Medical consultation for electroconvulsive therapy

INTRODUCTION — Electroconvulsive therapy (ECT) is a commonly performed procedure in the United States. Use of ECT is rising, and psychiatrists often request medical evaluation before ECT since many eligible patients are elderly with multiple medical comorbidities. This topic review will discuss the use, indications, anesthetic technique, procedure, and morbidity of ECT, as well as risk assessment and strategies to reduce the risk of the procedure.

INDICATIONS FOR ECT — The primary indication for ECT is for the treatment of major depression that is refractory to antidepressant medications [1]. Indications listed in the American Psychiatric Association guideline for the treatment of patients with major depressive disorder include psychotic depression, catatonic stupor, severe suicidality, food refusal leading to nutritional compromise, and pregnancy and other situations where a rapid antidepressant response is required (table 1) [2]. The report also recommends ECT for patients who have previously shown a positive response to it and for those who have medical conditions that prevent the use of antidepressant medications. The Canadian Psychiatric Association clinical guidelines for the treatment of depressive disorders suggest similar indications [3]. (See "Treatment of resistant depression in adults" and "Indications for electroconvulsive therapy (ECT) in unipolar depression and its efficacy".)

ECT is also used for the treatment of schizophrenia. In a systematic review, evidence supported some benefit in general functioning when compared with placebo, but this effect was less strong than for antipsychotic medication and was useful only for short-term relief of symptoms [4]. Limited evidence demonstrated a superior response to ECT plus antipsychotic medications when compared with antipsychotic medications alone.

Other psychiatric conditions for which ECT is effective include bipolar disorder, mania, and atypical psychosis [5]. ECT also may be effective or have application in patients with organic delusional disorder, organic mood disorder, obsessive compulsive disorder, catatonia secondary to medical conditions, neuroleptic malignant syndrome, neuroleptic-induced Parkinsonism, and neuroleptic-induced tardive dyskinesia [5,6].

TECHNIQUE AND ANESTHESIA — ECT is usually administered two or three times per week for a total of 6 to 12 treatments. The treatment typically causes a 30 to 60 second generalized tonic clonic seizure, an effect that is essential to the success of ECT. The patient is preoxygenated with supplemental oxygen (2 L/min) via nasal cannula while the procedure is being set up. Prophylactic beta blockers may be administered immediately before or during ECT to blunt the hypertensive, tachycardic response to the seizure. The anesthesia of choice is methohexital. Other induction agents include propofol, thiopental, etomidate, and ketamine. Skeletal muscle relaxation is used during ECT to minimize the motor seizure and prevent musculoskeletal injury. The standard agent is succinylcholine via intravenous infusion.
(See "Technique for performing electroconvulsive therapy (ECT) in adults").

**MORBIDITY AND MORTALITY** — Clinicians should be aware of certain potential side effects or complications of ECT. (See "Overview of electroconvulsive therapy (ECT) for adults", section on 'Adverse effects'.)

The American Psychiatric Association lists the following conditions as associated with increased risk [7]:

- Unstable or severe cardiovascular disease
- Space-occupying intracranial lesion with evidence of elevated intracranial pressure
- Recent cerebral hemorrhage or stroke
- Bleeding or otherwise unstable vascular aneurysm
- Severe pulmonary condition
- ASA (American Society of Anesthesiologists) Class 4 or 5 (table 2)

**Mortality** — ECT is one of the safest procedures performed under general anesthesia. With modern anesthetic technique, the rate is sufficiently low and the potential life saving benefit is compelling enough that absolute contraindications to treatment no longer exist. (See "Overview of electroconvulsive therapy (ECT) for adults", section on 'Adverse effects'.)

**Cardiovascular effects** — A 15- to 20-second parasympathetic discharge occurs during the procedure as the patient enters the tonic phase of seizure. This can lead to arrhythmias including bradycardia with or without hypotension, atrial arrhythmias, premature atrial and ventricular contractions, atrioventricular block, and asystole. Asystole can occur with the first treatment or at any time later in a patient's course [8]; patients are at higher risk with longer periods of subconvulsive seizures [9]. In one study of older adult patients, 66 percent had asystole lasting greater than 5 seconds with no lasting complications [10]. A history of hypertension or evidence of ischemia on electrocardiogram (ECG) did not predict asystole, nor did current use of calcium channel blockers, nitroglycerin, angiotensin converting enzyme (ACE) inhibitors, diuretics, or psychiatric medications (beta blockers were not included). Interestingly, patients with heart block and/or rhythm abnormalities were less likely to develop asystole (54 versus 16 percent).

The clonic phase of the seizure then leads to a catecholamine surge that causes tachycardia and hypertension. The duration of tachycardia usually correlates with the length of the seizure itself [11]. These hemodynamic responses continue into the postictal period and usually resolve within 10 to 20 minutes of the seizure [12]. Occasionally patients have persistent hypertension that requires treatment. (See 'Postprocedure hemodynamic changes' below.)

All patients are followed with ECG during the procedure; even healthy patients can have transient ECG changes. However, these changes are rarely significant, as illustrated by a study of 29 patients comprising a total of 80 treatments, in which no one had persistent T wave inversion, pathologic Q waves, or a demonstrable rise in cardiac enzymes at four and six hours (although some had a mild increase in creatine kinase [CPK], presumably from skeletal muscle) [13].
ECT can also cause a transient depression in the ejection fraction of healthy patients [14]. A study in 53 adults undergoing ECT found that seven developed new global left ventricular (LV) systolic dysfunction and eight developed regional wall motion abnormalities [15]. Among the 14 patients who developed global or regional abnormalities after the first ECT treatment, 13 had resolution of these abnormalities after the fourth ECT treatment (generally about one week later), and there were no short-term adverse events in any of the patients with LV dysfunction.

Several studies have investigated the incidence of serious cardiac complications from ECT:

- In a retrospective study of 42 patients who had undergone ECT, 12 of the 17 patients with underlying cardiac disease had cardiac complications [16]. Most of these were benign and self-limited, including atrial and ventricular ectopy and nonsustained ventricular tachycardia. There was evidence of ischemia in two patients and one cardiac arrest of unknown etiology. Seventy percent of all complications, self-limited or not, occurred in patients with cardiovascular disease identified on the history, physical examination, or ECG, and all occurred in patients greater than 50 years of age. This study did not, however, systematically document the degree of preexisting cardiac disease.

- A prospective study of 40 patients with cardiac disease found that 55 percent had at least one complication with ECT [17]. These included minor complications, such as transient arrhythmias or ST segment changes, and major complications, such as persistent ECG changes accompanied by chest pain, asystole, or persistent arrhythmias. Only 7.5 percent in the control group without cardiac disease had a cardiac complication, and all were transient and minor.

- A case-control study of 80 patients over the age of 50 found that the risk of major complications was 11.5 percent in patients at high risk for cardiovascular disease [18]. The patients were able to complete their course of therapy once the complications were treated.

These findings suggest that the incidence of important cardiac complications are relatively rare and almost always occur in older patients and those with underlying cardiovascular disease. Furthermore, even patients at high risk for cardiac complications tolerate ECT well and, if complications occur and are treated, the vast majority can complete the treatment course. As an example, one study treated 80 patients, one-half of whom had preexisting cardiac disease (depressed ejection fraction, conduction disease, or frequent premature ventricular contractions) [17]. Among those with cardiac disease, eight had major complications including chest pain, arrhythmia, ischemia, and one myocardial infarction. Anesthesiologists and cardiologists treated the complications as they occurred and 36 of 38 patients went on to complete treatment, including the patient with the myocardial infarction. Similarly, a second study treated 53 patients and, of the 27 patients at risk, 31 percent required changes in medication or pretreatment with subsequent treatments because of complications [18]. Twenty-five out of 27 patients completed the course of ECT; there were no deaths. Despite these successful outcomes, it is not known how many of the patients in these studies were on beta blockers or other antihypertensive medication prior to treatment.

Central nervous system and other effects — Several cerebral effects occur with ECT, including increases in cerebral blood flow and intracranial pressure. Memory loss, disorientation, and
Role of the Medical Consultant — The preprocedure evaluation of patients before ECT is similar to the approach to a patient undergoing any procedure that requires general anesthesia: to identify medical issues that place the patient at risk, to propose strategies to reduce risk, and to treat complications after the procedure.

Preprocedure Evaluation — A complete history and physical examination will help to identify pertinent risk factors. We agree with the recommendation from the American Psychiatric Association that no specific laboratory tests are required in the pre-ECT evaluation. We recommend measurement of serum electrolytes only for patients taking diuretics or other medications that increase the likelihood of an abnormality and for patients with established renal disease or congestive heart failure. We suggest an electrocardiogram in patients over the age of 50 years, since most cardiac complications occur in older patients. There are no indications for other routine laboratory studies.

The history should include a review of previous difficulties with anesthesia or ECT. As mentioned above, there are no absolute contraindications to ECT and most patients can have the procedure without serious complication, but clinicians should seek risk factors that may require intervention or management. Important considerations include risk factors for cardiac ischemia or arrhythmia, heart failure, and the presence of brain tumors or other neurosurgical issues. A history of skull fractures should be determined, as this may affect electrode placement.

The history should also include a medication history, to specifically inquire about use of herbal medications. In one study, 54 percent of outpatients with psychiatric conditions used alternative medication in addition to routine pharmacotherapy. Common alternative medications, including Ginkgo biloba, ginseng, St. John's wort, valerian, and kava kava, have CNS effects which might interfere with ECT. The treating psychiatrist should be made aware of a patient's herbal supplement use, to determine if the herbal products should be tapered prior to initiating ECT.

In addition, theophylline has been reported to cause status epilepticus after ECT and so should be tapered prior to treatment. The treating psychiatrist may also choose to taper or discontinue antidepressants and/or other psychotropics prior to treatment.

A summary of management strategies for preexisting medical conditions is shown in a table.

Strategies to reduce the risk of cardiac complications — The 2007 American Heart Association/American College of Cardiology (AHA-ACC) guideline update for noncardiac surgery assigns procedure-related risk to several types of surgery. Though not explicitly mentioned in the AHA-ACC guidelines, ECT can be treated like a low-risk procedure because it is usually well tolerated even in those at risk, the duration of hemodynamic changes is brief, and the mortality rate is low. As a result, it is difficult to prove a beneficial effect of any
intervention to reduce cardiovascular risk. Nevertheless, a cardiac risk assessment is warranted preoperatively. (See "Estimation of cardiac risk prior to noncardiac surgery").

Barring major clinical predictors of coronary risk (unstable angina, decompensated heart failure, severe valvular disease, malignant arrhythmias), most patients can undergo ECT with appropriate medical management [25]. Some have suggested that sedentary patients with intermediate risk factors should undergo further noninvasive risk stratification [26]. However, we feel that this population can proceed without further evaluation given the low morbidity of ECT and the potential use of prophylactic beta blockers.

**Postprocedure hemodynamic changes** — Short acting beta blockers are used to quickly treat persistent or severe tachycardia and hypertension after ECT. Other agents that have been useful for the treatment of postprocedure hypertension are intravenous nitroglycerin [27], nicardipine [28], and clonidine [12,25].

**Prophylactic beta blockers** — Short acting beta blockers, such as intravenous esmolol or labetalol, can be used to prevent transient and persistent hypertension and tachycardia [27-33]; glycopyrrolate is administered to prevent bradycardia in these individuals. Several authors have raised concern regarding widespread use of beta blockers because these drugs have been implicated in cases of prolonged asystole [9,34,35]. However, in these reported cases, atropine was not used and in some cases patients had subconvulsive treatments that can also cause bradycardia.

While it is clear that treatment of postprocedure hemodynamic changes is indicated, use of prophylactic beta blockers to reduce risk for patients undergoing ECT is controversial. The overall risk of cardiovascular complications due to ECT is low and no studies have documented reduced cardiac complications with the use of short acting beta blockers before ECT.

The 2001 American Psychiatric Association guidelines list three considerations for the use of prophylactic short acting beta blockers, that also are relevant in patients taking long acting beta blockers [7]:

- Beta blockers may increase the risk of asystole (though this appears to be less of a risk if atropine is used [10]).
- The absolute rise in the heart rate and blood pressure with ECT is no greater in those with baseline elevated blood pressure or heart rate [36,37].
- There is a theoretical risk that excessive blunting of the hemodynamic response with beta blockers may reduce the necessary supply of oxygen to the brain to protect against decreased seizure length and cognitive side effects.

In addition, there is some evidence that beta blockers might result in a decrease in seizure length and potentially decrease the efficacy of ECT [33,38], although this is not found in all studies [39,40].

The American Psychiatric Association chose not to make a specific recommendation with regard to using prophylactic beta blockers but does recommend that patients with unstable hypertension
be stabilized prior to starting ECT [7]. We do not recommend routine use of beta blocker in low risk patients, given the theoretical risks and unclear benefit. We suggest the use of prophylactic short-acting intravenous beta blockers for patients at very high risk of complications from transient hypertension (eg, intracranial aneurysm, unstable angina or recent myocardial infarction) [19]. Collaboration with the treating psychiatrist, anesthesiologist, and cardiology consultation is beneficial in such patients.

Typical bolus doses of labetalol are 5 to 20 mg IV, and for esmolol, 10 to 50 mg IV. The most authoritative study of the use of beta blockers during ECT found that labetalol produced dose-dependent reduction in HR and rate pressure product (RPP) using 5 and 10 mg doses, compared to placebo [41]. It is not necessary to prevent the transient hypertension and tachycardia associated with the seizure in most patients; the main goal of the use of these agents is to reduce the risk of myocardial ischemia (a possible result of the increased oxygen demand associated with tachycardia) in patients with coronary artery disease. Other agents, such as calcium channel blockers or nitroglycerine, are sometimes given during ECT [42].

**Other prophylactic medications** — Medications other than beta blockers have been used to try to prevent postprocedure hemodynamic changes. As an example, a small randomized trial found that prophylactic intravenous nicardipine was effective in a dose-dependent fashion at minimizing the acute hemodynamic response to ECT without shortening the duration of seizure [43].

**Coexisting cardiac disease**

**Hypertension** — Patients should receive their routine antihypertensive (other than diuretics) with a small sip of water approximately two hours before ECT. Diuretics should not be given, because it is better for the bladder to be empty during the procedure to prevent the patient from soiling himself as a result of the seizure.

**Coronary heart disease** — Antianginals such as nitrates and beta blockers should be continued in patients with documented coronary heart disease who are already taking them. Patients taking long acting beta blockers should receive atropine or glycopyrrolate with induction given the potential increase in the risk of bradycardia and a small study that showed that atropine may be protective [10].

If ECG changes or chest pain occur during the treatment period, treatments should be postponed until the patient is evaluated and treated for potential ischemia [25,44].

**Heart failure and valvular disease** — ECT should be delayed in patients with decompensated heart failure or significant valvular disease, pending cardiology consultation and completion of a thorough evaluation and optimization of cardiac status. One report of ten patients who completed ECT (total of 144 ECT sessions) with severe aortic stenosis (valve area 0.7 to 1.0 cm2) noted good control of blood pressure and heart rate with continuation of chronic antihypertensive medications and/or addition of a short-acting beta blocker [45]. All patients were asymptomatic and tolerated ECT. There were no deaths attributable to ECT or aortic stenosis. Two patients had drops in blood pressure requiring intervention, and seven patients required intravenous
medication to control systolic blood pressure >180 mm Hg. In patients with severe aortic stenosis, clinicians should strive for tight control of blood pressure and heart rate with attention to avoiding excessive preload or afterload reduction. Little data are available for management of patients with less severe valvular disease, though ECT can be performed safely, with appropriate precautions, in most patients with underlying cardiac conditions [46].

In patients with a remote history of heart failure or compensated disease, we recommend a baseline echocardiogram, if not recently performed, to assist with periprocedure management. In those with compensated systolic dysfunction, one may simply continue diuretics and vasodilator therapy and minimize volume overload. In patients with diastolic dysfunction, control of blood pressure should limit the occurrence of flash pulmonary edema. This group will benefit from prompt treatment of post-seizure hypertension if it develops.

Pacemakers and implantable defibrillators — Patients with pacemakers and automatic implantable cardiac defibrillators (AICDs) can safely undergo ECT. As an example, in a case series of ECT in 26 patients with pacemakers and three patients with AICDs there was only one serious cardiac event, an episode of supraventricular tachycardia [47].

The clinical team should be prepared to deactivate pacemakers with a magnet if any aberrant signals occur [25]. A cardiologist should deactivate AICDs before induction and reactivate the device after the seizure is complete to avoid excessive charge. The patient should have continuous ECG monitoring while the defibrillator is deactivated [48].

Coexisting neurologic and neurosurgical disease

Brain tumors — Early recommendations considered brain tumors and other space occupying lesions to be absolute contraindications to ECT [49]. This concern was based upon the observation that ECT raises cerebral blood flow, which in the presence of a brain tumor could translate into an increase in intracranial pressure (ICP) and neurologic deterioration.

These concerns were illustrated in a review of 35 patients with brain tumors undergoing ECT, 74 percent of whom had major adverse neurologic and cognitive side effects, with a one-month mortality of 28 percent [50]. Notable characteristics of patients who suffered neurologic deterioration included the presence of a depressive illness, no previous psychiatric illness or ECT, headache, and the presence of even soft neurologic findings on exam. However, this study may have had a selection bias given that only one patient was known to have a tumor before treatment.

Subsequently, one group reviewed 10 cases of successful treatment [51-54]. Eight patients had meningiomas in differing locations, and two patients had metastatic breast cancer. No one had abnormal neurologic examinations or evidence of increased ICP at baseline, and four had normal spinal fluid measurements. There were no adverse events other than a prolonged seizure after one treatment. Most recently a case report described successful treatment of a patient with primary brain cancer and documented elevated ICP [55]. He was treated with steroids and short acting beta blockers to minimize edema and elevations in blood pressure during the clonic phase of seizure.
Based on this information, ECT is probably safe in patients with brain tumors as long as there is no evidence of elevated ICP. Given that the available safety data are in the form of case series or reports, we agree with the American Psychiatric Association that the decision should be made on a case by case basis with the involvement of neurologic and possibly neurosurgical consultants [7]. Evaluation should consist of a detailed history and physical examination to seek evidence suggestive of elevated ICP, such as headache, papilledema, or abnormal neurologic examination. Any such evidence should prompt further investigation such as head CT or MRI. Although data are limited, providers have successfully used steroids to minimize edema [54,55], and neurologic consultants may consider using them in patients in whom elevated ICP cannot be ruled out [56,57].

**Stroke** — Patients who have suffered a stroke have a high rate of depression. In a study of 14 patients with completed strokes undergoing ECT one month or more after the event, none had deleterious neurologic sequelae [58]. Similarly, a second study of 24 patients with a history of stroke found no difference in the efficacy or cardiac complication rate compared with controls [59]. Rates of delirium were similar in both groups, although within the study group all patients with delirium had their strokes within the preceding year. Adequate blood pressure control is important for such patients.

**Dementia** — Reports exist of successful ECT in depressed patients with dementia and organic brain disease, with efficacy rates similar to that of nondemented patients [60,61]. In a review of 135 patients with organic and depressive dementia, 21 percent developed delirium or cognitive and memory deficits; all but one had cleared by time of discharge from the hospital [60]. The severity of delirium correlates with the degree of underlying dementia or organic brain syndrome, but is transient and does not interfere with treatment [61]. The clinical staff should be aware that delirium is a potential side effect in demented patients. These patients can often be managed with reassurance and alterations in level of supervision [62].

**Neuromuscular disease** — In patients with neuromuscular disease, particularly post-polio syndrome, depolarizing muscle relaxants such as succinylcholine may lead to severe hyperkalemia and circulatory collapse [63,64]. Thus, depolarizing muscle relaxants should be avoided. Patients with neuromuscular disease may receive short-acting, non-depolarizing agents for neuromuscular blockade [65].

**Epilepsy** — Use of ECT and anticonvulsants in patients with epilepsy is discussed separately. (See "Overview of electroconvulsive therapy (ECT) for adults", section on 'Psychotropic drugs'.)

**Diabetes** — There is no clear evidence of an effect of ECT on blood sugar control in diabetic patients. In a study of 19 patients with insulin-requiring type II DM, ECT itself did not lead to significant acute changes in blood sugar [66]. Individual patients who developed hyperglycemia or hypoglycemia after ECT were found to have changed their behaviors (eating or activity) in response to resolving depression, and these behavioral changes were felt to be responsible for the change in blood sugar control.
Patients generally do not take anything by mouth on the morning of ECT. As a general rule, we recommend holding oral hypoglycemic agents on the morning of the procedure. For insulin-requiring diabetics, on the morning of the procedure we recommend giving half the usual long-acting insulin dose and withholding short-acting insulin. A more detailed discussion of these issues can be found elsewhere. (See "Perioperative management of diabetes mellitus").

Anticoagulation — The safety of ECT in patients who are anticoagulated is controversial because of concerns about a possible increase in the risk of intracerebral hemorrhage [67]. In a case series of 35 patients receiving long-term anticoagulation with warfarin who underwent 284 ECT treatments, no major adverse effects occurred [68]. The INR on the day of ECT was below subtherapeutic (below 2.0) 36 percent of the time and supratherapeutic (above 3.5) only 3 percent of the time. These data suggest that if there is an increased risk of ECT in patients appropriately anticoagulated with warfarin, that risk is small.

Pregnancy — The treatment of psychiatric conditions in pregnancy poses challenges, as psychotropic medications may have significant side effects in both mother and fetus. However, ECT is generally thought be safe in pregnant patients by the American Psychiatric Association and the American College of Obstetricians and Gynecologists [7,69]. The safety of ECT and modifications in technique for treating pregnant patients are discussed separately. (See "Bipolar disorder in pregnant women: Treatment of mania, hypomania, and mixed episodes", section on 'Electroconvulsive therapy' and "Bipolar disorder in adults: Teratogenic and postnatal risks of pharmacotherapy", section on 'Electroconvulsive therapy' and "Technique for performing electroconvulsive therapy (ECT) in adults", section on 'Pregnancy'.)

As a pregnant patient considers ECT, the informed consent and evaluation should be performed with an obstetrician and an anesthesiologist.

RECOMMENDATIONS

- Perform a history and physical examination and obtain an ECG in all patients before planned ECT. Identify risk factors for cardiac complications of the procedure using one of the published cardiac risk indices or guidelines. Risk factors include coronary heart disease, valvular disease, and heart failure, as well as diabetes mellitus, hypertension, hypercholesterolemia, and advanced age. Suggestion for management of preexisting conditions is shown in a table (table 3).
- Delay ECT for patients with unstable angina, decompensated heart failure, or severe symptomatic valvular disease until these conditions are stabilized. Cardiology consultation is recommended in these patients.
- We do not suggest routine use of prophylactic beta blockers, which might decrease the seizure duration and increase risk of asystole. However, short-acting intravenous beta blockers should be considered for patients at very high risk of complications from transient hypertension (eg significant aortic stenosis, intracranial aneurysm, unstable angina or recent myocardial infarction). Cardiology consultation is useful in patients at very high risk.
- Continue nitrates, beta blockers, and other antihypertensive drugs in patients with preexisting cardiac conditions. If a patient is on a beta blocker, use atropine with induction to reduce the risk of asystole.
- Although ECT is likely safe for patients with high-risk neurosurgical lesions including recent stroke and brain tumor, neurosurgical consultation is recommended, as ECT is never an emergent procedure.
- Diabetic patients should generally hold oral hypoglycemics and short-acting insulin the morning of ECT, and the dose of long-acting insulin should be halved.
- Warfarin therapy can be continued in patients receiving ECT who have an INR below 3.5 and a strong indication for anticoagulation.
**TABLE 1. Indications for ECT**

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<tr>
<th>Definitely effective</th>
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<tr>
<td>Major depression</td>
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<td>Refractory to antidepressant therapy</td>
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<td>Need exists for rapid treatment response, such as in pregnancy</td>
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<td>Medical comorbidities prevent the use of antidepressant medication</td>
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<td>Previous response to ECT</td>
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<td>Psychotic features</td>
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<tr>
<td>Catatonic stupor</td>
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<tr>
<td>Severe suicidality</td>
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<td>Food refusal leading to nutritional compromise</td>
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<tr>
<td>Bipolar disorder</td>
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<td>Mania</td>
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<td>Atypical psychosis</td>
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<td>Schizophrenia</td>
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<tr>
<th>May be effective</th>
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<tr>
<td>Neuroleptic malignant syndrome</td>
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<td>Organic delusional disorder</td>
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<td>Organic mood disorder</td>
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<tr>
<td>Obsessive-compulsive disorder</td>
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<tr>
<td>Neuroleptic-induced Parkinsonism</td>
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<td>Neuroleptic-induced tardive dyskinesia</td>
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<td>Catatonia secondary to medical conditions</td>
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<tr>
<th>Condition</th>
<th>Management</th>
<th>Comments</th>
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<td>Aortic stenosis</td>
<td>Echocardiography should be performed to assess severity if not done within the past year or if there is a change in symptoms. If stenosis is moderate or severe, consult cardiologist and reassess indication for ECT.</td>
<td>Limited data suggest that ECT is safe with the use of short-acting intravenous beta-blockers to minimize procedure-related hypertension and tachycardia.</td>
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<td>Asthma or chronic obstructive pulmonary disease</td>
<td>Discontinue theophylline by tapering the dose, if possible. Continue outpatient regimen of bronchodilators and inhaled glucocorticoids. Provide standard treatment during an asthma exacerbation (inhaled beta-agonists and, if necessary, glucocorticoids, before proceeding with ECT).</td>
<td>Theophylline increases the risk of status epilepticus after ECT.</td>
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<tr>
<td>Atrial fibrillation</td>
<td>Continue outpatient medications for control of heart rate. Control heart rate with calcium-channel blockers if needed. Manage anticoagulation as described below.</td>
<td>ECT appears to be safe in patients with atrial fibrillation. Patients may have conversion to and from sinus rhythm during ECT. The effect of spontaneous rate conversion on embolization rates is unknown.</td>
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<tr>
<td>Coronary artery disease (stable)</td>
<td>Continue medications such as aspirin, statins, antihypertensive agents, and antianginal medications, including nitrates for chronic cardiac conditions. Continue aspirin and clopidogrel in patients with coronary stents.</td>
<td>Discontinuation of long-term cardiac medications on the morning of the procedure increases the risk of cardiac ischemia.</td>
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<tr>
<td>Diabetes</td>
<td>Measure blood glucose levels before and after ECT treatment. Give half the usual amount of long-acting insulin the morning of the procedure. Withhold oral agents until patient can eat. Provide short-acting insulin to treat elevations in blood glucose level. Perform ECT early in the morning if possible.</td>
<td>The effect of ECT on blood glucose is unpredictable because of changes in diet, appetite, and energy level that may result from ECT. Individual ECT treatments raise blood glucose levels in patients with diabetes to the same degree as in patients without diabetes.</td>
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<td>Hypertension (poorly-controlled)</td>
<td>Start or intensify antihypertensive medications; delay ECT until blood pressure is &lt;140/90 mmHg. Avoid beta-blockers.</td>
<td>Blood pressure increases during the postictal phase of ECT. Beta-blockers may shorten the seizure duration and reduce the efficacy of ECT.</td>
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<td>Hypertension (well-controlled)</td>
<td>Continue usual antihypertensive medication(s).</td>
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<tr>
<td>Implantable cardioverter-defibrillator (ICD)</td>
<td>Turn off detection mode of ICD during ECT. Perform continuous electrocardiographic monitoring throughout treatment with careful attention to grounding. Place resuscitative equipment by the patient's bedside in the event that external defibrillation is necessary.</td>
<td>ECT appears to be safe in patients with an ICD.</td>
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<tr>
<td>Implantable pacemaker</td>
<td>Test the pacemaker before and after ECT. A magnet should be available at the patient's bedside in the event that electrical interference leads to pacemaker inhibition and bradycardia.</td>
<td>In rare cases, postprocedural supraventricular tachycardia can occur.</td>
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<td>Need for long-term anticoagulation</td>
<td>Continue anticoagulation to maintain an international normalized ratio of up to 3.5, unless there is an increased risk of intracranial hemorrhage (eg, intracranial mass or aneurysm).</td>
<td>ECT appears to be safe in patients who require long-term anticoagulation.</td>
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<tr>
<td>Pregnancy</td>
<td>The informed-consent and risk-stratification process should include an obstetrician and an anesthesiologist. In addition to standard monitoring of the patient, noninvasive fetal monitoring should be used after 14-16 weeks. After 24 weeks, a nonstress test with a tocometer should be performed before and after treatments.</td>
<td>Pregnancy would require modification of the anesthetic technique, positioning of the patient, and monitoring requirements.</td>
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REFERENCES


