NCBI Bookshelf. A service of the National Library of Medicine, National Institutes of Health.

StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-.

Electroconvulsive Therapy

Authors

Irim Salik[1]; Raman Marwaha[2].

Affiliations

[1] Westchester MC/New York Med. College
[2] Case Western Reserve Un/MetroHealth MC

Last Update: September 19, 2022.

Continuing Education Activity

In a patient under intravenous sedation or general anesthesia, electroconvulsive therapy (ECT) uses an electric current to create a generalized cerebral seizure. Although it is primarily utilized to treat patients with severe depression, patients with schizophrenia, schizoaffective disorder, catatonia, neuroleptic malignant syndrome, and bipolar disorder may also benefit. This activity describes the indications, contraindications, and complications of ECT and highlights the role of the interprofessional team in the management of patients with mental health disorders.

Objectives:

- Determine the effects of electroconvulsive therapy.
- Identify the potential uses of electroconvulsive therapy.
- Assess misinformation associated with electroconvulsive therapy.
- Communicate electroconvulsive therapy, and review the role of the interprofessional team in managing patients who undergo ECT.

Access free multiple choice questions on this topic.

Introduction

In a patient under intravenous sedation or general anesthesia, electroconvulsive therapy (ECT) uses an electric current to create a generalized cerebral seizure. Although it is primarily utilized to treat patients with severe depression, patients with schizophrenia, schizoaffective disorder, catatonia, neuroleptic malignant syndrome, and bipolar disorder may also benefit. However, the practice has a stigma attached to it due to misinformation regarding procedural methodology.

Anatomy and Physiology

There are various pathophysiologic changes in brain regions found in patients with severe depression. These include reduced activity and volumetric reductions in the dorsal areas of the frontal lobes. Areas of the ventral and orbital frontal cortex have altered the processing of emotional stimuli.[1] In addition to the frontal lobe, functional alterations and volumetric reductions are apparent in the hippocampus, parahippocampal gyri, and amygdala.[2] The hypothalamic-pituitary-adrenal (HPA) axis becomes hypersensitive to stressors and exhibits chronically elevated levels of stress hormones and impaired feedback regulation.[3] The mesocorticolimbic dopamine system, as well as the HPA axis, are activated in patients with stress. Dopamine function is thought to be impaired in patients with

depression, causing impairment in essential functions, including concentration, motivation, and pleasure.[4] The anti-depressive effects of ECT reflect changes in the systems mentioned above.

Indications

ECT is indicated in patients with treatment-resistant depression or severe major depression that impairs activities of daily living. The definition of treatment-resistant depression is depression that is unresponsive to multiple antidepressant medication trials.[5] There are also suggestions for ECT as a treatment for suicidality, severe psychosis, food refusal secondary to depression, and catatonia.[6][7] Bipolar depressive and manic patients can also receive treatment with ECT.[8] ECT may have a safer profile than antidepressants or antipsychotics in debilitated, elderly, pregnant, and breastfeeding patients. Suicidal ideation is rapidly relieved by ECT, and the complete resolution was seen in 38% of patients after 1 week, 61% of patients after 2 weeks, and 81% of patients with the completion of ECT.[9] ECT is also recommended for patients who have responded favorably to ECT.

Contraindications

The resultant seizure from ECT can cause transient increases in blood pressure, myocardial oxygen consumption, heart rate, and intracranial pressure. Care is necessary for cardiovascular, pulmonary, or central nervous system-compromised patients. A pheochromocytoma and elevated intracranial pressure with mass effect are absolute contraindications to ECT. Relative contraindications include elevated intracranial pressure without mass effect, cardiovascular conduction defects, high-risk pregnancies, and aortic and cerebral aneurysms.[10]

Equipment

ECT treatment and recovery areas should include the standard American Society of Anesthesiologists (ASA) monitors. A stethoscope, blood pressure monitor, electrocardiography (EKG) monitor, pulse oximeter, suction apparatus, and an oxygen delivery system should be present. Anesthetic induction supplies, medication, and ventilatory and resuscitation equipment should be available. A nasal cannula or face mask to provide supplemental oxygen, bag valve mask, nerve stimulator to assess neuromuscular blockade, electromyograph (EMG), electroencephalography (EEG) leads, and multiple blood pressure cuffs should be available.

Personnel

ECT is administered by a team that typically includes an anesthesiologist, a psychiatrist, and a nurse.

Preparation

A complete history and physical examination may expose significant risk factors, including cardiac ischemia or arrhythmia, heart failure, or intracranial pathology.[11] History should also include the use of herbal medications such as Ginkgo biloba, ginseng, St John's wort, valerian, and kava, all of which may interfere with ECT. There is a risk of status epilepticus in patients on theophylline.[12] Short-acting intravenous beta-blockers may reduce ECT-related hypertension and tachycardia but may also shorten seizure duration and reduce ECT efficacy. Cardiac medications, including aspirin, statins, antihypertensive agents, antianginal medications, and clopidogrel, should be continued on the day of the procedure. Serum glucose levels require checking both preoperatively and in the recovery room, as ECT treatments can raise blood glucose levels. Although ECT appears safe in a patient with a defibrillator, detection mode should be turned off during the procedure, and equipment for external defibrillation should be available at the patient’s bedside. In pregnant patients, noninvasive fetal monitoring is recommended after 14 to 16 weeks, and a nonstress test with a tocometer after 24 weeks.

ECT utilizes general anesthesia. Anesthetic induction medications used include barbiturates such as thiopental and methohexital and nonbarbiturate agents such as propofol and etomidate. Seizure-induced by ECT should last longer

than 30 seconds. Methohexital is the most commonly used induction agent due to its quick onset, effectiveness, low cost, and minimal effect on seizure duration. Propofol and thiopental have been shown to reduce seizure duration. Etomidate has correlations with myoclonus and increased seizure duration.

Technique or Treatment

ECT is commonly performed in a dedicated suite, a post-anesthesia care unit, or an ambulatory surgery site, most frequently on an outpatient basis. Patients with severe debilitation, including substantial medical or psychiatric illness, may start on an inpatient basis and transition to an outpatient basis as needed. Patients should be appropriately nil per os (NPO) for the procedure, which includes no light meal for 6 hours, no full-fat meal for 8 hours, and no clear liquids for 2 hours before anesthesia.

Vital signs, including blood oxygen saturation, ECG, and EEG activity, are recorded continuously. EMG is recorded on the right foot to measure the motor component of seizure activity. A nerve stimulator monitors succinylcholine, a depolarizing muscle relaxant that reduces tonic-clonic contractions during the procedure. As an alternative to EMG, a blood pressure cuff is inflated on the patient's ankle to prevent succinylcholine from entering the foot, allowing a visual monitor of seizure activity with measurement of tonic-clonic contractions. Following intravenous induction, a bite block is placed to protect the patient's tongue and teeth. The beginning and termination of a cerebral seizure are monitored via EEG, recorded from right and left frontal and mastoid positions. Seizure induction is via 2 electrodes placed temporally or a right unilateral electrode, allowing electrical current to pass into the scalp.[13] A 2017 meta-analysis of numerous randomized trials of 792 patients specifically compared moderate bilateral ECT with high-dose right unilateral ECT, and remission was comparable.[14] Right unilateral ECT is utilized preferentially to minimize retrograde amnesia.

The ECT stimulus is either a brief pulse (0.5 to 2.0 milliseconds) or an ultra-brief pulse (less than 0.5 milliseconds) waveform. While the brief pulse is considered standard, the ultra-brief is more tolerable.[15] Electricity dose affects efficacy, the speed of response, and adverse cognitive effects. Seizure threshold is established via trial and error via incrementally higher current doses during the primary treatment session.[16][17] Following initial dose calculation, the dose at subsequent ECT sessions for bilateral ECT is 1.5 to 2 times the seizure threshold, and for right unilateral is 6 times the seizure threshold. During ECT treatment, the seizure threshold commonly increases as the patient develops tolerance.

As with any other procedure, the anesthesiologist's goal during ECT is to facilitate a safe and pain-free experience for the patient.[18] Preoxygenation of the patient via nasal cannula or face mask is followed by anesthetic induction and paralysis. Administration of an anticholinergic medication before ECT may prevent arrhythmias such as bradycardia or asystole and excessive oral secretions. To induce cerebral vasoconstriction via hypocarbia, the patient is often hyperventilated via a bag valve mask before delivery of the electrical stimulus to increase seizure intensity.[19]The gold standard for induction of anesthesia is methohexital, given at 0.75 to 1 mg/kg.[18] Methohexital is preferred to propofol because a meta-analysis of 2 randomized trials revealed that seizure duration was shorter with propofol.[20] Skeletal muscle relaxation during ECT is integral to minimizing a motor seizure and avoiding musculoskeletal injury. The depolarizing neuromuscular relaxant succinylcholine is used at 0.75 to 1 mg/kg with an elimination half-life of 41 seconds.[21][22] In cases where succinylcholine is contraindicated, including neuromuscular disease or injury, burn injury, pseudocholinesterase deficiency, or hyperkalemia, nondepolarizing neuromuscular relaxants are preferred.[5]

Once the patient is rendered unconscious, administration of a muscle relaxant follows, along with bag valve mask ventilation with 100 percent oxygen. A nerve stimulator is utilized to determine the adequacy of muscle relaxation and the clinical assessment of plantar reflexes and fasciculations in the calves and left foot. An inflated blood pressure cuff prevents muscle relaxants from entering the right foot. Muscle relaxants do not prevent masseter muscle contraction from the electrical pulse, requiring placement of a bite block for lingual and tooth protection.

Although most therapeutic ECT seizures last 15 to 70 seconds, EEG recording lasts about 25 percent longer than motor seizures.[23] Seizures lasting less than 15 seconds may not be clinically effective, while prolonged seizures may cause cognitive impairment. A missed or short seizure should have a follow-up with a short period of hyperventilation and restimulation with a higher electrical current. If a patient is experiencing a prolonged seizure greater than 2 minutes, an induction agent, such as propofol or methohexital, is either given at a half dose or a benzodiazepine to suppress seizure activity and avoid neurologic injury. In a patient with numerous missed seizures, anesthetic induction agents such as etomidate or ketamine may be useful as they exhibit fewer anticonvulsant effects as compared to methohexital. While caffeine had been previously administered to prolong seizures, it is no longer recommended due to its uncertain safety profile for this purpose.[24][12]

In pregnant patients receiving ECT, hyperventilation should be avoided as it can reduce placental blood flow, causing fetal hypoxia. Minimization of NPO time and adequate intravenous fluid hydration is essential to avoid dehydration and premature uterine contractions. Left lateral uterine displacement is essential in a female with a gestational age greater than 20 weeks to optimize maternal venous return and maintain optimal uterine blood flow. In pregnancies, greater than 24 weeks gestation considered viable, fetal heart rate and uterine activity should undergo continuous monitoring 30 minutes before and after each treatment by an obstetrician who can manage obstetric and neonatal emergencies. Because pregnant patients are at higher risk for aspiration pneumonitis due to their full stomach status, anticholinergic drugs that reduce lower esophageal sphincter tone should be avoided, as they can increase reflux.[25][26][27][28] In this case, a nonparticulate antacid such as sodium citrate is safer for aspiration prophylaxis.

Complications

Bilateral or bitemporal ECT causes more cognitive impairment than unilateral ECT, although this effect is transient. A meta-analysis of 1415 depressed patients treated with ECT revealed that global cognition, verbal memory, and autobiographical memory were worse with bilateral treatment 3 days after treatment.[29]

According to the American Psychiatric Association, patients receiving ECT are at higher risk if they show evidence of unstable or severe cardiovascular disease, a space-occupying intracranial lesion with evidence of elevated intracranial pressure, a history of an acute cerebral hemorrhage or stroke, an unstable vascular aneurysm, severe pulmonary disease, or qualify as American Society of Anesthesiologists (ASA) Class 4 or 5.

Physiologically, during the tonic phase of the seizure, a 15- to 20-second parasympathetic discharge occurs, which can lead to bradyarrhythmias, including premature atrial and ventricular contractions, atrioventricular block, and asystole. Patients with subconvulsive seizures are at higher risk for asystole.[30] Paradoxically, patients with heart block or underlying arrhythmias are less likely to develop asystole. The clonic phase of the seizure correlates with a catecholamine surge that causes tachycardia and hypertension, which lasts temporally with seizure duration.[31] Hypertension and tachycardia resolve within 10 to 20 minutes of the seizure, although some patients exhibit persistent hypertension that requires medical intervention.

Although patients with cardiac disease at baseline are at higher risk following treatment, ejection fraction can also decrease following ECT in healthy patients.[32] In a study of 53 adults undergoing ECT, 7 developed new global left ventricular (LV) systolic dysfunction, and 8 developed regional wall motion abnormalities.[33] Of these patients, no adverse outcomes were in patients with LV dysfunction. In a prospective cohort study of 100 subjects undergoing ECT, increased cardiac troponin levels were present in 8 patients, with only 2 having other evidence of myocardial ischemia or infarction.[34] Cerebral blood flow and intracranial pressure both increase with ECT therapy. Clinically, patients may exhibit confusion, delirium, disorientation, and memory loss. ECT is classified as a low-risk procedure by the AHA-ACC guidelines because it is well tolerated, and demonstrates only transient hemodynamic lability and low mortality rate.[35][36][37]

Clinical Significance

ECT is a relatively safe and low-risk procedure that is helpful in the treatment of depression, suicidality, severe psychosis, food refusal secondary to depression, and catatonia. It requires interprofessional care coordination among anesthesiologists, psychiatrists, and nurses. Most patients require several sessions to see a durable effect. The stigma associated with ECT is largely due to the lack of anesthesia with early treatments, resulting in significant injury and severe memory loss. The antidepressant effect is seen relatively quickly and may last up to a few years. Overall, the mortality rate is very low with ECT administered in a controlled setting, but it continues to cause mild memory loss in the long term. ECT is commonly utilized in pregnant patients and the elderly due to the avoidance of psychotropic medication side effects. Although its mechanism of action is multifactorial, ECT causes changes in cerebral blood flow and regional metabolism.

Enhancing Healthcare Team Outcomes

Today, ECT is now frequently used to treat a variety of mental health disorders besides depression. The procedure is relatively safe and does work. However, the delivery of ECT requires an interprofessional team that includes a nurse, anesthesiologist, psychiatrist, and a neurologist. The benefits of ECT are seen after several sessions, and the results are durable. The key is to educate the patient and family about ECT because the procedure has been associated with many false and illogical beliefs. The antidepressant effect is seen relatively quickly and may last up to a few years. Overall, the mortality rate is very low with ECT administered in a controlled setting, but it continues to cause mild memory loss in the long term. ECT is commonly utilized in pregnant patients and the elderly due to the avoidance of psychotropic medication side effects. Although its mechanism of action is multifactorial, ECT causes changes in cerebral blood flow and regional metabolism. Most patients who undergo ECT have a beneficial response without any adverse sequelae.[38]

Review Questions

- Access free multiple choice questions on this topic.
- Comment on this article.

References

1. Mayberg HS, Lewis PJ, Regenold W, Wagner HN. Paralimbic hypoperfusion in unipolar depression. J Nucl Med. 1994 Jun;35(6):929-34. [PubMed: 8195877]
2. Botteron KN, Raichle ME, Drevets WC, Heath AC, Todd RD. Volumetric reduction in left subgenual prefrontal cortex in early onset depression. Biol Psychiatry. 2002 Feb 15;51(4):342-4. [PubMed: 11958786]
3. de Kwaasteniet B, Ruhe E, Caan M, Rive M, Olabarriaga S, Groefsema M, Heesink L, van Wingen G, Denys D. Relation between structural and functional connectivity in major depressive disorder. Biol Psychiatry. 2013 Jul 01;74(1):40-7. [PubMed: 23399372]
4. Rigucci S, Serafini G, Pompili M, Kotzalidis GD, Tatarelli R. Anatomical and functional correlates in major depressive disorder: the contribution of neuroimaging studies. World J Biol Psychiatry. 2010 Mar;11(2 Pt 2):165-80. [PubMed: 19670087]
5. American Psychiatric Association. Task Force on Electroconvulsive Therapy. The Practice of ECT: Recommendations for Treatment, Training and Privileging. Convuls Ther. 1990 Jun;6(2):85-120. [PubMed: 11659302]
6. Husain MM, Rush AJ, Fink M, Knapp R, Petrides G, Rummans T, Biggs MM, O'Connor K, Rasmussen K, Litle M, Zhao W, Bernstein HJ, Smith G, Mueller M, McClintock SM, Bailine SH, Kellner CH. Speed of response and remission in major depressive disorder with acute electroconvulsive therapy (ECT): a Consortium for Research in ECT (CORE) report. J Clin Psychiatry. 2004 Apr;65(4):485-91. [PubMed: 15119910]
7. Kellner CH, Knapp R, Husain MM, Rasmussen K, Sampson S, Cullum M, McClintock SM, Tobias KG, Martino C, Mueller M, Bailine SH, Fink M, Petrides G. Bifrontal, bitemporal and right unilateral electrode placement in

ECT: randomised trial. Br J Psychiatry. 2010 Mar;196(3):226-34. [PMC free article: PMC2830057] [PubMed: 20194546]
8. Fink M. Indications for the use of ECT. Psychopharmacol Bull. 1994;30(3):269-75; discussion 276-80. [PubMed: 7878176]
9. Kellner CH, Fink M, Knapp R, Petrides G, Husain M, Rummans T, Mueller M, Bernstein H, Rasmussen K, O'connor K, Smith G, Rush AJ, Biggs M, McClintock S, Bailine S, Malur C. Relief of expressed suicidal intent by ECT: a consortium for research in ECT study. Am J Psychiatry. 2005 May;162(5):977-82. [PMC free article: PMC3684568] [PubMed: 15863801]
10. Taylor S. Electroconvulsive therapy: a review of history, patient selection, technique, and medication management. South Med J. 2007 May;100(5):494-8. [PubMed: 17534086]
11. Tess AV, Smetana GW. Medical evaluation of patients undergoing electroconvulsive therapy. N Engl J Med. 2009 Apr 02;360(14):1437-44. [PubMed: 19339723]
12. Devanand DP, Decina P, Sackeim HA, Prudic J. Status epilepticus following ECT in a patient receiving theophylline. J Clin Psychopharmacol. 1988 Apr;8(2):153. [PubMed: 3372714]
13. Bjølseth TM, Engedal K, Benth JŠ, Dybedal GS, Gaarden TL, Tanum L. Clinical efficacy of formula-based bifrontal versus right unilateral electroconvulsive therapy (ECT) in the treatment of major depression among elderly patients: a pragmatic, randomized, assessor-blinded, controlled trial. J Affect Disord. 2015 Apr 01;175:8-17. [PubMed: 25590761]
14. Kolshus E, Jelovac A, McLoughlin DM. Bitemporal v. high-dose right unilateral electroconvulsive therapy for depression: a systematic review and meta-analysis of randomized controlled trials. Psychol Med. 2017 Feb;47(3):518-530. [PubMed: 27780482]
15. Sienaert P, Spaans HP, Kellner CH. Pulse Width in Electroconvulsive Therapy: How Brief Is Brief? J ECT. 2018 Jun;34(2):73-74. [PubMed: 29489561]
16. Petrides G, Fink M. The "half-age" stimulation strategy for ECT dosing. Convuls Ther. 1996 Sep;12(3):138-46. [PubMed: 8872401]
17. Petrides G, Braga RJ, Fink M, Mueller M, Knapp R, Husain M, Rummans T, Bailine S, Malur C, O'Connor K, Kellner C., CORE (Consortium for Research in ECT) Group. Seizure threshold in a large sample: implications for stimulus dosing strategies in bilateral electroconvulsive therapy: a report from CORE. J ECT. 2009 Dec;25(4):232-7. [PMC free article: PMC2792571] [PubMed: 19972637]
18. Bryson EO, Aloysi AS, Farber KG, Kellner CH. Individualized Anesthetic Management for Patients Undergoing Electroconvulsive Therapy: A Review of Current Practice. Anesth Analg. 2017 Jun;124(6):1943-1956. [PubMed: 28277323]
19. Bergsholm P, Gran L, Bleie H. Seizure duration in unilateral electroconvulsive therapy. The effect of hypocapnia induced by hyperventilation and the effect of ventilation with oxygen. Acta Psychiatr Scand. 1984 Feb;69(2):121-8. [PubMed: 6422704]
20. Lihua P, Su M, Ke W, Ziemann-Gimmel P. Different regimens of intravenous sedatives or hypnotics for electroconvulsive therapy (ECT) in adult patients with depression. Cochrane Database Syst Rev. 2014 Apr 11;2014(4):CD009763. [PMC free article: PMC6464335] [PubMed: 24723301]
21. Bryson EO, Kellner CH, Li EH, Aloysi AS, Majeske M. Extreme variability in succinylcholine dose for muscle relaxation in electroconvulsive therapy. Australas Psychiatry. 2018 Aug;26(4):391-393. [PubMed: 29504412]
22. Torda TA, Graham GG, Warwick NR, Donohue P. Pharmacokinetics and pharmacodynamics of suxamethonium. Anaesth Intensive Care. 1997 Jun;25(3):272-8. [PubMed: 9209610]
23. Mayur PM, Gangadhar BN, Janakiramaiah N, Subbakrishna DK. Motor seizure monitoring during electroconvulsive therapy. Br J Psychiatry. 1999 Mar;174:270-2. [PubMed: 10448455]
24. Datto C, Rai AK, Ilivicky HJ, Caroff SN. Augmentation of seizure induction in electroconvulsive therapy: a clinical reappraisal. J ECT. 2002 Sep;18(3):118-25. [PubMed: 12394529]
25. Miller LJ. Use of electroconvulsive therapy during pregnancy. Hosp Community Psychiatry. 1994 May;45(5):444-50. [PubMed: 8045538]

26. Rabheru K. The use of electroconvulsive therapy in special patient populations. Can J Psychiatry. 2001 Oct;46(8):710-9. [PubMed: 11692973]
27. Yonkers KA, Wisner KL, Stowe Z, Leibenluft E, Cohen L, Miller L, Manber R, Viguera A, Suppes T, Altshuler L. Management of bipolar disorder during pregnancy and the postpartum period. Am J Psychiatry. 2004 Apr;161(4):608-20. [PubMed: 15056503]
28. Anderson EL, Reti IM. ECT in pregnancy: a review of the literature from 1941 to 2007. Psychosom Med. 2009 Feb;71(2):235-42. [PubMed: 19073751]
29. Semkovska M, Keane D, Babalola O, McLoughlin DM. Unilateral brief-pulse electroconvulsive therapy and cognition: effects of electrode placement, stimulus dosage and time. J Psychiatr Res. 2011 Jun;45(6):770-80. [PubMed: 21109254]
30. Kaufman KR. Asystole with electroconvulsive therapy. J Intern Med. 1994 Mar;235(3):275-7. [PubMed: 8120525]
31. Larson G, Swartz C, Abrams R. Duration of ECT-induced tachycardia as a measure of seizure length. Am J Psychiatry. 1984 Oct;141(10):1269-71. [PubMed: 6486265]
32. Fuenmayor AJ, el Fakih Y, Moreno J, Fuenmayor AM. Effects of electroconvulsive therapy on cardiac function in patients without heart disease. Cardiology. 1997 May-Jun;88(3):254-7. [PubMed: 9129846]
33. McCully RB, Karon BL, Rummans TA, Black JL, Andreen KM, Oh JK, Seward JB, Tajik AJ. Frequency of left ventricular dysfunction after electroconvulsive therapy. Am J Cardiol. 2003 May 01;91(9):1147-50. [PubMed: 12714169]
34. Duma A, Pal S, Johnston J, Helwani MA, Bhat A, Gill B, Rosenkvist J, Cartmill C, Brown F, Miller JP, Scott MG, Sanchez-Conde F, Jarvis M, Farber NB, Zorumski CF, Conway C, Nagele P. High-sensitivity Cardiac Troponin Elevation after Electroconvulsive Therapy: A Prospective, Observational Cohort Study. Anesthesiology. 2017 Apr;126(4):643-652. [PMC free article: PMC5350051] [PubMed: 28166110]
35. Zielinski RJ, Roose SP, Devanand DP, Woodring S, Sackeim HA. Cardiovascular complications of ECT in depressed patients with cardiac disease. Am J Psychiatry. 1993 Jun;150(6):904-9. [PubMed: 8494067]
36. Rice EH, Sombrotto LB, Markowitz JC, Leon AC. Cardiovascular morbidity in high-risk patients during ECT. Am J Psychiatry. 1994 Nov;151(11):1637-41. [PubMed: 7943453]
37. Abrams R. The mortality rate with ECT. Convuls Ther. 1997 Sep;13(3):125-7. [PubMed: 9342128]
38. Benson NM, Seiner SJ, Bolton P, Fitzmaurice G, Meisner RC, Pierce C, Busch AB. Acute Phase Treatment Outcomes of Electroconvulsive Therapy in Adolescents and Young Adults. J ECT. 2019 Sep;35(3):178-183. [PMC free article: PMC6581623] [PubMed: 30562200]

Disclosure: Irim Salik declares no relevant financial relationships with ineligible companies.

Disclosure: Raman Marwaha declares no relevant financial relationships with ineligible companies.

Bookshelf ID: NBK538266 PMID: 30855854