Technique for performing electroconvulsive therapy (ECT) in adults

AUTHOR: Charles Kellner, MD
SECTION EDITORS: Peter P Roy-Byrne, MD, Stephanie B Jones, MD
DEPUTY EDITOR: David Solomon, MD

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Literature review current through: **Oct 2023.**
This topic last updated: **Sep 23, 2022.**

INTRODUCTION

Electroconvulsive therapy (ECT) is practiced widely in the United States and the rest of the world [1,2]. It is used mainly to treat severe depression, but is also indicated for patients with other conditions, including bipolar disorder, schizophrenia, schizoaffective disorder, catatonia, and neuroleptic malignant syndrome.

There is no question about the efficacy and safety of ECT. Nevertheless, it remains controversial and stigmatized because of misinformation and outdated perceptions about how the treatment is performed.

The goal of ECT is to produce a generalized cerebral seizure under general anesthesia. The technique affects the rate of remission with ECT, which varies from 20 to 80 percent in research studies using different procedures [1]. However, response and remission rates in clinical practice, using standard techniques, are at the higher end of that range.

The technique for performing ECT is reviewed here. Other topics provide an overview of ECT and discuss the indications for treating unipolar major depression, bipolar disorder, and schizophrenia with ECT; the efficacy of ECT for treating unipolar depression, bipolar disorder, schizophrenia, and neuroleptic malignant syndrome; and medical consultation for ECT.

- (See "Overview of electroconvulsive therapy (ECT) for adults".)

- (See "Unipolar major depression in adults: Indications for and efficacy of electroconvulsive therapy (ECT)".)
- (See "Evaluation and management of treatment-resistant schizophrenia".)
- (See "Neuroleptic malignant syndrome", section on 'Electroconvulsive therapy'.)
- (See "Medical evaluation for electroconvulsive therapy".)

SETTING

ECT is performed in a dedicated ECT treatment suite, a hospital postanesthesia care unit, or another ambulatory surgery site.

ECT may be performed on an inpatient or outpatient basis, and there is an increasing trend toward outpatient ECT. Patients with severe medical or psychiatric illness may start ECT on an inpatient basis and as they improve, switch to outpatient treatment. Continuation and maintenance ECT are almost always given on an outpatient basis. (See "Overview of electroconvulsive therapy (ECT) for adults" and "Overview of electroconvulsive therapy (ECT) for adults", section on 'Continuation and maintenance ECT'.)

PRE-ECT ORDERS

The orders for each ECT treatment include:

- No food or drink (NPO) after midnight
- Intravenous line

The patient receives general anesthesia as part of ECT and thus should not eat solid food for six to eight hours prior to the procedure, and not drink clear liquids for two hours, except for necessary medications with a small sip of water [3,4]. In particular, patients should take their usual prescribed cardiovascular medications. The patient should empty his or her bladder before the treatment.

TREATMENT PROTOCOL

ECT is administered by a team that typically includes a psychiatrist, anesthesiologist, and nurse. Most patients are treated according to a protocol that addresses the following issues [4]:

- **Patient safety** – Confirm the identity of the patient and that the patient has had nothing to eat or drink (NPO), has taken necessary medications, and signed the informed consent

form. In addition, the patient is monitored after the seizure for approximately 30 to 60 minutes.

- **Equipment** – ECT device, electrodes, device to monitor vital signs and pulse oximetry, nasal cannula or face mask to provide supplemental oxygen, bag valve mask ("Ambu bag"), oxygen supply, suction apparatus, nerve stimulator to assess degree of motor relaxation, electromyograph, and blood pressure cuffs (one for the arm and one for the ankle).
- **Medications** – Anesthetic and neuromuscular blocker; anticholinergic and cardiovascular medications are used as indicated.
- **Documentation** – Record vital signs, ECT device settings, and duration of seizure.

PHYSIOLOGIC MONITORING

Physiologic functions monitored during ECT include:

- Vital signs
- Blood oxygen saturation
- Cardiac rhythms (ECG)
- Electrical activity of the brain (electroencephalography [EEG])

In addition, electromyography (EMG) may be performed to evaluate the electrical activity of the muscles and a nerve stimulator may be used to monitor the effects of succinylcholine (used to reduce tonic-clonic contractions).

EEG monitoring is crucial because it enables the practitioner to confirm that a cerebral seizure has occurred and has ended in a timely fashion. The EEG is typically recorded with one or two channels, from right and left frontal and mastoid positions (figure 1). ECT devices display the EEG tracing on a paper record and/or computer screen, and may also provide information about the adequacy of the recorded seizure by means of automated indices. Despite the device interpretation, interpretation of the EEG remains an important skill for the ECT practitioner.

EMG is generally recorded from the right foot in order to measure the length of the motor component of the seizure. This corresponds with the possible use of right unilateral electrode placement. A blood pressure cuff is placed and inflated on the right ankle to prevent succinylcholine from entering the foot. An alternative to using the EMG is to simply place a blood pressure cuff on the ankle and have a member of the treatment team watch the foot after the electric stimulus is administered and record the duration of tonic-clonic contractions.

ELECTRODE PLACEMENT

Standard choice — The electric current used to induce the seizure passes briefly through the brain via two electrodes (either metal or disposable adhesive) applied to the scalp. Psychiatrists typically position the stimulating electrodes in one of three ways (figure 2) [5]:

- **Bilateral** – Bilateral, also known as bitemporal, is the original, "gold standard" placement. One electrode is placed on each temple, with the center of the electrode placed 2 to 3 cm above the midpoint of the line connecting the outer canthus of the eye and the external auditory meatus on each side of the head. Bilateral placement has the greatest antidepressant efficacy and quickest speed of response, but may cause the most memory impairment.

- **Right unilateral** – Right unilateral electrode placement consists of placing one electrode on the right temple and one on the scalp, just to the right of the vertex (d'Elia placement). Specifically, one electrode is positioned as in bilateral on the right side, and the center of the other electrode is placed 2 to 3 cm to the right side of the vertex of the skull. This technique avoids initial stimulation of the left cerebral hemisphere, which is usually dominant for language functions. Right unilateral results in slightly lower remission rates, but generally causes fewer adverse cognitive effects such as memory impairment.

- **Bifrontal** – Bifrontal consists of placing each electrode on the forehead above the outer canthus of each eye. The center of each stimulating electrode is placed 4 to 5 cm above the outer canthus of the eye along a vertical line perpendicular to a line connecting the pupils. Some clinicians think that bifrontal is as effective as bilateral, although there is a much smaller evidence base for bifrontal. Cognitive impairment with bifrontal may be comparable to right unilateral. In a randomized, assessor blinded trial that compared bifrontal with right unilateral ECT (up to 16 treatments) in 73 depressed older adult patients, cognitive function as assessed with the Mini-Mental State Examination was comparable for the two groups [6].

We suggest:

- Bilateral ECT in more urgent clinical situations, such as life-threatening depression, profound distress, and/or catatonia

- Right unilateral ECT when there is a significant concern about minimizing retrograde amnesia

Patients whose symptoms do not respond to right unilateral ECT can be switched to bilateral [7]. We suggest doing so after three to five treatments, depending upon the patient's clinical status and preference [8].

Clinicians should avoid stimulating over or adjacent to a skull defect, regardless of the type of electrode placement [3]. Thus, the pre-ECT evaluation should address whether there is a history of cranial injury or neurosurgery with resultant skull defect, which may indicate the need for skull radiographs. (See "Overview of electroconvulsive therapy (ECT) for adults", section on 'Pre-ECT evaluation'.)

Efficacy — For depressed patients, multiple randomized trials suggest that the efficacy of bilateral ECT and right unilateral ECT is either comparable, or that bilateral ECT is modestly superior to right unilateral ECT:

- A 2003 meta-analysis of 22 randomized trials (1408 patients) found that bilateral ECT was superior to unilateral ECT; however, the difference was small and likely to be of only minimal to moderate clinical significance [9].

- A 2017 meta-analysis of seven randomized trials (792 patients) specifically compared **moderate dose** bilateral ECT (1.0 to 2.5 times seizure threshold) with **high dose** right unilateral ECT (five to eight times seizure threshold), reflecting standard practice in administering bilateral and right unilateral ECT [10]. Remission in the two groups was comparable. Additional information about stimulus dose is discussed elsewhere in this topic. (See 'Stimulus dose' below.)

A limitation of the randomized trials in both meta-analyses is that severely ill patients were excluded or could not provide informed consent to participate [11].

Some depressed patients do not respond to unilateral ECT and are then switched to bilateral ECT with good results [12-14]. In a randomized trial that compared right unilateral ECT with bilateral ECT in patients with major depression, 31 patients assigned to right unilateral ECT did not substantially improve after eight or more treatments and were then switched to bilateral ECT [15]. Among these 31 patients, remission occurred in 15 (48 percent).

A meta-analysis of five randomized trials in patients with unipolar or bipolar depression (n = 338) found that improvement with bilateral ECT and bifrontal ECT was comparable [16]. A second meta-analysis of six randomized trials (n = 400 patients) found that improvement with right unilateral ECT and bifrontal ECT was comparable [16].

Side effects — Cognitive impairment is worse with bilateral (bitemporal) ECT than right unilateral ECT, but the difference is generally short lived:

- A meta-analysis of 39 observational studies evaluated neuropsychological function in 1415 depressed patients who were treated with ECT and assessed with standardized tests at baseline and after the course of ECT was completed [17]. Tests conducted within three days of the last ECT treatment showed that impairment of global cognition, delayed verbal memory, and autobiographical memory were greater with bilateral ECT than unilateral ECT. However, testing more than three days post-ECT found that cognition was comparable for the two electrode placements.

- One randomized trial compared bilateral and unilateral ECT in 149 patients with unipolar or bipolar major depression and found that the adverse cognitive effects of each placement were comparable [18,19]. This somewhat surprising finding may be attributable to the stimulus dosing of right unilateral at six times seizure threshold, whereas the stimulus dosing for bilateral ECT was one and a half times seizure threshold. (See 'Stimulus dose' below.)

- A second randomized trial compared bilateral ECT (one and a half times seizure threshold) with right unilateral ECT (six times seizure threshold) in 138 patients with unipolar or bipolar major depression, and found that recall of autobiographical memory was better with unilateral than bilateral ECT, both at the end of treatment and at the six-month follow-up assessment [20]. In addition, the median time to recovery of orientation following ECT sessions was faster with unilateral than bilateral ECT (19 versus 26 minutes). However, cognitive functioning at the end of treatment and at the six-month follow-up was comparable in other domains, including global cognition, executive functioning (eg, planning), attention and working memory, verbal learning for delayed recall, psychomotor speed, and visuo-spatial functioning and memory.

A meta-analysis of four randomized trials (n = 287 patients with unipolar or bipolar depression) found that cognitive impairment for up to seven days was greater with bilateral ECT than bifrontal ECT; however, heterogeneity across studies was large [16]. A second meta-analysis of five randomized trials (n = 558 patients) found that cognitive impairment was comparable with right unilateral ECT and bifrontal ECT [16].

STIMULUS

For many years it was assumed that the electric stimulus was relevant only for its ability to elicit a grand mal seizure. It is now clear that the type and intensity of the stimulus contribute to the efficacy and adverse cognitive effects of ECT [21,22].

Stimulus type — The type of stimulus used in ECT devices is a brief pulse (0.5 to 2.0 milliseconds) or ultra-brief pulse (<0.5 milliseconds) wave form. Brief pulse is considered standard due to its efficacy compared with ultra-brief pulse; however, ultra-brief pulse is a reasonable alternative based upon its superior tolerability [23]. The choice thus depends upon urgency of response, prior treatment history, and patient preferences. The outmoded sine wave stimulus should not be used because it causes more cognitive impairment [22].

Evidence regarding the use of brief pulse or ultra-brief pulse stimuli includes a systematic review of six acute studies (four randomized trials and two prospective observational studies) that compared right unilateral brief pulse ECT with right unilateral ultra-brief pulse ECT in 689 patients with major depression [24]. Remission occurred in more patients treated with brief pulse than ultra-brief pulse (45 versus 34 percent). In addition, the number of ECT treatments that were required was less with brief pulse (9 versus 10). However, retrograde memory, anterograde memory, and global cognitive functioning were each worse with brief pulse than ultra-brief pulse. The magnitude of cognitive impairment was mild to moderate, depending upon the specific cognitive domain.

Stimulus dose — The dose of the electric stimulus (amount of electricity) affects efficacy, speed of response, and adverse cognitive effects. Stimulus dose is usually described in terms of either absolute charge (in millicoulombs) or multiple of seizure threshold. Seizure threshold may be estimated or determined empirically at the first treatment session [25,26]. Establishing the seizure threshold is accomplished by initially setting the ECT device to use a small amount of charge to induce the seizure. If this dose does not produce a seizure, the device is then reset to deliver an incrementally higher dose in an attempt to induce a seizure. This process is repeated until a seizure occurs, which then establishes the seizure threshold. The parameters that determine the stimulus dose include the current and duration of each electrical pulse, frequency of pulses, and the total duration of the series of pulses.

Stimulus dosing strategies differ between electrode placements. The suggested stimulus dose for bilateral or bifrontal ECT is 1.5 to 2 times seizure threshold. The suggested dose for right unilateral is approximately six times seizure threshold, based upon data showing that seizures need to be elicited with stimuli that are several times greater than seizure threshold for unilateral placement to be maximally effective [4,21,27]. (See 'Electrode placement' above.)

The higher dose required for right unilateral occasionally becomes a problem because ECT devices manufactured in the United States are limited to a charge output of 576 millicoulombs by Food and Drug Administration regulations. This prevents a small number of patients with a relatively high seizure threshold from being treated at fully six times seizure threshold [28].

It is common for seizure threshold to increase during an acute course of ECT. This may necessitate incrementally increasing the dose of the electric stimulus periodically.

Clinicians should follow the specific instructions of the ECT device as specified by the manufacturer.

ANESTHESIA TECHNIQUE

The goal of anesthesia for ECT is to provide the patient with a safe, comfortable experience [29]. The anesthesiologist preoxygenates the patient and administers medications to induce unconsciousness and to relax or paralyze skeletal muscles. An anticholinergic drug may be given prior to ECT to prevent bradycardia or asystole.

The subsections below summarize anesthesia for ECT; a more detailed discussion is presented separately. (See "Anesthesia for electroconvulsive therapy".)

Preoxygenation — The patient is preoxygenated with supplemental oxygen via nasal cannula or face mask while the procedure is being set up, with the goal of maintaining oxygen saturation at or near 100 percent. This makes ECT safer with fewer adverse effects than either a spontaneous (epileptic) seizure or ECT in the past. In addition, the patient is often hyperventilated via bag valve mask immediately prior to delivering the electrical stimulus. This induces cerebral hypocarbia, which increases seizure intensity [30].

Anticholinergic medication — Premedication with an anticholinergic agent, either glycopyrrolate (0.2 mg, intravenous [IV]) or atropine (0.4 mg, IV), is often used to prevent vagally-mediated bradycardia and excess oral and respiratory secretions. Bradycardia with hypotension, and in some cases asystole, is most common at the first ECT treatment, when there is a greater chance of administering a subconvulsive stimulus that leads to parasympathetic discharge unopposed by subsequent sympathetic activity from a seizure.

However, anticholinergics may cause or exacerbate tachycardia. (See 'Cardiovascular medication' below.)

Anesthetic medication — The goal of anesthesia is an adequate, but not excessively deep, level of anesthesia that does not interfere with inducing an effective grand mal seizure. The

induction agent of choice is methohexital, given intravenously at a dose of 0.75 to 1 mg/kg. Propofol is a commonly used alternative, but it is more potently anticonvulsant than methohexital and can reduce seizure duration. Other induction agents include thiopental, etomidate, and ketamine [29,31]. There has been interest in the use of ultrashort-acting narcotics such as remifentanil to replace at least some of the standard induction agents, but this has not been widely adopted [32].

Evidence that supports using methohexital rather than propofol for depressed patients treated with ECT includes a meta-analysis of two randomized trials (n = 78 patients) that compared the two drugs and found that seizure duration was longer with methohexital [33]. A second analysis (four trials, 165 patients) showed that improvement of depression was comparable with the two drugs [33].

A retrospective, national registry study with over 5000 patients found that ECT outcomes were better with relatively low doses of thiopental or propofol anesthesia, compared with high doses [34].

Muscle relaxation medication — Skeletal muscle relaxation is used during ECT to minimize the motor seizure and prevent musculoskeletal injury. This is particularly important for patients with osteoporosis. The standard agent for muscle relaxation is succinylcholine, a depolarizing neuromuscular blocker, given intravenously at a dose of 0.75 to 1 mg/kg [35]. Most patients recover muscle strength within minutes of administration (elimination half-life 47 seconds) [36].

Nondepolarizing muscle relaxants such as cisatracurium, mivacurium (where available), rocuronium, or vecuronium are used only in special circumstances when succinylcholine cannot be used. Such circumstances include pseudocholinesterase deficiency, severe neuromuscular disease or injury (quadriplegia, amyotrophic lateral sclerosis, or muscular dystrophy), severe muscular rigidity, recent severe and widespread burns, or significant hyperkalemia [3,31]. The availability of sugammadex as a reversal agent for rocuronium or vecuronium has simplified the use of these nondepolarizing muscle relaxants in ECT [37].

Pseudocholinesterase is needed to metabolize succinylcholine, and its absence will lead to prolonged apnea and muscle relaxation. A pseudocholinesterase level (also referred to as dibucaine number) is measured only in patients with a personal or family history of prolonged apnea following exposure to muscle relaxants.

It is important to ascertain that the patient is unconscious before proceeding with the muscle relaxant, to avoid patient distress. The patient is rendered apneic by the muscle relaxant, at which point the anesthesiologist provides ventilation by bag and mask with 100 percent oxygen.

The adequacy of muscle relaxation is determined using a nerve stimulator and observing the decrement and eventual disappearance of response. In addition, the clinician can assess knee and plantar withdrawal reflexes, and can simultaneously observe for fasciculations in the calves and left foot and wait until they subside, usually within one to two minutes of administering the muscle relaxant. Perfusion of the right foot with the muscle relaxant is prevented by an inflated blood pressure cuff on the right ankle.

Despite administration of succinylcholine, the masseter muscles contract from direct electrical stimulation. Thus, prior to administering the electrical stimulus, a member of the treatment team inserts a bite block into the patient's mouth to protect the tongue and teeth. Proper technique is to ensure that the tongue is pushed inferiorly and posteriorly in the mouth, and that the chin is held firmly against the bite block.

Cardiovascular medication — Prophylactic medications may be administered immediately before or during ECT for the purpose of blunting the hypertensive, tachycardic response to the seizure-induced sympathetic discharge. The most commonly used drugs in this situation are beta blockers. The use of beta blockers for this purpose is discussed separately. (See "Medical evaluation for electroconvulsive therapy", section on 'Prophylactic beta blockers'.)

The sympathetic discharge during ECT may cause atrial and ventricular arrhythmias such as premature ventricular contractions or ventricular tachycardia. Increased heart rate and blood pressure increase myocardial oxygen consumption, with the potential for myocardial ischemia and malignant arrhythmias. Administration of beta blockers may be necessary to treat tachycardia, hypertension, and/or myocardial ischemia.

SEIZURE DURATION

Most therapeutic ECT seizures last 15 to 70 seconds on the electroencephalographic (EEG) recording. The EEG recording typically lasts 10 to 30 percent longer than the motor seizure [38].

Problems with ECT seizure induction include [3]:

- Missed seizures – The electrical stimulus does not induce a seizure
- Short seizures – Last less than 15 seconds
- Prolonged seizures – Last longer than two to three minutes

Short seizures (less than 15 seconds) may not be maximally effective, and prolonged seizures may be associated with increased cognitive impairment.

Missed seizures should be followed by a brief (eg, 20 seconds) period of hyperventilation and restimulation at an incrementally higher stimulus dose. Short seizures should be followed by a longer period of hyperventilation (eg, 60 seconds) and restimulation at an incrementally higher stimulus dose.

A prolonged seizure is a potentially serious complication that needs to be recognized and treated promptly. We suggest terminating seizures at two minutes. The standard procedure is to administer one-half the induction dose of the anesthetic (when it is either methohexital or propofol). Alternatively, a dose of benzodiazepine (eg, diazepam 5 mg intravenous [IV]) may be given [3].

Managing missed or short seizures — There are several procedures to use for persistent missed or short seizures [3,39-41]:

- Decreasing or discontinuing anticonvulsant mood stabilizers and benzodiazepines, if possible.
- Hyperventilating the patient before and during the seizure.
- Decreasing the anesthetic dose to the minimum compatible with full unconsciousness.
- Switching anesthetic to etomidate (0.15 to 0.30 mg/kg IV) or ketamine (1 to 2 mg/kg IV), which are less anticonvulsant than methohexital.
- Intravenous caffeine has been used to prolong seizures, but is no longer recommended because its clinical benefits are uncertain.

PREGNANCY

Many aspects of administering ECT to pregnant patients are comparable to the technique used for patients who are not pregnant. As an example, the electrical stimulus dose is not adjusted for pregnancy because it is not clear that pregnancy changes seizure threshold during ECT [42,43]. Electrical stimulus dosing is discussed elsewhere in this topic. (See 'Stimulus' above.)

However, pregnant patients should ideally receive ECT in settings that include psychiatrists who are experienced in administering ECT to pregnant patients, anesthesiologists with experience in providing anesthesia to pregnant patients for ECT, and obstetricians with access to fetal monitoring equipment [44-47].

ECT technique for pregnant patients should be modified as follows [43,47-50]:

- Avoid hyperventilating the patient (hyperventilation can diminish fetal oxygenation by decreasing placental blood flow and reducing the dissociation of oxygen from hemoglobin).

- Hydrate patients with intravenous fluids prior to each ECT treatment to minimize the risk of premature contractions.

- For pregnancies between 12 and 23 weeks of gestation, document the fetal heart rate before and after each ECT treatment.

- When the gestational age exceeds 20 weeks, place a wedge beneath the patient's right hip during each ECT treatment to displace the uterus from the aorta and vena cava and thus optimize maternal venous return, cardiac output, blood pressure, and uterine blood flow.

- For pregnancies ≥24 weeks of gestation (the fetus is considered viable if born at this gestation age), continuously monitor the fetal heart rate and uterine activity for 30 to 60 minutes before and after each ECT treatment. A clinician with experience placing monitoring devices and interpreting these tracings should be available.

- Administer ECT at facilities with resources for treating obstetric and neonatal emergencies.

Pregnant patients are at increased risk for gastric reflux and aspiration pneumonitis, which can be minimized with one or more of the following measures during each ECT treatment [42,43,47,48,51]:

- Withhold anticholinergic drugs – Glycopyrrolate or atropine may decrease lower esophageal sphincter tone and thus increase reflux. However, for pregnant patients who require an anticholinergic drug to prevent excessive bradycardia, we suggest glycopyrrolate, which does not cross the placenta as readily as atropine. Use of anticholinergic medication during ECT is discussed elsewhere in this topic. (See 'Anticholinergic medication' above.)

- Administer one or two of the following drugs 40 to 60 minutes prior to the procedure to mitigate the effects of aspiration if it occurs: a nonparticulate antacid (eg, sodium citrate 30 mL orally), a histamine-2 receptor antagonist (eg, famotidine 20 mg intravenously [IV]), a proton pump inhibitor (eg, omeprazole 20 mg orally), or prokinetic drug (eg, metoclopramide 10 mg IV).

- Intubate the patient, especially at week 25 of gestation and beyond – Intubation is the least preferred option for managing aspiration risk because of its associated morbidity,

especially during pregnancy due to weight gain, edema, and hypervascularity; even minor trauma can lead to profuse bleeding. Intubation of pregnant patients is discussed separately. (See "Airway management for the pregnant patient".)

The teratogenic risks and postnatal effects of ECT are discussed separately. (See "Teratogenicity, pregnancy complications, and postnatal risks of antipsychotics, benzodiazepines, lithium, and electroconvulsive therapy", section on 'Electroconvulsive therapy'.)

ISSUES RELATED TO COVID-19

Issues related to performing ECT during the coronavirus disease 2019 (COVID-19) pandemic are discussed separately. (See "COVID-19: Psychiatric illness".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Depressive disorders".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, “The Basics” and “Beyond the Basics.” The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword(s) of interest.)

- Basics topics (see "Patient education: Electroconvulsive therapy (ECT) (The Basics)")
- Beyond the Basics topics (see "Patient education: Electroconvulsive therapy (ECT) (Beyond the Basics)")

OUTSIDE SOURCES OF PATIENT EDUCATION

One of the requirements for obtaining informed consent for ECT is that patients receive adequate information about the procedure. Additional written material is available for patients and family members to augment discussions with the psychiatrist at http://jama.ama-assn.org/cgi/reprint/285/10/1390.

Educational material explaining ECT is also available as part of a document entitled "Brain Stimulation Therapies" that is published by the National Institute of Mental Health. This publication can be obtained through a toll-free number, 866-615-646, or online at the Web site http://www.nimh.nih.gov/health/topics/brain-stimulation-therapies/brain-stimulation-therapies.shtml. The Web site also provides information about depression in language intended for the lay public.

SUMMARY

- The goal of electroconvulsive therapy (ECT) is to produce a generalized cerebral seizure under general anesthesia. ECT technique may affect the rate of remission, which is reported to vary from 20 to 80 percent (but is typically nearer the high end of this range). (See 'Introduction' above.)

- The patient receives general anesthesia as part of ECT and thus should not eat solid food for six to eight hours prior to the procedure, and not drink clear liquids for two hours, except for necessary medications with a small sip of water. The patient should empty his or her bladder before the treatment. (See 'Pre-ECT orders' above.)

- The equipment used for the procedure includes the ECT device, device to monitor vital signs and pulse oximetry, nasal cannula or face mask to provide supplemental oxygen, bag valve mask, oxygen supply, suction apparatus, nerve stimulator to assess degree of motor relaxation, electromyograph, and blood pressure cuffs (arm and ankle). (See 'Treatment protocol' above.)

- The electric current used to induce the seizure passes briefly through the brain via two electrodes applied to the scalp. Psychiatrists typically position the stimulating electrodes in one of three ways: bilateral, right unilateral, or bifrontal. We suggest bilateral ECT in more urgent clinical situations, and right unilateral ECT when there is a significant concern about minimizing retrograde amnesia. (See 'Electrode placement' above.)

- The type of stimulus used in contemporary ECT devices is a brief pulse (0.5 to 2.0 milliseconds) or ultra-brief pulse (<0.5 milliseconds) waveform. Brief pulse is considered standard, but there is evidence that ultra-brief pulse stimuli may cause less cognitive impairment. The outmoded sine wave form of stimulus causes more cognitive impairment and should no longer be used. (See 'Stimulus type' above.)

- The dose of the electric stimulus is usually estimated empirically at the first treatment session. If the initial small dose does not produce a seizure, incrementally higher doses are given until a seizure occurs. The suggested stimulus dose at subsequent treat sessions for bilateral ECT is 1.5 to 2 times seizure threshold, and for right unilateral is 6 times seizure threshold. (See 'Stimulus dose' above and 'Electrode placement' above.)

- The patient is preoxygenated with supplemental oxygen via nasal cannula or face mask while the procedure is being set up. Premedication with an anticholinergic agent, either glycopyrrolate (0.2 mg, intravenous [IV]) or atropine (0.4 mg, IV), is often used to prevent vagally-mediated bradycardia. Prophylactic beta blockers may be administered immediately before or during ECT to blunt the hypertensive, tachycardic response to the seizure. (See 'Preoxygenation' above.)

- The anesthetic of choice is methohexital, given intravenously at a dose of 0.75 to 1 mg/kg. 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, given intravenously at a dose of 0.75 to 1 mg/kg. (See 'Anesthesia technique' above and "Anesthesia for electroconvulsive therapy".)

- Most therapeutic ECT seizures last 15 to 70 seconds on electroencephalographic recording. Technical concerns include seizures that are missed, short, or prolonged. (See 'Seizure duration' above.)

- ECT technique is modified for pregnant patients. (See 'Pregnancy' above.)

Topic 1712 Version 28.0

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