fasciculations or a poor block. Consider using an increased dose of succinylcholine in patients with CIEDs.

Potential adverse outcomes we are trying to avoid include physical damage (including lead fracture or repositioning) resulting in device failure, changes in device function (inadvertent reprogramming), inappropriate defibrillator activation, and inadvertent reset to backup mode. Note that all ICD devices are also potentially pacemakers, in that they all retain the ability to overdrive pace tachyarrhythmias or pace asystole after a defibrillatory shock. Always have backup external pacing available in case of device failure.

Abbreviations: CIED, cardiac implantable electronic device; ECT, electroconvulsive therapy; EMI, electromagnetic interference; EPS, electrophysiology service; ICD, implantable cardioverter-defibrillator.

contrary to the historical belief that more anterior electrode placements are safer in patients with DBS implants.[48]

ECT is contraindicated in patients with cochlear implants according to the U.S. Food and Drug Administration and manufacturers.[49] However, one investigation gave 12 unilateral ECT to cadaveric human heads with cochlear implants and found no electrical injury to the device.[50] In the case of a 17-year-old male, 2 unilateral ECT on the side contralateral to the implant were well tolerated and without device malfunction.[51]

The RNS system (NeuroPace, Mountain View, CA) is a targeted responsive neurostimulation device approved for the treatment of medically refractory partial-onset epilepsy.[52] This implanted device consists of a closed-loop system that monitors cortical electrical activity in the brain and produces a brief electrical stimulus counteracting the electrocorticographic patterns heralding a seizure, as programmed by the physician. Although the manufacturer's device information recommends against ECT, currently there are no data supporting this contraindication; however, because this device potentially can terminate a seizure, thus potentially decreasing the effectiveness of ECT, consideration should be given to temporarily disabling the RNS system before ECT.

ANESTHETIC MANAGEMENT DURING ECT

ECT often is administered in off-site or remote locations, outside of the traditional operating room (OR) setting. This may be a dedicated suite for ECT, an area used for multiple different procedures, or even the postanesthesia care unit. General anesthesia is an integral part of modern ECT practice and requires the same standard American Society of Anesthesiologists monitors and medications that are available in the OR be used while providing anesthesia for ECT in any setting.[53] Heart rate, blood pressure, ECG, capnography, and temperature monitoring should be readily available. Although the vast majority of cases are managed without intubation, equipment to emergently secure the airway must be immediately available, along with a fully stocked "code" and "malignant hyperthermia" cart (Table 3).

Preanesthetic Medications

Many patients will require premedication in the period immediately before the treatment, primarily to prevent or lessen the headache or nausea that frequently occurs after ECT, decrease oral secretions that may complicate airway management, or reduce the degree of hemodynamic perturbations in the at-risk patient.

Headache is the most commonly reported complaint after ECT, occurring in roughly 50% of patients immediately or shortly after the patient regains consciousness following treatment.[54] The headache may be associated with nausea and, rarely, with photophobia, and it is usually bilateral and constant but may be unilateral or pulsatile. For most patients who do experience a headache, it will be mild, transient, and responsive to over-the-counter analgesics such as acetaminophen and ibuprofen. For patients who respond to these medications, prophylaxis may be given in the form of oral medication with a sip of water prior to treatment. For patients with a history of post-ECT headache unresponsive to these first-line agents, prophylaxis with 30 mg IV ketorolac often is effective.[55]

Unless the patient presents with a history of post-ECT headache or is felt to be at high risk for the development of one, prophylactic analgesics are generally not given before the first treatment. Should the patient complain of headache in the posttreatment period, oral ibuprofen or acetaminophen or IV ketorolac is administered at that time and prophylactically at subsequent ECT sessions. Patients with a history of migraine or other more severe headache may require antimigraine medications.[56]

Although less common than headache, nausea after ECT may occur in up to 25% of patients. The cause may be related to the anesthetic or to the ECT treatment, or in cases of difficult ventilation requiring high positive pressures, to air in the stomach. Nausea after treatment may occur with or without headache and can prolong recovery times. Primary treatment and prophylaxis is with the serotonin 5-HT3 receptor antagonist ondansetron (4–8 mg IV).[57] Most patients at increased risk for post-ECT nausea are given ondansetron IV before treatment rather than older medications with increased risk for untoward side effects (typically sedation). The older dopamine-blocking agents (prochlorperazine, haloperidol, droperidol, trimethabenzamide, and metoclopramide) should be reserved for refractory cases. If nausea cannot be prevented by routine prophylaxis, consideration should be given to changing the anesthetic agent. In refractory cases, propofol, which has antiemetic properties, may be the anesthetic agent of choice.

Table 3. Quick Reference Guide to Drugs Commonly Used for ECT

Drug	Class	Primary Use	Dose (IV)	Onset	Duration	Potential Issues
Glycopyrrolate	Anticholinergic	Vagolytic	0.2 mg	60–90 s	2–3 h	Urinary retention
		antisialagogue	0.2 mg	20 min	6–7 h	
Ketorolac	NSAID	Headache prophylaxis	15–30 mg	5–30 min	6–8 h	GI Bleed
Ondansetron	5-HT3 receptor antagonist	Antiemetic	4–8 mg	5–30 min	6–8 h	
Methohexital	Hypnotic	Induction	0.8–1.2 mg/kg	15–30 s	6–8 min	Hypotension
Propofol	Hypnotic	Induction	1–1.5 mg/kg	15–30 s	6–8 min	Hypotension
Ketamine	Hypnotic	Induction	1–2 mg/kg IV	30–90 s	6–8 min	Dysphoria
			5–10 mg/kg IM	1–2 min		
Etomidate	Hypnotic	Induction	0.2–0.5 mg/kg	30–45 s	6–8 min	Adrenal suppression
Remifentanil	Opioid	Induction	3.5–4.5 mcg/kg	15–30 s	6–8 min	Prolonged apnea
Succinylcholine	Paralytic	Paralysis	0.9–1.2 mg/kg	60–90 s	5–8 min	Prolonged apnea
Labetalol	β-blocker	Antihypertensive	5–10 mg	2–5 min	2–6 h	Hypotension
Esmolol	β-blocker	Antihypertensive	20–50 mg	15–30 s	2–3 min	Hypotension
Metoprolol	β-blocker	Antihypertensive	1–10 mg	2–5 min	4–8 h	Bradycardia
Hydralazine	Vasodilator	Antihypertensive	2.5–5 mg	15–20 min	2–4 h	Hypotension
Nitroglycerine	Vasodilator	Antihypertensive	40–80 mcg	1–2 min	3–5 min	Hypotension
Midazolam	Benzodiazepine	Sedation	0.5–2 mg IV	15–30 s	30–45 min	
			5–10 mg IM	1–2 min		
Atropine	Anticholinergic	Vagolytic	0.4–0.8 mg	2–15 min	2–3 h	Tachycardia

Abbreviations: ECT, electroconvulsive therapy; IV, intravenous; NSAID, nonsteroidal anti-inflammatory drug.

For patients at risk for bradycardia during treatment, glycopyrrolate 0.2 mg may be administered IV immediately before ECT.[29] For patients with a history of copious oral secretions, glycopyrrolate should be administered IV at least 20 minutes before the treatment, to allow secretions already present in the oropharynx to be swallowed.

Choice of Induction Agent

Current practice is to induce general anesthesia before ECT using an IV hypnotic agent such as methohexital, propofol, etomidate, or ketamine. Because all general anesthetics have the potential to interfere with the generation of the therapeutic seizure, the choice of anesthetic must be tailored to the individual needs of each patient. Methohexital historically has been considered as the gold standard, because it provides nearly ideal kinetics and has only moderate anticonvulsant properties. Propofol remains an acceptable alternative and may actually be preferred for some patients. It is potently anticonvulsant, which presents a problem for patients with a high seizure threshold or history of very short seizures, but may actually be the induction agent of choice in patients at risk for prolonged seizures.[58] Etomidate is a reasonable alternative for patients at risk for a pronounced hypotensive response to either propofol or methohexital, but the theoretical potential for repeated doses to cause adrenal suppression make it a less attractive agent for repeated brief procedures. Ketamine has a long history of use as an alternative anesthetic induction agent in ECT, especially when seizure enhancement and rapid response to therapy are desired.

Methohexital. The ultrashort-acting barbiturate methohexital is currently the induction agent of choice for ECT in the United States.[2] Methohexital, although not perfect, is a nearly ideal agent for many reasons. Its pharmacodynamics ensure a rapid onset of general anesthesia followed by rapid recovery, allowing for early discharge from the ambulatory suite. It is currently a generic medication and reasonably inexpensive. In addition, methohexital possesses only modest anticonvulsant properties. Doses of approximately 1 mg/kg of ideal body weight typically produce an appropriate level of anesthesia in the time it takes the drug to circulate from the injection site to the active site in the brain, usually 20 to 30 seconds.[59]

Propofol. Propofol has similar pharmacokinetics to methohexital. It works rapidly, inducing a state of general anesthesia within 20 to 30 seconds after the injection of a bolus dose, and quickly redistributes from the active site in the central nervous system into lipid depots, allowing for a return to consciousness within 8 to 10 minutes. The patient who has received a propofol-based anesthetic typically recovers feeling well and rested.[60] Unfortunately, propofol is potently anticonvulsant and may raise the seizure threshold or shorten the therapeutic seizure.[61,62] Despite this, propofol remains a first-line induction agent at many ECT services,[63] although some services may reserve propofol for patients with lower seizure thresholds and a history of prolonged seizures, those with refractory posttreatment nausea, or a history of severe postictal agitation.

Ketamine. Ketamine is a "dissociative" anesthetic, a noncompetitive *N*-methyl-D-aspartate (NMDA) receptor antagonist developed in 1964 as a derivative of phencyclidine.[64] It currently is used as a drug to induce general anesthesia, as an adjunct to inhaled or infused general anesthetics to provide analgesia for surgery, and as a stand-alone drug to provide analgesia for painful procedures. When administered as an IV bolus dose of 0.9 to 1.1 mg/kg, peak plasma levels are reached and ketamine reliably induces a state of general anesthesia, typified by unresponsiveness to voice commands or to painful stimulus within 60 to 90 seconds.[65] For some patients, a depth of anesthesia appropriate for ECT may not be reached for up to 120 seconds, requiring patience on the part of the treatment team. Nystagmus, a key physical sign caused by ketamine, may help establish depth of anesthesia.

Ketamine produces both anesthetic and dissociative effects, and the predominant effect depends on the concentration of the drug at receptor sites. At lower doses, the drug produces feelings of relaxation. At greater

doses, ketamine produces a state of general anesthesia. In addition to its NMDA effects, ketamine causes a moderate sympathomimetic action via increased catecholamine outflow and decreased reuptake, resulting in mild tachycardia and hypertension. This may complicate hemodynamic management in the case of patients at risk for cardiac ischemia, or reduce the risk for bradycardia or asystole in the case of patients with a low resting heart rate. Ketamine also may result in increased salivation and consideration should be given to pretreatment with glycopyrrolate.

Investigators have studied ketamine as a way to enhance seizures or the antidepressant effect of ECT and to reduce cognitive effects. Although some studies fail to demonstrate seizure enhancement with ketamine anesthesia,[66,67] some have suggested it is a reasonable choice for patients with an elevated seizure threshold.[68] When administered at a slightly greater dose (1.3 mg/kg), ketamine has been shown to increase seizure duration and midictal electroencephalogram amplitude compared with methohexital,[69,70] as well as overall seizure quality.[71]

Ketamine has been shown to enhance synaptic plasticity in rat models, to reduce post-ECT reorientation times, and to improve word recall after ECT, although others have found no difference in performance on posttreatment tests of cognition.[72] Several studies have shown that ketamine possesses intrinsic antidepressant properties, and, in fact, is beginning to be used to treat patients with major depressive disorder or bipolar depression refractory to other medications.[73–75] More recently, investigations have focused on the potential for capitalizing on ketamine's intrinsic antidepressant properties when used as the induction agent for patients receiving ECT.[76,77]

Ketofol. For patients who cannot tolerate the clinical effects of ketamine, the ketamine and propofol combination drug commonly referred to as ketofol may be more appropriate. By administering propofol and ketamine simultaneously, less of each agent is required to induce a state of general anesthesia appropriate for ECT. The use of these 2 medications simultaneously takes advantage of the different therapeutic and adverse effect profile of each: ketamine's intrinsic antidepressant and proconvulsant effect, and propofol's smoother induction and emergence properties, as well as its ability to counteract the hypertension caused by ketamine. A lower dose of propofol reduces the potential anticonvulsant impact on the treatment, whereas the addition of ketamine may be associated with greater antidepressant effect.[78,79]

Remifentanil. Remifentanil is an anilidopiperidine analog of fentanyl developed for use as an analgesic agent for procedures associated with intense pain but little need for postoperative analgesia. It is a potent μ-opioid receptor agonist that produces intense analgesia but is rapidly degraded via ester hydrolysis. Because of its unique structure, it possesses pharmacokinetics ideal for use in ECT. It typically is used for patients in whom it is difficult to induce an acceptable seizure, because of its low anticonvulsant properties.[80] Remifentanil may be administered as a bolus (3–4 μg/kg IV) to allow for the administration of a reduced dose of methohexital or propofol (0.2–0.3 mg/kg) or as a stand-alone anesthetic. Remifentanil is not a hypnotic and its use as a stand-alone anesthetic may theoretically increase the risk for awareness.

Etomidate. Etomidate is a short-acting IV general anesthetic with similar pharmacokinetics to methohexital and propofol. It frequently is used as the sole anesthetic for ECT in some institutions because of its minimal effect on seizure threshold and safer cardiovascular risk profile, and it is the agent of choice in the hemodynamically unstable patient.[81] Etomidate does suppress corticosteroid synthesis by reversibly inhibiting 11-β-hydroxylase leading to primary adrenal suppression,[82] theoretically making it a less desirable choice for patients who are to receive multiple treatments over an acute course of ECT. However, this adverse effect has never been demonstrated with ECT. Etomidate has been associated with an increased risk for nausea after administration, so its use should be avoided in patients with a history of post-ECT nausea.

Inhalational Agents. Although anesthesia for ECT is almost always induced using IV agents, there are some circumstances which warrant consideration of an inhalational induction. Patients who cannot tolerate the insertion of an IV cannula because of severe needle phobia, patients who cannot tolerate an IV induction because of the pain associated with injection or other unpleasant side effects, or those who are psychotic or agitated and unable to cooperate with catheter insertion may benefit from an inhalational induction.[83] Sevoflurane is the agent of choice in this setting because of its tolerability.[84] The use of an inhalational agent for induction of anesthesia is common in the OR setting and typically used for pediatric patients, but rarely if ever used in the ECT suite. Thus, the decision to use this technique will require either a move to a different setting which may be unfamiliar to the psychiatric team, or the movement of an anesthesia delivery unit into the ECT suite. Most authors suggest discontinuing the sevoflurane and switching to an IV agent once IV access has been achieved because sevoflurane anesthesia for ECT has been associated with shorter motor seizures and higher postictal blood pressures.[85,86]

Depth of Anesthesia Appropriate for ECT

Once the induction agent of choice has been administered, it is imperative to achieve a depth of anesthesia appropriate for ECT before the administration of the paralytic agent. The patient must be rendered unconscious before the administration of succinylcholine so as to avoid experiencing the sensation of being awake and paralyzed, and to prevent awareness during the actual stimulus. To confirm that an appropriate level of anesthesia has been achieved, it is appropriate to wait until there is a loss of response to verbal commands. Many anesthesiologists may rely on the loss of the "eyelash reflex" as an indication that the proper depth has been achieved. However, this test is one of a series of physical signs that was used to indicate the plane of ether anesthesia, and does not necessarily apply to today's IV anesthetics. This reflex may not be inhibited after induction with methohexital, even when the patient is under an

appropriate level of anesthesia.[87] Relying solely on the loss of the eyelash reflex may result in administration of unnecessarily high doses of methohexital, with potential reduction of efficacy of ECT.[88]

Muscle Relaxation

The goal of providing neuromuscular blockade during ECT is to reduce the potential for injury during treatment while simultaneously allowing for the prompt recovery of spontaneous respirations. A degree of muscle relaxation sufficient to prevent injury can be achieved by using a succinylcholine dose of approximately 0.90 to 1.10 mg/kg in most patients.[89] Greater doses of succinylcholine can prolong the period of apnea, and lower doses can increase the risk for injury during the initial onset of the stimulus. As there is a wide range of variability in patient response to succinylcholine, current practice is to administer a modal dose and adjust at subsequent treatments based on clinical response. In patients who are at increased risk for complications resulting from inadequate muscle relaxation, the dose of succinylcholine can be increased by 40% to 50% to ensure complete relaxation.[90]

Common practice is to inflate a standard blood-pressure cuff or tourniquet wrapped around the right ankle to 20% above systolic blood-pressure before administering the paralytic agent. This prevents paralysis of the foot and allows for electromyography monitoring, providing evidence that the stimulation has resulted in a generalized grand-mal seizure and that the motor component of the seizure has terminated. It is essential to allow enough time between succinylcholine administration and ECT stimulation to ensure maximal neuromuscular blockade is achieved. There is considerable variability in time to relaxation between patients, so it is not simply enough to allow a period of time to pass (typically 1 minute in younger patients and 2 minutes in older patients) before initiating the stimulus. A peripheral nerve stimulator should be used to confirm full neuromuscular blockade, and stimulation should only occur once muscle fasciculations in the distal extremities have subsided, the limbs are completely relaxed, and the plantar reflexes have been abolished.[91]

For patients in whom succinylcholine is contraindicated (eg, muscular dystrophy, prolonged immobilization, paralysis, burns, malignant hyperthermia, etc) nondepolarizing muscle relaxants should be used. In situations where the drug sugammadex is available, this does not present a problem, as rocuronium may be administered at the standard dose of 0.6 mg/kg and reversed with 2 mg/kg of sugammadex once the seizure has terminated, provided that a train of four count of at least one has been achieved.[92] Current recommendations are to allow 24 hours between sugammadex administration and readministration of rocuronium to ensure that the sugammadex has been eliminated.[93] Although associated with only mild adverse effects such as cough, altered taste sensation and short term prolongation of both the QT interval and activated partial thromboplastin time, sugammadex can potentially decrease the efficacy of hormonal-based contraceptives and this issue should be discussed with patients at risk for pregnancy who are using hormonal-based oral contraceptives.[94] If sugammadex is not available, a reduced dose of an intermediate-acting muscle relaxant such as rocuronium (0.4 mg/kg) may be administered, although this procedure requires close monitoring of neuromuscular function via nerve stimulator, reversal of neuromuscular blockade, and additional anesthesia via bolus or infusion to prevent awareness.[95]

Ventilation Management

Unlike many procedures requiring anesthesia, airway management during ECT has the potential to significantly alter the treatment course of any given patient. Proper management can decrease the necessity for increasing the electrical stimulus charge in the case of inadequate seizure length,[96] reduce the need for restimulation in cases of abortive seizures,[97] and reduce the potential for postictal delirium.

Hyperventilation to Produce Hypocapnia. Because most patients are managed with a bag-valve-mask (BVM) device that does not allow for accurate carbon dioxide (CO_2) measurement, the goal for effective ventilatory management during ECT is to lower CO_2 levels to a point that will enhance seizure activity, without the necessity of setting a numerical goal for end-tidal CO_2. Hypocapnia achieved through hyperventilation is used as an activation method in standard electroencephalography for the provocation of epileptic activity[98] and has long been used to enhance seizure quality. This technique is most important in patients with a greater seizure threshold or a history of inadequate seizure length.[99] The seizure enhancement is not thought to be due to changes related to varying oxygen (O_2) tension.[97] Recent investigations have focused on controlled hyperventilation and its effect on multiple parameters associated with seizure efficacy, and involved quantitative measurements of $PaCO_2$ during treatment.[100] In these studies, the CO_2 level was reduced to a partial pressure below 30 mm of mercury (mm Hg). Although many studies support the use of this technique, the level of contribution to seizure parameter improvement has been questioned by some investigators.[101] It should be noted, however, that this technique has the potential for generating prolonged seizures and should be avoided in younger patients with a history of experiencing prolonged seizure activity during ECT.[102]

Hyperoxia. Current practice includes preoxygenation before the induction of general anesthesia via either nasal cannula oxygen or facemask. These devices must be removed before providing positive pressure ventilation via BVM device. The practice of preoxygenation improves the safety of the procedure by increasing the oxygen reserves of the patient and thus increasing the period of tolerable apnea. Passive preoxygenation is followed by active hyperventilation with 100% oxygen via BVM device at the rate of 40 to 50 breaths per minute, further increasing blood oxygen partial pressures. Benefits of hyperoxia include both an improvement in seizure quality and a reduction of the required charge of electricity applied during the stimulus.[103]

Aspects of ECT With Which the Anesthesiologist Should Be Familiar

It is helpful for the anesthesiologist to be knowledgeable about several aspects of the ECT procedure itself, to best

collaborate with the psychiatric proceduralist. Refinements in ECT practice are the result of ongoing research, both clinical and basic, much of which is summarized in guidelines published by major psychiatric associations around the world.[104]

Electrode Placement. ECT involves the induction of a generalized cerebral seizure via 2 electrodes placed on the patient's scalp. Since the inception of ECT, practitioners have experimented with various electrode placements, but only 3 have become standard in modern practice: bilateral (also referred to as bitemporal), right unilateral, and bifrontal.[105] Rarely, for a left-handed patient with right hemisphere language dominance, left unilateral electrode placement can be used.[106,107] Electrode placement affects both efficacy and cognitive effects of the treatment; bilateral and bifrontal result in slightly better efficacy and speed of response, while right unilateral is associated with milder cognitive effects. If a patient does not respond to right unilateral ECT after several treatments, they may be switched to bilateral.[108] Conversely, if a patient develops unacceptable cognitive effects with bilateral, they may be switched to right unilateral. The electrodes themselves may be either disposable adhesive pads or metal disks; in either case, attention must be paid to ensuring appropriately low impedance at the scalp-electrode interface.

The Electrical Stimulus. The type of electrical stimulus used to induce seizures during ECT has evolved from the now-outmoded sine wave to modern brief and ultrabrief pulse square waves. These modern waveforms are more efficient at inducing seizures and are associated with fewer cognitive effects. The overall stimulus "package" delivered is described by 4 parameters: amplitude, pulse width, frequency, and duration. These can either be individually adjusted or controlled by preset algorithms on the ECT device. Seizure threshold refers to the lowest electrical stimulus charge needed to induce a seizure; this is often determined by a dose titration procedure at the first treatment session, in which incremental stimuli are given until a seizure is elicited. Subsequent treatments are delivered at a multiple of seizure thresholds, typically 1.5 to 2.5× seizure threshold for bilateral, 6× seizure threshold for right unilateral, with the goal of insuring maximum efficacy without excessive cognitive effects.[109]

With dose titration, there is a possibility that the patient will not seize. Direct stimulus of the vagus nerve in the setting of a subconvulsive stimulus can produce a significant parasympathetic response causing asystole. If a seizure is not elicited, the psychiatrist will request vigorous hyperventilation for 20 to 40 seconds and then administer a second or third stimulus at an increased charge. It is important to ensure that adequate levels of neuromuscular blockade and anesthesia remain during these subsequent stimulations.

Seizure Adequacy. A maximally therapeutic seizure is believed to be one of adequate duration and robust electroencephalogram expression. In practice, seizures of <15 seconds motor duration may be considered inadequate, with possible restimulation of the patient.[29] Clinical response should take precedence over an arbitrary cut point for seizure duration, and patients may have good outcomes despite short seizures. Attempts should be made to find the cause of a short seizure (eg, excessive anesthetic dose, concomitant anticonvulsant medication, inadequate hyperventilation) and to eliminate the cause, if possible.

Management of Sympathetic and Parasympathetic Response to Stimulus

The expected physiologic response to ECT includes an initial increase in parasympathetic tone due to direct stimulation of the vagus nerve, followed by an increase in sympathetic tone coincident with the generation of the therapeutic seizure. Hypertension and tachycardia, as well as hypotension associated with bradycardia, are well documented effects of treatment with ECT.[110] Although these hemodynamic changes generally are time-limited and well tolerated in the majority of patients, the anesthesiologist should be prepared to intervene in the case of a persistent or undesired response to stimulus.

Parasympathetic Response. A cardiac pause is frequently observed during stimulus application but presents little risk, since cardiac activity almost always resumes.[111] Other factors associated with poststimulus asystole include preexisting unstable cardiac disease, increased age (older than 65 years), the use of β-blockers (either chronically or in the pretreatment period) or other sympatholytic medication, hypoxia, and exposure to an acetylcholinesterase inhibitor.[111,112] A stimulus that does not provoke a seizure, either during dose-titration or in patients with an intrinsically high threshold, may result in a prolonged period of asystole, so atropine should be immediately available. More often, a subconvulsive stimulus results in time-limited bradycardia of no significance. Asystole in the postictal period is a much rarer event but can occur in some patients.[113,114] Patients at risk for postictal asystole are typically younger, well-conditioned (good physical and cardiac health) men with a low resting heart rate.[115] Recommendations for subsequent management of patients who have experienced an episode of asystole during ECT are included in Table 4.

Sympathetic Response. After the successful generation of a therapeutic seizure, it is expected that the patient will experience the physiologic effects of significantly increased sympathetic tone. Hypertension and tachycardia are, for the most part, transient and well-tolerated effects of the seizure

Table 4. Recommended Management Strategies for Subsequent Treatments in the Patient Who Has Experienced a Period of Asystole During ECT[a]

- Avoidance of all beta blocking agents in the peritreatment period
- Administration of hydralazine for antihypertensive prophylaxis
- Pretreatment with glycopyrrolate for bradycardia
- Pretreatment placement of external pacing pads
- External pacing at 40 beats per minute if necessary

Abbreviation: ECT, electroconvulsive therapy.

[a]Bryson EO, Liebman L, Ahle G, Kellner CH. Asystole during electroconvulsive therapy. *J ECT*. 2014;30:259–260.[114]

activity. In one study, hemodynamic data were collected during 450 sequential ECT treatments, demonstrating an average increase in systolic blood pressure of approximately 45 mm Hg and an average increase in heart rate of approximately 50 beats per minute.[116] The majority of patients in this study (72%) had no cardiac history and in these patients β-blocking agents were not administered before the treatment. Those patients with a positive cardiac history (hypertension, documented coronary artery disease, or valvular disease) received either labetalol or esmolol before the treatment, with a small number requiring additional labetalol for persistent hypertension or esmolol for persistent tachycardia. Despite this, none of the patients in this series experienced any adverse events due to hypertension, tachycardia, or as a sequela of treatment with β-blocking agents.

The decision to treat hypertension related to ECT should be made by the anesthesiologist on a case-by-case basis. The need for prophylactic administration of antihypertensive agents before ECT depends on the perceived risk for a periprocedural cardiac event and is intimately related to the medical comorbidities of the individual patient. The anesthesiologist should weigh the risks of treatment (primarily prolonged postanesthesia care unit stays due to postural hypotension) with the risks of an untoward event (eg, myocardial ischemia, increased intracranial pressure, ruptured aneurysm) when deciding whether or not to administer prophylactic treatment. The agent most commonly used in this setting is labetalol, though esmolol or nitroglycerin are acceptable, short-acting alternatives. Persistent posttreatment hypertension may require treatment with long-acting agents. Tachycardia frequently accompanies the hypertension seen during ECT, and this is also well tolerated by the majority of patients.

Some otherwise-healthy patients may experience changes in cardiac function related to an increased heart rate during ECT.[117] Rate-related left bundle branch block (LBBB) occurs when there is a delay of normal conduction from the intraventricular septum to the left ventricle associated with an increase in heart rate above a specific threshold.[118–120] In many patients, ischemic cardiomyopathy may exist but be asymptomatic, which is why new-onset LBBB must be thoroughly and urgently evaluated even in the absence of symptoms.[121,122] Anesthesiologists caring for patients receiving ECT should be aware that rate-related LBBB can occur during ECT, and that LBBB is not always a benign condition. If new onset LBBB occurs during ECT, immediately assess the patient for signs and symptoms of acute coronary syndrome and refer to cardiology for evaluation.

Posttreatment Management

Postictal Agitation. Although ECT is well tolerated by most individuals, approximately 10% of patients develop transient emergence agitation, typically characterized by restlessness, confusion, and delirium.[123] If mild, post-ECT agitation may be treated supportively without pharmacologic intervention. Simple behavioral interventions such as gravitational restraint, distraction with an inflated glove, or verbal reorientation should be attempted before administering medications. Patients with more severe agitation are at increased risk for injuring themselves, as they frequently pull out their IV lines and remove monitoring devices like ECG and pulse oximeter leads. Once it is clear that this behavior is not related to some other etiology, such as hypoxia, the administration of a benzodiazepine (eg, midazolam 1–2 mg IV), antipsychotic medications (eg, droperidol 1 mg IV), or a bolus dose of propofol (eg, 30 mg IV) will treat most cases. For subsequent treatments, changing the anesthetic to propofol is often all that is necessary to avoid agitation. In refractory cases, an infusion of dexmedetomidine has been reported to be helpful.[123] Dexmedetomidine, an α-2 adrenergic agonist with properties similar to clonidine, was first used in the context of ECT in an attempt to blunt the acute hyperdynamic response to ECT,[124] but was also found to attenuate posttreatment agitation when given as premedication[125] or immediately following treatment.[126]

Pulmonary Edema. Although very rare, pulmonary edema after ECT has been reported. Etiologies include flash pulmonary edema secondary to a hypertensive crisis, negative pressure pulmonary edema, cardiogenic pulmonary edema, and asthma treated with clenbuterol hydrochloride.[127,128] Despite the fact that clinically evident pulmonary edema is rare, the anesthesiologist providing care for patients undergoing ECT should be prepared to manage this potentially serious complication. The 2 etiologies for pulmonary edema that have been reported most frequently are cardiogenic and negative pressure pulmonary edema. Cardiogenic pulmonary edema is usually related to a profound sympathetic surge leading to hypertension and tachycardia[129]; negative pressure pulmonary edema typically occurs when the patient has residual incomplete neuromuscular blockade and attempts to inspire against an obstructed glottis.[130]

Excess catecholamines are associated with increased vascular permeability and likely contribute to the development of pulmonary edema in this setting. The increased heart rate and blood pressure that result from this sympathetic activity significantly increase myocardial oxygen demand; in some cases, changes in the ECG suggestive of ischemia have been reported during this period.[131] In patients with subclinical coronary disease, this may lead to acute heart failure and flash pulmonary edema.[132] Attenuating the hemodynamic response to treatment by frequent dosing with short-acting antihypertensive agents (such as esmolol or nitroglycerin) can be achieved in patients thought to be at risk.

Febrile Reactions and Neurologic Dysfunction. Extremely rare complications include febrile reactions unrelated to malignant hyperthermia that have been described in 2 patients,[133,134] and neurologic dysfunction including focal deficits.[135–137] Focal neurologic dysfunction immediately after ECT is rare but may result from cerebrovascular ischemia, ruptured aneurysm,[138,139] or in the case of transient dysfunction, be electrophysiologic in nature. Hemiparesis resulting from a postictal neurologic deficit localized to the area of seizure focus, first described in detail by Todd in 1849, is thought to result from transient hyperpolarization of cells, cellular toxicity mediated by NMDA receptors, and localized changes in cerebral blood flow and metabolism.[140]

Takotsubo Cardiomyopathy. Takotsubo cardiomyopathy is a reversible stress-induced cardiomyopathy characterized by left ventricular hypokinesis and apical ballooning triggered by severe physical or emotional stress, or medical procedures including ECT.[141] A 2011 case review describes 7 cases of this syndrome following ECT,[142] and an additional 5 have been reported since. Retreatment with ECT after Takotsubo cardiomyopathy has been reported, and was managed with β-blockers.[143]

CONCLUSIONS

Proper anesthetic management is directly linked to the remarkable safety and efficacy of ECT. A preprocedural patient evaluation will alert the treatment team to any medical conditions that will require special management before, during, or after the procedure. Most patents with cardiac pacemakers or other implanted devices can be safely treated. Expert management of ventilation can increase treatment efficacy and improve patient outcomes. Management of hypertension and tachycardia with IV β-blockers or other agents may be recommended in patients at risk for cardiac ischemia, whereas most healthy patients will not require any intervention for transient increases in blood pressure and heart rate. Postictal agitation may be effectively managed with additional sedation and prevented at subsequent treatments by preemptive use of such medications. An individualized patient-specific approach to anesthetic management, with personalized adjustments based on clinical response will lead to optimal treatment outcomes, patient comfort, and safety. ■

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