Electroconvulsive therapy (ECT)

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Introduction

Overview

Although seizures typically indicate a state of brain dysfunction, there are circumstances in which the biological effects of a seizure may exert therapeutic benefits. The standard technique for inducing controlled therapeutic seizures in humans is electroconvulsive therapy (ECT), a safe and remarkably effective treatment that involves the application of an electrical stimulus to the scalp of a patient under general anesthesia and muscle relaxation. ECT remains a cornerstone of treatment for severe mood disorders and certain other neuropsychiatric conditions, including those in patients with neurologic disorders. In addition, the neurobiological effects of ECT may have beneficial effects on a number of neurologic disorders, including Parkinson disease, epilepsy, and delirium. As with any procedure in medicine, the safety and efficacy of ECT depend critically on appropriate technique and proper patient selection and preparation.

Key points

- The primary diagnostic indications for which ECT is safe and effective include each of the major mood episodes (ie, major depressive, manic, mixed), schizophrenia and schizoaffective disorder, and catatonia (especially malignant variants, such as neuroleptic malignant syndrome).
- ECT is the single most effective, as well as the most rapidly effective, treatment for major depression.
- There are no absolute contraindications to ECT.
- The cognitive side effects of ECT are short-lived, do not cause major functional impairment, and must be distinguished from the symptoms of illness being treated.
- Misinformation and misunderstanding of these cognitive side effects remain the primary source of stigma and controversy that limit access to and prescription of ECT.
- Although we have learned a great deal about the neurobiology of ECT, its precise mechanism of action is unknown.

Historical note and terminology

The therapeutic use of seizures began in the early 1900s, when physicians observed that certain patients diagnosed as having schizophrenia appeared to improve after experiencing an unprovoked seizure. After experimenting with various pharmacologic means of inducing seizures (eg, metrazol, insulin), researchers discovered that electric current was the safest and most precise means of eliciting a controlled seizure. Enthusiasm for “electroshock therapy” spread across Europe, and the first use in humans took place in Italy in 1938. For several years, the procedure did not involve anesthesia, leaving some patients to experience intense anxiety and spinal fractures. Modern day ECT bears little resemblance to the electroshock therapy of the early 20th century. General anesthesia with muscle relaxation was introduced in the 1950s, and 2 decades later, brief pulse stimulation (pulse width 0.5-1.0 ms) replaced the far less efficient sine wave stimulation. These 2 major technical advances resulted in vastly improved safety, efficacy, and social acceptance. In today's world of medication-resistant psychiatric illness, ECT remains the treatment clinicians recommend for their most severely ill patients.

Clinical aspects

Description

ECT is administered by a specialized team of psychiatrists, anesthesiologists, and nurses. Prior to commencing the course of therapy, this team conducts a thorough general medical and neuropsychiatric examination of the patient in order to confirm the indications for ECT, to optimize the safety of the treatment by identifying and treating any general medical illnesses, to optimize the efficacy of the treatment by reviewing and potentially modifying the patient's list of medications, and to commence the informed consent process. Although some illnesses may increase the risks of ECT, there are no absolute contraindications to the procedure.

ECT is typically administered in a special treatment suite. The patient should have nothing to eat or drink for at least 8 hours prior to the procedure. Once baseline vital signs, pulse oximetry, and an electrocardiogram (ECG) have been obtained, the patient is administered a short-acting anesthetic agent (typically methohexital) followed by a short-acting neuromuscular blocking agent (typically succinylcholine). Throughout the procedure, the patient is ventilated with 100% oxygen by mask, and vital signs are continuously monitored. Once the patient is asleep and fully paralyzed, a specially designed bite block is inserted into the patient's mouth, and a brief electrical stimulus is then delivered across electrodes placed on the patient's properly prepared scalp. The electrical stimulus dosage and the location of the treating electrodes (right unilateral, bitemporal, bifrontal) are both important to the efficacy and side effects of the procedure. The electrical stimulus elicits a generalized seizure that typically lasts 25 to 90 seconds and is monitored continuously with electroencephalography (EEG). During the initial tonic motor phase of the convulsion, EEG activity is variable, consisting of low-voltage fast activity with polyspike rhythms. EEG activity rapidly evolves into the hypersynchronous polyspikes and waves that characterize the clonic motor phase. These regular patterns begin to slow and eventually disintegrate as the seizure ends, usually terminating abruptly in a “flat” EEG. Ventilatory support is maintained until the patient emerges from the anesthesia, and further recovery is provided in an environment with as little stimulation as possible. The entire procedure takes about 20 minutes, and patients are often able to have a snack within an hour of the procedure, after which they are discharged.

When used to treat acute illnesses, ECT is typically administered 3 times a week, on a Monday, Wednesday, Friday schedule, until a therapeutic benefit has been maximized (typically 6 to 12 treatments). Most patients receive the treatment on an outpatient basis, depending on their clinical status and the availability of appropriate logistical support. Response rates range from 65% to 90%, depending on a number of factors. After a successful acute course ECT, ECT may also be employed as continuation or maintenance therapy, the goal of which is to prevent recurrence. During a continuation or maintenance course of therapy, ECT is administered at decreasing frequencies (tapering from weekly to monthly or longer) and continued for at least 12 months.

Indications

The primary diagnostic indications for which ECT is safe and effective include each of the major mood episodes (ie, major depressive, manic, mixed), schizophrenia and schizoaffective disorder, and catatonia (especially malignant variants, such as neuroleptic malignant syndrome). Despite having the broadest spectrum of therapeutic activity of any modern biological treatment in neuropsychiatry, ECT is used mainly to treat patients suffering from depression (Lisanby 2007). ECT is indicated anytime a patient is gravely ill and in need of a rapid, definitive response (eg, acutely suicidal, melancholic with severe inanition) and is typically used when other treatments (typically medications) have failed or are poorly tolerated, when the patient has a history of a good response to ECT, or when the patient prefers ECT (American Psychiatric Association Committee on Electroconvulsive Therapy 2001).

Secondary indications for ECT include mood disorders due to general medical or neurologic conditions (eg, secondary mood disorders) as well as certain neurologic conditions themselves.

Contraindications

There are no absolute contraindications to ECT. It is safe and effective in children and adolescents, women who are pregnant, and the elderly. In persons with certain general medical conditions, ECT may be safer than pharmacotherapy.

ECT results in a marked activation of the autonomic nervous system, and the relative balance of parasympathetic and sympathetic nervous system activity determines the observed cardiovascular effects. These effects combine to produce a brief increase in cardiac workload and occasional transient arrhythmias, which are well tolerated by most patients. Patients with significant cardiac disease may require modifications in ECT technique to optimize the cardiovascular safety of the procedure.

The ECT seizure is also associated with a variety of transient and benign changes in cerebral physiology, including cerebral blood flow, cerebral blood volume, and cerebral metabolism. The brief increase in intracranial pressure is rarely of clinical consequence, but it is the reason for the well-known proscription against ECT in patients with a space-occupying intracranial mass. Transient disruption of blood-brain barrier permeability likely occurs during the seizure and may account for a transient increase in T1 relaxation times on brain magnetic resonance imaging (MRI).

Outcomes

ECT is the single most effective, as well as the most rapidly effective, treatment for major depression (Kellner et al 2012). Randomized, controlled data from the Consortium for Research in ECT (CORE) indicate that remission occurs in 70% to 90% of patients who receive ECT (Husain et al 2004). Remission rates are even higher among patients with psychotic depression and among the elderly (Petrides et al 2001; Rhebergen 2015). This success compares to remission rates of approximately 30% for pharmacotherapy and repetitive transcranial magnetic stimulation (Rush et al 2006; Ren et al 2014). ECT's efficacy does not depend on whether the depression is unipolar or bipolar. However, ECT using ultrabrief pulse width appears less efficacious than standard brief pulse ECT (Tor et al 2015).

Some patients report immediate improvement after a single ECT treatment, and approximately 60% of patients with major depression achieve full remission after 9 treatments (Husain et al 2004). Despite its robust results, ECT is not curative. Symptom recurrence occurs in a majority of patients if ECT is discontinued (Jelovac et al 2013). Randomized, controlled data indicate that maintenance therapy with ECT (utilizing the same technique as was effective during the acute course) and pharmacotherapy (lithium plus nortriptyline) are comparable, with both forms of therapy sustaining remission rates for about half of patients after 6 months (Kellner et al 2006). Randomized, controlled data also indicate that, among elderly persons, the combination of continuation ECT plus venlafaxine is superior to venlafaxine alone at 24 weeks after the completion of an acute course of ECT (Kellner et al 2016). Many patients with chronically recurring forms of severe illness receive lifelong maintenance ECT.

For patients suffering from mania, ECT remits symptoms approximately 80% of the time (Loo et al 2011). The treatment can be life-saving for patients suffering from manic delirium.

Remission rates are comparable in schizophrenia, although a higher number of treatments (eg, 12 to 15) may be required (American Psychiatric Association Committee on Electroconvulsive Therapy 2001). The combination of ECT with clozapine appears to be more effective than ECT alone (Petrides et al 2015).

Adverse effects

The safety of ECT compares favorably with that of any treatment requiring general anesthesia. ECT mortality in adults is reported as approximately less than 1 death per 10,000 patients (roughly 1 per 80,000 treatments), which is about the same as mortality from general anesthesia for minor surgery (American Psychiatric Association Committee on Electroconvulsive Therapy 2001). Such systemic side effects as headache, nausea, and myalgia are common during and shortly after the post-ECT recovery period. These symptoms are generally mild, transient, and quite responsive to symptomatic treatment (Andrade et al 2016).

Cognitive side effects are common after ECT and include a brief period of confusion and, rarely, frank delirium immediately on awakening from anesthesia. ECT is also associated with transient impairments in retrograde and anterograde memory, and roughly half of patients will subjectively report some degree of memory trouble (Rose et al 2003). Autobiographical memory is less affected (Lisanby et al 2000). The frequency and severity of these amnesic side effects vary with a number of patient factors (eg, age, preexisting cognitive impairment, general medical health) and ECT technical factors (eg, stimulus electrode placement, stimulus waveform). In general, the amnesic effects of ECT are short-lived (days to weeks in duration), do not cause major functional impairment, and must be distinguished from the amnesic effects of illness (eg, depressive pseudodementia) (American Psychiatric Association Committee on Electroconvulsive Therapy 2001; Semkovska and McLoughlin 2010). There is no evidence that ECT causes structural brain damage. Misinformation and misunderstanding of these cognitive side effects remain the primary source of stigma and controversy that limit access to ECT (Payne and Prudic 2009).

Special considerations

Mood disorders are common in patients with neurologic illness, and many of those patients will require treatment with ECT. Although there are no controlled data on the efficacy of ECT in these conditions, extensive clinical experience and

a primarily retrospective literature support such use. The administration of ECT in such patients may require modifications in the technique of the procedure in order to optimize safety and efficacy. The neurologist can play an important role in ensuring the appropriate and safe use of ECT in these individuals. ECT should be considered in depressed patients with Alzheimer disease, Parkinson disease, Huntington disease, stroke, epilepsy, multiple sclerosis, traumatic brain injury, and brain tumor.

In addition, the neurobiological effects of ECT may have beneficial effects on a number of neuropsychiatric disorders, most notably those discussed here.

Neuroleptic malignant syndrome (NMS). ECT has been used to successfully treat severe, refractory cases of NMS (Trollor and Sachdev 1999). Such patients should be off all dopamine-blocking drugs, however, and a non-depolarizing muscle relaxant (rather than succinylcholine) may be indicated in patients with prolonged immobility.

Parkinson disease. ECT may be a useful treatment for the motor disturbances of severe Parkinson disease when pharmacotherapy is unsuccessful or not tolerated, irrespective of the procedure's effect on the patient's mood. These data include a randomized controlled trial with sham ECT of 11 patients with the “on-off” syndrome (Andersen et al 1987). This salutary effect is not surprising given that ECT has been shown to enhance dopaminergic function (Kellner et al 2012). The beneficial effects of ECT on motor function may be maintained for months with maintenance ECT treatments.

Intractable seizures. Animal studies demonstrate that electroconvulsive seizures have anticonvulsant properties in that they block kindling and raise seizure threshold. This anticonvulsant effect whereby seizure threshold rises and seizure duration shortens during a course of ECT is also seen in humans (Kellner and Bernstein 1993). There are clinical reports of clinicians who have leveraged this anticonvulsant property of ECT to terminate status epilepticus in some patients, or decrease the frequency of seizures in others (Griesemer et al 1997). It has been suggested that a course of ECT might be considered in some patients with intractable epilepsy prior to undergoing brain surgery.

Delirium. ECT has been administered safely and effectively in patients with delirium associated with a broad range of etiologies (Nielsen et al 2014). ECT may be effective for delirium even when the underlying etiology has not been identified or corrected, and patients with delirium do not appear to be at any greater risk of cognitive side effects from ECT. On the basis of this large clinical experience, ECT is routinely used for the management of delirium in Scandinavia; ECT is probably underutilized as a treatment for delirium in the United States.

Dementia. Growing evidence suggests that ECT may be helpful for treating symptoms of agitation and aggression in patients with dementia. In such settings, ECT appears to be safe, well-tolerated, associated with a reduction in the PRN (as required) use of sedating medications, and, in some cases, rapidly effective (Acharya et al 2015).

Anti-NMDA Receptor Encephalitis. Accumulating case report data suggest that ECT may be helpful in treating the neuropsychiatric symptoms of anti-NMDA receptor encephalitis, particularly catatonia (Coffey and Cooper 2016). ECT may have a disease-modifying effect on this form of autoimmune encephalitis.

Clinical vignette

A 75-year-old married man with Parkinson disease developed a major depressive episode characterized by an overwhelmingly apprehensive mood that did not react to positive or pleasurable circumstances. The mood disturbance lasted for 6 months and was accompanied by anorexia with weight loss, insomnia, loss of pleasure, impaired concentration, guilty ruminations, somatic delusions of having a terminal illness, and thoughts of suicide via drug overdose. His psychiatrist prescribed an antidepressant medication that, although it had been effective in the past, provided little symptom relief and caused intolerable side effects. He was tapered off his antidepressant medication and commenced a course of bifrontal ECT. After receiving 6 ECT treatments on a Monday, Wednesday, Friday schedule, his depression symptoms were 50% improved, and he began experiencing confusion between treatments. The ECT schedule was modified to twice weekly treatments. After his ninth ECT treatment, his depression symptoms fully remitted, the confusion resolved, and he noticed modest improvement in his parkinsonism. He had difficulty recalling certain events that occurred during the course of ECT, but did not find this side effect distressing. Given his history of recurrent episodes of major depression, he elected to continue receiving ECT as part of a maintenance course of therapy. Once the schedule was tapered to one ECT treatment each month, his memory impairments resolved while the improvement in his motor symptoms was maintained. During the next several months of his

maintenance course of ECT, his depressive illness remained fully remitted, and he required no antidepressant medication.

Scientific basis

Although we have learned a great deal about the neurobiology of ECT, its precise mechanism of action is unknown. A number of well-documented observations have led to theories behind its powerful effects (Kellner et al 2012). ECT increases release and enhances transmission of dopamine, serotonin, and norepinephrine. It increases GABA transmission and has anticonvulsant properties. It acts on the hypothalamic-pituitary-adrenal axis to normalize levels of hormones, and it increases brain-derived neurotrophic factor. Functional imaging studies suggest that ECT induces neurogenesis and alters brain connectivity as well as metabolic activity in frontal and subcortical structures.

References cited

Acharya D, Harper DG, Achtyes ED, et al. Safety and utility of acute electroconvulsive therapy for agitation and aggression in dementia. Int J Geriatr Psychiatry 2015;30(3):265-73. PMID 24838521

American Psychiatric Association Committee on Electroconvulsive Therapy. The practice of electroconvulsive therapy: recommendations for treatment, training, and privileging: a task force report of the American Psychiatric Association (Report No. 0890422060). Washington (DC): American Psychiatric Association, 2001.**

Andersen K, Balldin J, Gottfries CG, et al. A double-blind evaluation of electroconvulsive therapy in Parkinson's disease with “on-off” phenomena. Acta Neurol Scand 1987;76(3):191-9. PMID 2446463

Andrade C, Arumugham SS, Thirthalli J. Adverse effects of electroconvulsive therapy. Psychiatr Clin North Am 2016;39(3):513-30. PMID 27514303

Coffey MJ, Cooper JJ. Electroconvulsive therapy in anti-N-methyl-D-aspartate receptor encephalitis. J ECT 2016;32(4):225-9. PMID 27295461

Griesemer DA, Keller CH, Beale MD, Smith GM. Electroconvulsive therapy for treatment of intractable seizures. Initial findings in two children. Neurology 1997;49(5):1389-92. PMID 9371927

Husain MM, Rush AJ, Fink M, et al. 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;65(4):48591. PMID 15119910

Jelovac A, Kolshus E, McLoughlin DM. Relapse following successful electroconvulsive therapy for major depression: a meta-analysis. Neuropsychopharmacology 2013;38(12):2467-74. PMID 23774532

Kellner CH, Bernstein HJ. ECT as a treatment for neurologic illness. In Coffey CE, editor. The clinical science of electroconvulsive therapy. Washington, DC: American Psychiatric Press, 1993:183-210.

Kellner CH, Greenberg RM, Murrough JW, Bryson EO, Briggs MC, Pasculli RM. ECT in treatment-resistant depression. Am J Psychiatry 2012;169(12):1238-44.** PMID 23212054

Kellner CH, Husain MM, Knapp RG, et al. A novel strategy for continuation ECT in geriatric depression: phase 2 of the PRIDE study. Am J Psychiatry 2016;173(11):1110-8. PMID 27418381

Kellner CH, Knapp RG, Petrides G, et al. Continuation electroconvulsive therapy vs pharmacotherapy for relapse prevention in major depression: a multisite study from the Consortium for Research in Electroconvulsive Therapy (CORE). Arch Gen Psychiatry 2006;63(12):1337-44. PMID 17146008

Lisanby SH. Electroconvulsive therapy for depression. N Engl J Med 2007;357(19):1939-45.** PMID 17989386

Lisanby SH, Maddox JH, Prudic J, Devanand DP, Sackeim HA. The effects of electroconvulsive therapy on memory of autobiographical and public events. Arch Gen Psychiatry 2000;57(6):58190. PMID 10839336

Loo C, Katalinic N, Mitchell PB, Greenberg B. Physical treatments for bipolar disorder: a review of electroconvulsive therapy, stereotactic surgery and other brain stimulation techniques. J Affect Disord 2011;132(1-2):1-13. PMID

20858566

Nielsen RM, Olsen KS, Lauritsen AO, Boesen HC. Electroconvulsive therapy as a treatment for protracted refractory delirium in the intensive care unit--five cases and a review. J Crit Care 2014;29(5):881.e1-6. PMID 24975569

Payne NA, Prudic J. Electroconvulsive therapy: part II: a biopsychosocial perspective. J Psychiatr Pract 2009;15(5):369-90.** PMID 19820554

Petrides G, Fink M, Husain MM, et al. ECT remission rates in psychotic versus nonpsychotic depressed patients: a report from CORE. J ECT 2001;17(4):244-53. PMID 11731725

Petrides G, Malur C, Braga RJ, et al. Electroconvulsive therapy augmentation in clozapine-resistant schizophrenia: a prospective, randomized study. Am J Psychiatry 2015;172(1):52-8. PMID 25157964

Ren J, Li H, Palaniyappan L, et al. Repetitive transcranial magnetic stimulation versus electroconvulsive therapy for major depression: a systematic review and meta-analysis. Prog Neuropsychopharmacol Biol Psychiatry 2014;51:181-9. PMID 24556538

Rhebergen D, Huisman A, Bouckaert F, et al. Older age is associated with rapid remission of depression after electroconvulsive therapy: a latent class growth analysis. Am J Geriatr Psychiatry 2015;23(3):274-82. PMID 24951182

Rose D, Fleischmann P, Wykes T, Leese M, Bindman J. Patients' perspectives on electroconvulsive therapy: systematic review. BMJ 2003;326(7403):1363. PMID 12816822

Rush AJ, Trivedi MH, Wisniewski SR, et al. Acute and longerterm outcomes in depressed outpatients requiring one or several treatment steps: a STAR*D report. Am J Psychiatry 2006;163(11):190517. PMID 17074942

Semkovska M, McLoughlin DM. Objective cognitive performance associated with electroconvulsive therapy for depression: a systematic review and meta-analysis. Biol Psychiatry 2010;68(6):568-77.** PMID 20673880

Tor PC, Bautovich A, Wang MJ, Martin D, Harvey SB, Loo C. A systematic review and meta-analysis of brief versus ultrabrief right unilateral electroconvulsive therapy for depression. J Clin Psychiatry 2015;76(9):e1092-8. PMID 26213985

Trollor JN, Sachdev PS. Electroconvulsive treatment of neuroleptic malignant syndrome: a review and report of cases. Aust N Z J Psychiatry 1999;33(5):650-9. PMID 10544988

**References especially recommended by the author or editor for general reading.

Other topics to consider

Affective disorders in neurologic disease
Catatonia
Neuroleptic malignant syndrome
Neurologic disorders presenting with behavioral signs and symptoms
Schizophrenia
Transcranial magnetic stimulation