Overview of electroconvulsive therapy (ECT) for adults

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INTRODUCTION

Electroconvulsive therapy (ECT) uses a small electric current to produce a generalized cerebral seizure under general anesthesia. ECT 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, which is practiced widely in the United States and the rest of the world [1]. Nevertheless, it remains controversial and stigmatized because of misinformation and outmoded perceptions about how the treatment is performed [2].

This topic provides an overview of ECT. The indications for and efficacy of ECT in unipolar major depression, technique for performing ECT, medical consultation for ECT, and indications for and efficacy of ECT in bipolar disorder are discussed separately.

- (See "Unipolar major depression in adults: Indications for and efficacy of electroconvulsive therapy (ECT)".)
- (See "Technique for performing electroconvulsive therapy (ECT) in adults".)
- (See "Medical evaluation for electroconvulsive therapy".)
- (See "Bipolar disorder in adults: Indications for and efficacy of electroconvulsive therapy".)

HISTORY OF ECT

The use of seizures to treat psychiatric disorders has progressed over time, improving the efficacy and tolerability of ECT.

ECT began with observations that patients with schizophrenia often improved temporarily after a spontaneous seizure [3-5]. Initially, physicians induced seizures with medications. In 1938, Italian psychiatrists used an electric current to induce seizures as a treatment for schizophrenia [6]. ECT quickly spread around Europe and the rest of the world, and was first used in the United States in 1939. Patients suffered bone fractures occasionally and considerable anticipatory anxiety, but advances in anesthesia techniques in the 1950s allowed for the use of general anesthesia and muscle relaxation for ECT, which eliminated these problems.

The use of ECT in the United States has fluctuated [7,8]. ECT use declined in the 1970s, and subsequently increased, possibly as a result of greater treatment resistance to pharmacotherapy in depressed patients, increased recognition of the limitations of pharmacotherapy, and better public acceptance.

Current use — ECT is offered at most major psychiatric treatment centers. However, a study of a private insurance claims database in the United States found that among nearly one million patients with unipolar major depression or bipolar disorder, ECT was administered to only 0.25 percent [9].

ECT patients today are more likely to be of higher socioeconomic status, of White race, and receive ECT in a private sector psychiatric facility [7,10]. In addition, patients with mood disorders who are treated with ECT have higher rates of psychiatric comorbidity, compared with patients who are not treated with ECT [9]. State hospitals rarely offer the treatment, even though many of the patients would meet indications for ECT.

MECHANISM OF ACTION

Much is known about the neurobiology of ECT, but its exact mechanism of action remains to be elucidated [11]:

- Human and animal studies show that ECT increases release of monoamine neurotransmitters, particularly dopamine, serotonin, and norepinephrine [2,12-14]. ECT also enhances monoamine transmission by desensitizing presynaptic adrenergic autoreceptors.

- The neuroendocrine hypothesis suggests that ECT relieves depression by causing the hypothalamus or pituitary gland to release hormones, including prolactin, thyroid

stimulating hormone, adrenocorticotropic hormone, and endorphins [2].

- ECT has anticonvulsant properties (perhaps related to neurohormones and enhanced gamma-aminobutyric acid transmission), which has led to the suggestion that these properties are responsible for the therapeutic effects of the treatment [2,15].

- The neurotrophic/tropic hypothesis suggests that ECT works by inducing neurogenesis (brain structural plasticity) and increasing neurotrophic signaling, thus reversing many of the deleterious atrophic brain changes induced by severe, chronic depression [2,16-24]. Multiple studies demonstrate increases in gray matter volume and cortical thickness in several brain areas, most prominently the amygdala and hippocampus [25]. In addition, brain-derived neurotrophic factor appears to increase after ECT.

- Positron emission tomography studies demonstrate decreased metabolic activity in frontal and cingulate cortex after ECT [26].

- ECT is reported to change brain connectivity [27]. One functional magnetic resonance imaging study before and after successful treatment with ECT revealed a reduction in global connectivity within the left dorsal lateral prefrontal cortex [28], whereas another functional magnetic resonance imaging study found increased right hippocampal connectivity [29] and a structural study found increased connectivity in dorsal fronto-limbic circuits [30]. Change in connectivity is being studied as a potential predictor of ECT response [31,32].

- Quantitative electroencephalograms demonstrated increased slow (delta) wave activity in the prefrontal cortex after ECT, which was associated with clinical response [33]. In addition, pre-ECT quantitative electroencephalogram cordance was associated with ECT response [34].

- Genetic factors may be associated with response to ECT [35].

PRE-ECT EVALUATION

The goals of the pre-ECT evaluation are to:

- Determine whether ECT is indicated (table 1). (See "Unipolar major depression in adults: Indications for and efficacy of electroconvulsive therapy (ECT)".)

- Establish baseline psychiatric and cognitive status to serve as reference points for evaluating response and cognitive effects. (See 'Clinical monitoring' below.)

- Identify and treat any general medical conditions that might be associated with an increased risk of adverse events from ECT (table 2). (See 'Patients with comorbid general medical illness' below.)

- Initiate and continue the informed consent process. (See 'Informed consent' below.)

A complete medical history should be taken. Medical consultation prior to ECT is indicated for most patients 40 years or older, and is frequently obtained in younger patients as well. Particular attention should be paid to any history of cardiopulmonary and central nervous system disease and prior surgeries (with inquiry about type of anesthesia and any complications). (See "Medical evaluation for electroconvulsive therapy" and "Technique for performing electroconvulsive therapy (ECT) in adults", section on 'Electrode placement'.)

Appropriate physical examination (including dental evaluation) and laboratory evaluation, guided by the relevant history, should be performed. The patient's handedness and any dental problems should be noted. Specific laboratory tests are not indicated for patients without known medical comorbidities. (See "Preoperative medical evaluation of the healthy adult patient".)

PATIENTS WITH COMORBID GENERAL MEDICAL ILLNESS

Inducing a seizure causes transient increases in blood pressure, pulse, and intracranial pressure, which can have deleterious effects. Organ systems of most concern are [36]:

- Cardiovascular
- Pulmonary
- Central nervous system

Seizures increase cardiac workload and oxygen demand. Thus, it is important to try to maximize the patient's cardiovascular status before ECT. Additionally, medication to blunt increases in heart rate and blood pressure is indicated prior to ECT and during the procedure in patients for whom these increases would be detrimental.

A separate topic discusses strategies to reduce the risk of cardiac complications and the use of ECT in patients with coexisting cardiac disease (including hypertension, coronary heart disease, heart failure, and valvular disease), patients with pacemakers or implantable defibrillators, and patients who are anticoagulated, as well as patients with coexisting pulmonary disease, diabetes, or neurologic disease. (See "Medical evaluation for electroconvulsive therapy".)

PREGNANCY

ECT is sometimes indicated for pregnant patients, and the procedure can safely be used for these patients.

- (See "Unipolar major depression in adults: Indications for and efficacy of electroconvulsive therapy (ECT)".)
- (See "Bipolar disorder in pregnant women: Screening, diagnosis, and choosing treatment for mania and hypomania".)
- (See "Teratogenicity, pregnancy complications, and postnatal risks of antipsychotics, benzodiazepines, lithium, and electroconvulsive therapy", section on 'Electroconvulsive therapy'.)
- (See "Technique for performing electroconvulsive therapy (ECT) in adults", section on 'Pregnancy'.)

CONCURRENT MEDICATIONS

The patient's current medications, including prescription, over-the-counter, and complementary or alternative drugs should be reviewed prior to ECT. The ECT psychiatrist, anesthesiologist, or medical consultant should decide which to continue and which to taper and discontinue.

Psychotropic drugs — Many psychotropic medications may be continued during a course of ECT for their synergistic effect without compromising safety, including antidepressants, antipsychotics, and lithium [37]. Morning doses on the day of ECT are given after the patient has recovered from that day's procedure. Anticonvulsants and benzodiazepines often interfere with ECT (eg, decrease seizure duration) and may need to be tapered and discontinued [38].

Antidepressants can improve the efficacy of ECT.

- A review of 10 systematic reviews, 7 meta-analyses, and 3 practice guidelines found that for depressed patients, a tricyclic may improve the antidepressant effect of ECT [39]. As an example, a randomized trial assigned depressed patients (n = 319) receiving an acute course of ECT to concomitant nortriptyline, venlafaxine, or placebo. Remission occurred in more patients who received nortriptyline or venlafaxine compared with placebo (63 and 60 versus 49 percent) [40], with a statistically significant difference between nortriptyline and placebo.

- A subsequent systematic review of randomized trials in patients with unipolar major depression found ECT plus adjuvant antidepressants were statistically superior to ECT plus placebo or active placebo (eg, atropine) [41]:

  - Tricyclics – A meta-analysis of six trials (n = 540) found a small to moderate clinical benefit with adjunctive amitriptyline, imipramine, or nortriptyline.

  - Selective serotonin reuptake inhibitors/serotonin-norepinephrine reuptake inhibitors – A meta-analysis of two trials (n = 261) found a small to moderate clinical benefit with adjunctive paroxetine or venlafaxine.

  - Monoamine oxidase inhibitors – Although a meta-analysis of three trials (n = 226) found that adjunctive phenelzine or tranylcypromine was not statistically superior to placebo, the clinical effect of active drug was comparable to that observed in the other two meta-analyses.

Antidepressants are generally safe to use with ECT and do not affect its tolerability. As an example, a randomized trial of depressed patients receiving an acute course of ECT with concomitant nortriptyline, venlafaxine, or placebo found the number of adverse events did not differ significantly between the three groups [40]. Other types of antidepressants are also well tolerated during ECT, including selective serotonin reuptake inhibitors, tetracyclics, and monoamine oxidase inhibitors [42,43].

Antipsychotic medications are often continued during ECT and may provide a synergistic antipsychotic effect, especially second-generation antipsychotics [44]. In addition, antipsychotics generally do not appear to affect the tolerability of ECT. Second-generation antipsychotics and high potency first-generation antipsychotics are thought to be safe. A review found that early reports warned of hypotension and cardiac complications from combining chlorpromazine and ECT, but later reports did not substantiate these problems [45]. Nonetheless, combining of low potency, first-generation antipsychotics with ECT should be done cautiously.

Lithium has the potential to increase the adverse cognitive effects of ECT and to prolong the effects of succinylcholine (which is typically used during ECT to reduce tonic-clonic movements), but these problems are rare [46]. A review of several case series found that concomitant lithium and ECT is safe [46]. We suggest reducing lithium levels below the full therapeutic range at the time of each ECT treatment by withholding one or two doses of lithium prior to each treatment.

Benzodiazepines should be tapered and discontinued whenever possible during ECT because of their anticonvulsant properties, which can increase seizure threshold, shorten seizure duration,

and decrease the intensity of the ECT seizure [47]. These effects may decrease the clinical efficacy of ECT.

However, for patients who need to continue their benzodiazepine during ECT, it is reasonable to switch from drugs with long half-lives to one with a short half-life, and withhold the evening dose before each ECT treatment. In addition, small doses of benzodiazepines may not be a problem. Patients who are very anxious about the ECT procedure itself may take a benzodiazepine (eg, 1 mg of lorazepam sublingual) 30 to 60 minutes before the treatment.

Another reasonable alternative for patients who continue their benzodiazepine is to use intravenous flumazenil, which reverses the anticonvulsant effect of benzodiazepines and is given one to three minutes before anesthetic induction [48]. The typical dose of flumazenil is 0.4 to 0.5 mg, but doses up to 1.0 mg may be necessary for patients who have been chronically taking high doses of benzodiazepines. If patients receiving flumazenil manifest symptoms of benzodiazepine withdrawal within one hour post-ECT, subsequent ECT treatments should be followed by administration of intravenous midazolam (eg, 1 mg).

Anticonvulsants may theoretically interfere with the efficacy of ECT, similar to benzodiazepines, although the evidence for this is sparse [49]. Most patients on anticonvulsants for mood stabilization can be successfully treated by withholding the prior evening or night's dose, without the need to discontinue the medication [50]. However, if seizures are difficult to elicit or too short, we reduce the dose and/or discontinue the medication. The duration of an adequate ECT seizure is discussed separately. (See "Technique for performing electroconvulsive therapy (ECT) in adults", section on 'Seizure duration'.)

Patients with epilepsy should continue their anticonvulsants during a course of ECT. Clinicians should reduce the dose if ECT seizures are difficult to elicit or too short, or withhold a dose the evening or night before the ECT treatment.

Other drugs

- Cardiovascular medications – Patients should receive their routine antihypertensive (other than diuretics) and antianginal medications with a small sip of water approximately two hours before ECT. Diuretics should not be given because it is better to minimize the amount of urine in the bladder during the procedure, to prevent urinary incontinence as a result of the seizure.

  Use of cardiovascular medications in patients receiving ECT is discussed separately. (See "Medical evaluation for electroconvulsive therapy".)

- Anticoagulants – The use of anticoagulant medication in patients receiving ECT is discussed separately. (See "Medical evaluation for electroconvulsive therapy", section on 'Anticoagulation'.)

- Hypoglycemics – The use of hypoglycemic medications in patients receiving ECT is discussed separately. (See "Medical evaluation for electroconvulsive therapy", section on 'Diabetes'.)

- Asthma medications – Glucocorticoids and beta-adrenergic agonists may be given before ECT, as needed, to prevent bronchoconstriction. Patients who routinely use a fast-acting inhaler should do so immediately before the treatment. Theophylline should be avoided or maintained at the lowest effective blood level because it has been associated with prolonged seizures and status epilepticus [51].

- Gastrointestinal medications – Patients with gastroesophageal reflux disease should receive their H2 blocker, proton pump inhibitor, or metoclopramide with a sip of water two hours before ECT to prevent reflux and possible aspiration. Sodium citrate (30 cc po) may be given immediately before treatment to neutralize any acid remaining in the stomach [42].

INFORMED CONSENT

The psychiatrist should ensure that the consent process conforms to all state and local laws and statutes, and hospital policies. Informed consent requires that the patient [42]:

- Receive adequate information about depression and ECT
- Is capable of understanding and acting reasonably on this information
- Is given the opportunity to consent in the absence of coercion

ECT consent forms are more detailed compared with those for most other medical procedures because ECT is closely scrutinized. The American Psychiatric Association Task Force report provides a sample template for ECT consent that has been widely adopted (table 3 and table 4) [42].

Informational material written for the layperson is typically provided to patients and family members as part of obtaining informed consent. In addition, patients and family members may watch video to augment the written material and discussion with the psychiatrist recommending ECT. (See 'Outside sources of patient education' below.)

The vast majority of ECT is administered with fully informed consent given by the patient for a series of treatment [2]. In circumstances when the patient is too ill (eg, catatonic or psychotic) and lacks capacity to provide consent, and the need for ECT is urgent or emergent, the psychiatrist should seek substitute consent by a court [52].

The patient should be cautioned not to make major personal or financial decisions during or immediately after the acute course of ECT. In addition, the patient should not drive until the cognitive effects of ECT have resolved. Patients who are receiving continuation or maintenance ECT should not drive until 24 hours after each treatment [53].

TREATMENT COURSE

Number of treatments — There is no standard number of treatments for an acute ECT course and no way to predict how many treatments a particular patient will need. Most patients remit with 6 to 12 treatments, but some require only three while others require 20 or more [42].

In a study of 184 patients with unipolar major depression, the mean number of treatments needed to reach remission was 7 (standard deviation 3) [54]. A second study of 230 patients with unipolar or bipolar major depression found that 90 percent or more of the remissions occurred with nine or fewer treatments, depending upon the type of electrode placement [55]. (See "Technique for performing electroconvulsive therapy (ECT) in adults", section on 'Electrode placement'.)

Acute ECT should last until the patient remits, reaches an improvement plateau, or develops limiting adverse effects. It is becoming more common to taper the frequency of ECT treatments, rather than abruptly stop a course.

Once remission is achieved, however, ongoing continuation and maintenance treatments are frequently indicated. (See 'Continuation and maintenance ECT' below.)

Frequency of treatment — The frequency of treatments varies by country and the clinical urgency. Standard practice in the United States is to give ECT three times per week on a Monday-Wednesday-Friday schedule. The routine in many other countries is twice a week, particularly for older adult patients. Urgently ill patients (eg, catatonic or severely malnourished) may be given daily bilateral ECT until some improvement is evident [53].

Meta-analyses of five randomized trials (169 patients with unipolar major depression) found that symptomatic improvement, remission, and number of ECT treatments were comparable for twice-weekly and thrice-weekly ECT; however, duration of treatment was significantly longer for

twice-weekly ECT (by nearly five days) [56]. The data were insufficient to analyze differences in cognitive impairment; nevertheless, some randomized trials indicate that cognitive impairment is greater with bilateral, brief pulse ECT administered three times/week compared with two times/week [57,58].

Clinical monitoring — Depressive symptoms and cognition should both be monitored, and we encourage clinicians to serially measure severity of symptoms with a standardized scale (measurement based care). Several depression rating scales are available; we suggest that clinicians use clinician-administered rating scales rather than patient self-report scales because the validity of self-report scales can be compromised by confusion and amnesia from ECT. The scale should be administered at baseline, weekly during the course of ECT, and when the ECT course is completed. The most commonly used clinician-administered scale is the Hamilton Rating Scale for Depression (table 5) [59]. A reasonable alternative is the Montgomery-Asberg Depression Rating Scale (figure 1A-C) [60]. Measurement based care for treating depression is discussed separately. (See "Using scales to monitor symptoms and treat depression (measurement based care)".)

Many scales are also available for monitoring cognition; the most common is the Mini-Mental State Examination [61]. It is easy to use and adequate for routine clinical use, although its sensitivity may be limited [62]. More comprehensive neuropsychologic testing may be indicated for selected patients (eg, those with known cognitive deficits at baseline). (See "The mental status examination in adults", section on 'Cognitive screening tests'.)

ADVERSE EFFECTS

The adverse effects of ECT include general medical and cognitive effects.

Adverse general medical effects — ECT is one of the safest procedures performed under general anesthesia. As an example, a retrospective study of administrative health care databases identified patients hospitalized for depression who were treated with ECT (n >5000) or were not (n >5000) [63]. Propensity scoring was used to match the two groups on more than 75 potential confounding factors observed at baseline, such as sociodemographic and clinical characteristics. The primary outcome was serious medical events, defined as a composite of hospitalization for general medical (nonpsychiatric) reasons and nonsuicide mortality, occurring within 30 days of starting ECT or the corresponding index date for patients not receiving ECT. Numerically fewer serious medical events occurred in patients treated with ECT than non-ECT patients (0.25 and 0.33/person-year). In additional analyses that examined medical

hospitalization and nonsuicide death separately, the incidence of each was numerically fewer in the ECT group.

Mortality — Death related to ECT is rare. A review of 10 systematic reviews, 7 meta-analyses, and 3 practice guidelines found that the mortality associated with ECT was comparable to the mortality associated with minor procedures involving general anesthesia [39]. The mortality rate of ECT is at most 2 to 4 deaths per 100,000 treatments [64,65], and some studies estimate that the rate is less than 2 deaths per 100,000 treatments [66,67]. As an example, a 2017 study pooled data from 15 studies and more than 750,000 ECT treatments, and found that the ECT-related mortality rate was 2 per 100,000 treatments [68]. In the nine studies published after 2001 (n >400,000 ECT treatments), the mortality rate was less than 1 per 100,000 treatments.

Mortality is mostly related to cardiopulmonary events. Patients with coronary heart disease may be at risk for cardiac ischemic events because the seizure increases cardiac workload and oxygen demand. The cardiovascular effects of ECT are discussed separately. (See "Medical evaluation for electroconvulsive therapy", section on 'Cardiovascular effects'.)

Other — Other adverse general medical effects include:

- Aspiration pneumonia – Risk is increased in patients who do not have an empty stomach [53].
- Fracture – Patients with severe osteoporosis are at increased risk of fracture, and extra care should be taken to ensure excellent muscle relaxation [69].
- Dental and tongue injuries – These may occur when the oral bite block is not fully protective [70]. Loose teeth are at risk for dislodgement during the procedure and possible aspiration, and should be stabilized or extracted before ECT.
- Headache – This is the most common, nonserious adverse medical effect of ECT [71-73]. Patients should be told to expect some headache after each treatment. It is typically managed with acetaminophen or ibuprofen given after the treatment. Prophylaxis with intravenous ketorolac 30 mg may be considered for patients with significant post-ECT headache [74].
- Nausea – Transient, postprocedure nausea is common and is the result of the anesthesia and airway manipulation, which may introduce air into the stomach. Prophylaxis with intravenous ondansetron 4 mg should be given to patients with significant post-ECT nausea.

- Other somatic symptoms – Patients sometimes complain of other somatic symptoms such as myalgias, which may be a result of the fasciculations induced by succinylcholine. Myalgias are common immediately after the first treatment and less common thereafter. Temporary jaw and neck discomfort may also occur after the first treatment, as a result of the jaw clench during the stimulus and/or fasciculations in these muscles [53]. However, one study assessed a group of depressed patients both pre-ECT and during an acute course of ECT, and found that the frequency of somatic symptoms other than headache did not increase [75]. The authors suggested that these other somatic symptoms may have been due to the depressive illness, the use of psychotropic medications, or withdrawal from such medications.

Concerns that ECT causes structural brain damage have been dispelled by multiple human and nonhuman primate studies [1,39].

Adverse cognitive effects — Concern about adverse effects on cognition, primarily memory, remains an impediment to prescribing ECT [76]. However, the risks of major depression and the potential benefits of ECT outweigh the potential memory loss from ECT.

The patient's pretreatment cognitive status appears to be associated with ECT induced memory loss. As an example, a study of patients (n = 50) who were treated with ECT found that memory loss was greater in patients with low cognitive reserve, based upon years of education and occupational attainment, than patients with high cognitive reserve [77].

In addition, the adverse cognitive effects of ECT are affected by electrode placement, stimulus type and dose, and anesthesia [78]. (See "Technique for performing electroconvulsive therapy (ECT) in adults".)

We monitor cognition during an acute course of ECT by administering the Mini-Mental State Examination (MMSE) at baseline prior to onset of ECT, and again after the last ECT treatment. In addition, we assess patients with the MMSE at any point if we are concerned about cognition. Given that the MMSE is not in the public domain, many clinicians use the Montreal Cognitive Assessment (MoCA) instead. The MoCA is accessible online and in multiple languages at www.mocatest.org. Details regarding the MMSE and MoCA are discussed separately. (See "Evaluation of cognitive impairment and dementia", section on 'Cognitive testing'.)

Incidence and course — Many patients report some adverse cognitive effects during and after a course of ECT:

- A 2003 review of seven observational studies (n >1100 patients treated with ECT) found that the proportion of patients who reported any memory loss ranged from approximately

50 to 80 percent [79]. Memory loss for at least six months beyond the end of ECT was reported by about 30 to 55 percent.

- A national registry study identified patients (n >1200; mean age 53 years) who were treated with brief-pulse ECT between 2011 and 2014 and found that subjective memory worsening was endorsed by approximately 25 percent (and subjective memory improvement in 10 percent) [80]. After adjusting for each of the study variables, the analyses showed that memory worsening was more likely to occur in:
  - Females than males (31 versus 18 percent)
  - Patients aged 18 to 39 years than patients 65 years or older (32 versus 22 percent)
  - Patients without subjective memory disturbance before ECT than patients with memory disturbance (39 versus 17 percent)
  - Patients concurrently treated with lithium than patients not treated with lithium (31 versus 25 percent)
  - Patients who did not remit, compared with patients who remitted (28 versus 22 percent)

  Factors not associated with memory worsening included the major depression diagnosis (unipolar or bipolar), presence of psychotic features or personality disorder, and the use of psychotropic medications other than lithium.

However, objective tests indicate that cognitive abnormalities caused by ECT are generally short lived. A meta-analysis of 84 observational studies evaluated neuropsychological function (24 cognitive tests, eg, MMSE and Word List learning) in 2981 depressed patients who were treated with ECT and assessed with standardized tests at baseline and after the course of ECT was completed; all post-ECT assessments were compared with baseline assessments. The primary findings were as follows [81]:

- Up to 3 days post-ECT, cognitive performance was impaired on most tests, and many of the effects of ECT were clinically moderate to large.
- 4 to 15 days post-ECT, impairment was observed on one test, whereas small to moderate improvement of cognition was observed on several tests.
- More than 15 days posttreatment, cognitive performance was improved on most tests; the effects were clinically small to moderate.

Reassuring data were also found in subsequent studies:

- In a prospective observational study of ECT-naïve patients with unipolar major depression (n = 20), neuropsychological tests assessed attention, verbal memory, working memory, and executive functioning (eg, planning) at baseline and one week after a course of ECT [82]. The tests demonstrated no adverse effects following ECT.

- A retrospective study examined routinely collected data from patients (n = 126) assessed prior to ECT and for up to six months afterwards [83]. The large majority of patients were treated for unipolar major depression; all patients received bilateral ECT twice weekly at a dose two times seizure threshold. Assessments included an objective test of visuospatial memory. Compared with baseline, visuospatial memory was worse one month and three months post-ECT, but was superior at the six-month assessment.

Impaired cognition due to depression often improves after a course of ECT:

- A review of ECT in older adult patients with impaired cognition due to depression ("pseudodementia") examined six studies that tested cognition before and after ECT in patients mostly 55 years or older [84]. Patients were assessed with a cognitive screening instrument, either the MMSE [61] or the Mattis Dementia Rating Scale [85]. Global cognitive functioning in patients with cognitive impairment improved in all studies.

- Subsequently, a prospective observational study of patients (n = 31) with a mean age of 46 years found that six weeks after a course of ECT, neuropsychological testing demonstrated that attention, visual learning, and processing speed were improved from baseline, and that other cognitive domains were not altered [86].

- Another prospective observational study enrolled patients who were hospitalized for unipolar major depression and treated with ECT (n = 22) or usual care (n = 24); neuropsychological testing showed that verbal memory was impaired in both patient groups, compared with healthy controls (n = 35) [87]. Improvement of verbal memory was greater in patients who received ECT than usual care.

Although randomized trials suggest that concurrent treatment with different medications may mitigate the adverse cognitive effects of ECT, the use of these medications is not part of standard treatment with ECT. Adjunctive medications that were superior to placebo in reducing cognitive impairment include exogenous triiodothyronine [88] and acetylcholinesterase inhibitors such as donepezil, galantamine, and rivastigmine [89]. However, the sample sizes in the trials were small and further replication is required.

It does not appear that ECT is associated with an increased risk of dementia. A national registry study identified patients with a first time diagnosis of a mood disorder (n >162,000), including patients who were treated with ECT (nearly 6000), and followed them for a median of five years [90]. After adjusting for the probability (propensity) to receive ECT, based upon potential confounding factors such as sex, educational level, and use of psychotropic medications, the analyses found that the subsequent risk of dementia was comparable for patients treated with ECT and those not treated with ECT, including patients aged 70 years or older at baseline. This finding is consistent with other evidence that suggests ECT improves (rather than injures) brain structure and function. (See 'Mechanism of action' above.)

Types of impairment — ECT causes three basic types of cognitive impairment [76]:

- Acute confusion
- Anterograde amnesia
- Retrograde amnesia

The acute confusional state is the result of both the seizure and the anesthesia. It typically resolves 10 to 30 minutes after the procedure. The confusional state may include postictal agitation, which typically can be managed with a single intravenous dose of midazolam (eg, 1 to 3 mg) [91] or propofol (eg, 0.5 mg/kg) [92].

Anterograde amnesia is the decreased ability to retain newly acquired information. It occurs during a course of ECT and typically resolves within two weeks after completing the course. We thus suggest that patients refrain from driving during an acute course of ECT and for at least 24 hours after a single ECT (during continuation and maintenance ECT), or from making important business or personal decisions for one to two weeks after completing an acute course of ECT [93].

Retrograde amnesia involves forgetting recent memories and is the most anxiety-producing cognitive effect of ECT. The memories are for events that occur during the course of ECT and a period of weeks to a few months prior to that; however, some patients report retrograde amnesia that extends back for years [94]. The deficits are greatest and most persistent for knowledge about public or world events (impersonal memory) compared with knowledge about the self (personal memory) [95]. Bilateral ECT causes more retrograde amnesia than right unilateral [78,94]. Retrograde amnesia recovers more slowly than anterograde amnesia [95].

Some of the lost memories of events prior to the course of ECT may be expected to return, while others may not. A systematic review found four studies in which the proportion of patients who reported persistent or permanent memory loss ranged from 29 to 55 percent [79]. The existence and etiology of more severe memory impairments than typically experienced is

controversial [78,94,96-98]. Regardless, the typical ECT patient is profoundly depressed and accepts some degree of memory loss as a reasonable tradeoff for resolution of the depressive episode.

CONTINUATION AND MAINTENANCE ECT

Continuation ECT is the practice of providing a single ECT treatment, at an interval of one to eight weeks, during the first six months after remission. The purpose is to prevent relapse of the mood or psychotic episode that prompted the acute course of ECT. Maintenance ECT refers to ECT given beyond continuation ECT, to patients who have recovered from the acute mood or psychotic episode but require treatment to prevent recurrence of a new episode. Continuation and maintenance ECT are usually outpatient procedures.

Mood and psychotic disorders are chronically recurring illnesses. Therefore, continuation and maintenance treatment (with pharmacotherapy, ECT, psychotherapy, or a combination) are required for most patients who recover from an initial episode. As an example, in a prospective, observational study of 154 patients with major depression who remitted after successful treatment with ECT in community hospitals, 64 percent relapsed during 24 weeks of follow-up [99].

We suggest continuation and maintenance ECT for depressed patients who have successfully completed a course of acute ECT and:

- Relapse without continuation and maintenance treatment (either pharmacotherapy or ECT), and require a second a course of acute ECT
- Fail one or more courses of continuation and maintenance pharmacotherapy
- Prefer continuation and maintenance ECT

The electrode placement used for continuation and maintenance ECT should be the same as that used for acute ECT. (See "Technique for performing electroconvulsive therapy (ECT) in adults", section on 'Electrode placement'.)

Pharmacotherapy is often combined with continuation and maintenance ECT, with improved outcomes [100,101].

The schedule for continuation and maintenance ECT generally starts with weekly treatments for several weeks and gradually tapers to the maximal interval that allows the patient to remain euthymic. Following an acute course of ECT three times per week, we suggest treatments be

administered weekly for one to three weeks, followed by gradually increasing the interval between treatments (eg, biweekly for four weeks and then monthly for two months) [54]. Some patients continue to taper down to one treatment every three months, depending upon their clinical status and preference. There is no single continuation/maintenance ECT schedule that is best for all patients; the schedule should be flexible and adjusted to the needs of the patient, taking into account the patient's history of relapse and the severity of prior depressive episodes. As an example, patients who suffer subsyndromal symptoms short of a full recurrence may temporarily require more frequent treatments. Patients who benefit from maintenance ECT can receive it indefinitely. Use of self-report and clinician administered rating scales to monitor the patient's clinical status is discussed separately. (See "Using scales to monitor symptoms and treat depression (measurement based care)".)

Efficacy — Following acute ECT, continuation and maintenance treatment with ECT plus pharmacotherapy is more effective than pharmacotherapy alone. Evidence supporting the use of ECT plus medication includes the following [100]:

- In one study, geriatric patients with unipolar major depression were acutely treated with ECT plus venlafaxine; those who remitted (n = 120) were randomly assigned to six months of open-label continuation treatment with ECT plus pharmacotherapy (continuation venlafaxine plus lithium) or pharmacotherapy alone [102]. Continuation ECT consisted of four weekly treatments following randomization and subsequent treatments as warranted by symptoms of recurrence. Venlafaxine was continued at the same dose achieved during acute treatment (target dose was 225 mg/day), and lithium was prescribed to achieve a serum concentration of 0.4 to 0.6 mEq/L (0.4 to 0.6 mmol/L). Depression rating scale scores completed by masked (blind) raters were lower with ECT plus pharmacotherapy than pharmacotherapy alone; the median time to relapse for the ECT plus pharmacotherapy group and the pharmacotherapy alone group was eight and six weeks. In addition, global cognitive functioning at baseline and post-ECT was comparable.

- A one-year, open-label, randomized trial compared maintenance ECT (weekly for six weeks and then every two weeks) plus pharmacotherapy (antidepressants in nearly all patients; lithium and antipsychotics were used in varying numbers of patients) with pharmacotherapy alone in 56 patients who responded to an acute course of ECT for unipolar or bipolar major depression [101]. Relapse occurred in fewer patients who received combination treatment than pharmacotherapy alone (32 versus 61 percent).

Continuation ECT alone appears to be comparable to continuation pharmacotherapy alone. A six-month, open-label randomized trial compared continuation treatment with either ECT or pharmacotherapy in 184 patients who remitted from unipolar major depression after an acute

course of bilateral ECT [54]. Patients were assigned to a fixed schedule of continuation bilateral ECT or combination pharmacotherapy with lithium plus nortriptyline. In both groups, 46 percent of patients remained euthymic. In addition, the two continuation treatments did not differ in memory effects [103].

Cognitive effects of continuation and maintenance ECT are generally minor or nonexistent:

- A controlled trial randomly assigned depressed patients who remitted with an acute course of ECT to continuation ECT or continuation pharmacotherapy and evaluated them with an extensive battery of memory tests at baseline and after 24 weeks of treatment [54,103]. Neurocognitive performance was comparable for the two groups.
- A randomized trial compared maintenance ECT plus pharmacotherapy with pharmacotherapy alone in patients who remitted from major depression after an acute course of ECT [101]. Cognitive functioning and subjective memory were comparable in the two groups.
- An observational study evaluated 20 patients (multiple diagnoses) receiving maintenance ECT with a comprehensive neuropsychologic battery and retested them one year later [104]. There were no significant differences in test scores over time. In addition, test scores for the patients receiving maintenance ECT did not differ significantly from a control group of patients not receiving ECT.

STIGMA, ACCESS, AND TRAINING

Stigma — Stigma remains the biggest impediment to the appropriate use of ECT. The level of knowledge about modern ECT among patients and medical practitioners is low [105]. The portrayal of ECT in the media as a crude and coercive treatment remains problematic, frightening off patients who might benefit from it. Improved education about ECT among health care professionals will lead to more favorable perceptions about its use [106]. In addition, allowing family members to watch administration of ECT in the treatment suite can be reassuring and help diminish stigma [107,108].

Access — Access to ECT in the United States is variable and very limited in some rural areas. It is rarely available to patients in state psychiatric hospitals. Patients with no or limited insurance coverage may be unable to pay for ECT, leading to a situation in which ECT is preferentially available to more affluent patients with better insurance [9].

Training — ECT is a standard part of training in general psychiatry residency programs in the United States, and many programs offer in-depth hands-on training. Guidelines for curriculum are suggested by the American Psychiatric Association [42]. Several post-residency hands-on training courses are offered throughout the United States. These may be identified by contacting the American Psychiatric Association at 888-357-7924.

Individual United States hospitals issue privileges to psychiatrists to perform ECT, but there is no board certification for ECT in the United States [1].

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 the website.

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, 866-615-6464, or online at the website. The website also provides information about depression in language intended for the lay public.

SUMMARY

- **Indications for electroconvulsive therapy (ECT)** – Indications for ECT include severe unipolar depression, bipolar disorder, schizophrenia, schizoaffective disorder, catatonia, and neuroleptic malignant syndrome. (See "Unipolar major depression in adults: Indications for and efficacy of electroconvulsive therapy (ECT)" and "Bipolar disorder in adults: Indications for and efficacy of electroconvulsive therapy" and "Evaluation and management of treatment-resistant schizophrenia" and "Catatonia: Treatment and prognosis" and "Neuroleptic malignant syndrome", section on 'Electroconvulsive therapy'.)

- **Mechanism of action** – Although the exact mechanism of action of ECT is unknown, accumulating data suggest that ECT reverses many of the deleterious atrophic brain changes induced by severe central nervous system illnesses. (See 'Mechanism of action' above.)

- **Pre-ECT evaluation** – The pre-ECT evaluation should include a complete medical history emphasizing cardiopulmonary disease and prior surgeries. Appropriate physical examination and laboratory evaluation, guided by the relevant history, should be performed. Medical consultation prior to ECT is indicated for most patients 40 years or older and is frequently obtained in younger patients as well. (See 'Pre-ECT evaluation' above.)

- **Patients with comorbid general medical illness** – Inducing a seizure causes transient increases in blood pressure, pulse, and intracranial pressure, which can have deleterious effects. Organ systems of most concern are the cardiovascular, pulmonary, and central nervous systems. Medication to blunt increases in heart rate and blood pressure is indicated prior to ECT and during the procedure in patients for whom these increases would be detrimental. (See 'Patients with comorbid general medical illness' above and "Medical evaluation for electroconvulsive therapy".)

- **Concurrent medications**

- **Psychotropics** – Many psychotropic medications may be continued during a course of ECT for their synergistic effect without compromising safety, including lithium at subtherapeutic serum levels, antidepressants, and antipsychotics. Morning doses on the day of ECT are given after the patient has recovered from that day's procedure. Anticonvulsants and benzodiazepines often interfere with ECT and may need to be tapered and discontinued. (See 'Psychotropic drugs' above.)

- **Other drugs** – Most cardiac, antihypertensive, and anti-reflux medications should be taken with a small sip of water approximately two hours before each ECT treatment. (See 'Other drugs' above.)

- **Informed consent** – The consent process should conform to all state and local laws and statutes, and hospital policies. Informed consent requires that patients receive adequate information about their illness and ECT, are capable of understanding and acting reasonably on this information, and are given the opportunity to consent in the absence of coercion. (See 'Informed consent' above.)

- **Number of treatments** – A typical course of ECT consists of 6 to 12 treatments, individualized for each patient. Treatment usually continues until remission of symptoms. (See 'Number of treatments' above.)

- **Frequency of treatment** – Standard practice in the United States is to give ECT three times per week. The routine in many other countries is twice a week. (See 'Frequency of treatment' above.)

- **Continuation and maintenance ECT** – Continuation and maintenance ECT is the practice of providing a single, intermittent ECT treatments after remission to prevent recurrence of the mood or psychotic episode that prompted the acute course of ECT. (See 'Continuation and maintenance ECT' above.)

- **Adverse effects**

  - **General medical** – The mortality rate of ECT is at most 2 to 4 deaths per 100,000 treatments, making it one of the safest procedures performed under general anesthesia. Mortality is mostly related to cardiopulmonary events. Other adverse medical effects include aspiration pneumonia, fracture, dental and tongue injuries, headache, and nausea. (See 'Adverse general medical effects' above.)

  - **Cognitive** – Many patients experience some adverse cognitive effects during and after a course of ECT, including acute confusion, anterograde amnesia, and retrograde

amnesia. However, objective tests indicate that neuropsychological impairment caused by ECT is generally short lived, and impaired cognition due to depression typically improves after a course of ECT. In addition, ECT does not appear to be associated with an increased risk of dementia. We monitor cognition with a standardized instrument. (See 'Adverse cognitive effects' above.)

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