REVIEW ARTICLE

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Electroconvulsive Therapy

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Electroconvulsive therapy (ECT) has been an essential treatment for severe mood and psychotic disorders for many decades, and its use is supported by evidence of efficacy and safety.[1-3] This brief review discusses current indications for ECT and recent advances in treatment. Over the past 15 years, new treatments — for example, vagus-nerve stimulation, transcranial magnetic stimulation, and intranasal administration of esketamine — have been approved for use in depression. Trials comparing new treatments directly with ECT have been inadequate,[2,3] and none of these approaches have been considered a replacement for ECT in severely depressed and certain psychotic patients.[3,4]

Untreated severe depressive and psychotic illnesses are associated with high rates of suicide and hospitalization, prolonged illness, and reductions in productivity and quality of life.[5] Studies have indicated that the use of ECT results in a decreased risk of suicide,[6] improved functional outcomes[7] and quality of life,[7,8] and decreased rates of rehospitalization.[9-11] In specific populations, ECT can rapidly ameliorate depressive, psychotic, and catatonic symptoms and can decrease suicidal drive. Trials of ECT for major depressive disorder in patients with treatment-resistant depression have shown pooled response rates of 60 to 80% and pooled remission rates of 50 to 60%.[2,7] High rates of response to ECT have been reported in patients with psychotic depression or catatonia. In a study involving patients with treatment-resistant schizophrenia, ECT efficacy rates ranged from 40 to 70%,[12] and in some Asian countries, the primary indication for ECT is schizophrenia.[7,9] For example, in an uncontrolled study of treatment-resistant schizophrenia, in which ECT was combined with clozapine, 50% of patients had at least a 40% reduction in symptoms after a course of ECT.[13] In patients with bipolar mania and those with mixed mood states (the concurrent presence of manic and depressive symptoms), ECT has led to symptom control within weeks.[14,15]

However, a study has shown that ECT has been underused.[1] Furthermore, ECT is less accessible to uninsured or underinsured patients and those receiving care in public hospitals than to other patient populations,[16] it has been underused in some large health care systems,[17] it has been limited to inpatient settings,[18] and it is less accessible to minority racial or ethnic groups than to other groups.[19,20] Stigma and lack of knowledge have constrained the use and acceptance of ECT.[1,21,22]

The contemporary practice of ECT involves induction of brief general anesthesia (typically lasting less than 10 minutes), pharmacologic muscle relaxation, and continuous monitoring of oxygen saturation, blood pressure, and heart rate and rhythm. An electrical charge is delivered to the brain through scalp electrodes, which results in a generalized seizure typically lasting for 20 to 60 seconds. Most patients receive between 6 and 12 treatments spaced over a period of 2 to 4 weeks as an initial course of treatment.

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CLINICAL INDICATIONS

In 2018, the Food and Drug Administration (FDA) reclassified ECT devices from class III (high risk) to class II (moderate risk, requiring special controls) for use

Table 1. Features of Initial and Maintenance Electroconvulsive Therapy (ECT).

Initial course
Consists of 6 to 12 treatments, on average; usually performed 2 or 3 times/wk for 2–4 wk
No set minimum or maximum number of treatments
Continued until patient has maximal sustained response or side effects limit further use
Performed in inpatient or outpatient setting, depending on illness severity or logistics
Continuation or maintenance treatment
Follows completion of an index course
Performed in outpatient setting
Gradually tapered, with increasing intervals between treatments
Goal is to deliver the fewest treatments needed to sustain a response
May continue months to years (no set maximum number of treatments)

in the treatment of severe major unipolar or bipolar depressive episodes or catatonia in persons 13 years of age or older, whose disorder is "treatment resistant or who require a rapid response due to the severity of their psychiatric or medical condition."[23] These stipulations are analogous to the "on label" uses of drugs. The FDA did not define the terms "severe" and "treatment resistant" and did not specify the circumstances under which a rapid response might be required. The FDA also did not rule on other uses of ECT in practice, leaving them to clinical judgment. ECT has been tested for other conditions, including bipolar disorder (manic or mixed states),[14,15] schizophrenia,[7,12,13] and Parkinson's disease, according to narrative reviews and meta-analyses.[24] There have been reports of uncontrolled, off-label use of ECT for the treatment of self-injurious behaviors due to autism,[25] behavioral and psychological symptoms of dementia,[26] and status epilepticus.[27] However, these reports are preliminary. The use of ECT in patients with these conditions cannot be endorsed at this time because the evidence base remains too small.

Most patients referred for ECT have debilitating, severe illness that has persisted despite multiple medications.[3,4,28] For depressive and schizophrenic disorders, health care economic studies suggest that ECT may be cost-effective after no more than two medication trials, as an alternative to allowing patients to remain impaired for months or years.[9,12,28] Similar health care economic findings have supported the use of ECT for depression complicating schizophrenia and for treatment-resistant schizophrenia either with or without clozapine.[12,13,29] When patients are ill with potentially life-threatening symptoms, including suicidality, psychosis, and other complications of illness (e.g., dehydration, malnutrition, catatonia, worsening of pain, deconditioning, and pressure sores) that require a rapid response, ECT may be recommended as a primary treatment without repeated medication trials.[30] A comprehensive informed-consent process is an integral part of the evaluation for ECT. Some patients lack the capacity to provide informed consent; in such cases, local statutes for establishing capacity are followed, which may include such measures as the court appointment of a surrogate decision maker. Analyses of statutes governing the administration of ECT show a correlation between restrictive policies and diminished use of, or access to, ECT.[31,32]

COURSE OF TREATMENT

ECT comprises two phases of care (Table 1). In the initial phase, the goal is a response or, ideally, remission. Patients are assessed clinically after each treatment for benefit and side effects, with subsequent periodic assessments based on commonly used mood and cognition rating scales. The initial phase continues until patients have a maximal sustained treatment response or side effects limit further use of the treatment.

After patients have had sufficient clinical improvement, ECT is generally not stopped abruptly, owing to the risk of relapse. In a randomized 24-week trial,[33] cessation of ECT and subsequent administration of placebo resulted in an estimated relapse rate of 84%, as compared with the use of a single antidepressant agent (nortriptyline) or a combination of an antidepressant (nortriptyline) and lithium, which resulted in relapse rates of 60% and 39%, respectively. In another randomized trial,[34] which compared continuation of ECT (10 treatments) with combination pharmacotherapy (a tricyclic antidepressant plus lithium) for 6 months after an initial course of ECT, relapse rates were similar in the two treatment groups; about 46% of patients in each group had a sustained remission.

Current practice for preventing a relapse is to taper ECT after an initial course and continue treatment with pharmacotherapy, maintenance ECT, or both, guided by the number and severity

of prior depressive episodes and the number of previous medication trials.[35] Combination pharmacotherapy after an initial course of ECT may include an antidepressant and lithium, although other mood stabilizers and other classes of medications have been used.[33,34] During the maintenance phase of ECT, a single outpatient ECT treatment is provided at increasing intervals, typically starting weekly, then monthly or longer, with the goal of delivering the fewest treatments needed to maintain remission. In the PRIDE (Prolonging Remission in Depressed Elderly) study, ECT once a week for 4 weeks after the initial course, with the combination of venlafaxine and lithium, as well as additional maintenance ECT as needed for recurrent symptoms,[36] was associated with a sustained response in more than 80% of patients over a period of 6 months. A similar strategy of tapering ECT for the treatment of schizophrenia or other conditions has not been well studied. In one systematic review, maintenance ECT was reportedly beneficial in treatment-resistant schizophrenia.[37] In a randomized trial, antidepressant monotherapy after ECT was not adequate for relapse prevention in patients with treatment-resistant depression.[33] Adjunctive cognitive behavioral therapy may aid in preventing relapse of depression in patients receiving maintenance ECT.[38] Although some of these strategies have reduced rates of relapse, the duration of benefit from ECT varies across patients, and relapse prevention remains an important clinical challenge.

TECHNIQUE OF ECT

Three configurations of electrode placement are used in the contemporary practice of ECT: bilateral (bitemporal), right unilateral, and bifrontal placement. All three are effective when used with an appropriate charge dose.[39] Bilateral electrode placement is the standard of care in many countries; it has high efficacy and may provide a faster onset of action than right unilateral placement but is associated with more cognitive side effects.[39,40] Right unilateral ECT, in which both electrodes are applied over the right hemisphere, has been as effective as bilateral ECT for many, but not all, patients in randomized trials.[39,40] The electrical stimulus and charge doses are manipulated to maximize the efficacy of right unilateral ECT while limiting its adverse cognitive effects as compared with bilateral ECT.[41-44] Patients who do not have a response to several treatments with right unilateral electrode placement may benefit from a switch to bilateral placement.[44,45] The efficacy and side-effect profile of bifrontal placement are similar to those of bilateral placement.[39] For urgent clinical situations, bilateral stimulation is often recommended because of its slightly more rapid response.[30,40,45]

Electrical stimulus dosing in ECT is based on research suggesting that the amount of charge delivered above the threshold for producing a seizure (the seizure threshold) affects treatment outcome, particularly with right unilateral electrode placement.[44] The seizure threshold is determined at the first treatment session by a stepwise method in which progressively higher charge stimuli are delivered until a generalized seizure is elicited, with both electroencephalographic and motor manifestations.[41,42] Individualized stimulus dosing at subsequent treatments, depending on electrode placement, is then delivered at a charge of 1.5 to 6 times the seizure threshold. The electrical stimulus, including pulse width, frequency, duration, and current amplitude, can be manipulated to increase the efficiency of seizure induction and propagation. An ultrabrief pulse width (<0.5 msec) has been shown to elicit a generalized seizure more efficiently than longer pulse widths at lower charge and is associated with fewer adverse cognitive effects.[42,43]

SAFETY

The estimated mortality with ECT is approximately 2.1 deaths per 100,000 treatments.[46-48] The most frequent complications are acute cardiopulmonary events, which have been estimated to occur in less than 1% of treatments.[46,47] In addition to the routine preprocedural evaluation by clinicians in primary care or internal medicine and anesthesiology, a cardiology consultation may be indicated for patients with risk factors for or a history of cardiac disease. Serious adverse events associated with ECT, which are rare, include cardiac arrhythmias with or without hemodynamic changes, respiratory distress, prolonged apnea, aspiration, prolonged paralysis, and prolonged seizures. Common but minor side effects include headache, jaw soreness, myalgias, postprocedure nausea and vomiting, and fatigue, all of which are self-limited or require only symptomatic treatment.

Table 2. Indications for ECT According to Treatment Guidelines.*

Indication	APA	CANMAT	RANZCP	WFSBP
Major depression	3rd or 4th step	1st or 2nd step	1st, 2nd, or 3rd step	3rd or 4th step
Bipolar disorder				
Depression	3rd or 4th step	1st or 2nd step	2nd or 3rd step	3rd or 4th step
Mania	3rd or 4th step	1st or 2nd step	2nd or 3rd step	4th or 5th step
Mixed state	4th or 5th step	Not stated	Not stated	Not stated
Schizophrenia	Treatment-resistant	Not included	3rd or 4th step	3rd or 4th step
Other conditions	Yes	Not included	Not included	If clinically indicated
Comments	Patient can choose to consider ECT at any point for any of the indications listed above	Best evidence guides treatment choice	None	None

* "Step" refers to the step in treatment. All four guidelines recommend a gradual tapering of ECT and consideration of maintenance ECT as a relapse-prevention strategy after an initial course of ECT. APA denotes American Psychiatric Association, CANMAT Canadian Network for Mood and Anxiety Treatments, RANZCP Royal Australian and New Zealand College of Psychiatrists, and WFSBP World Federation of Societies of Biological Psychiatry.

COGNITIVE EFFECTS

Patient, provider, and public concerns about cognitive impairment remain obstacles to the use of ECT.[1,21,49] The contemporary practice of ECT results in fewer cognitive side effects than treatments in the past. Studies have sought to characterize the type, extent, and course of cognitive deficits resulting from ECT and to identify techniques for preventing or mitigating these effects.[43,44,49-51] The main cognitive domains that have been measured in relation to ECT are anterograde and retrograde memory, attention, executive function, and processing speed.[49-51]

Cognitive effects vary among patients and ECT techniques.[43,44,50] Greater cognitive impairment has been associated with bilateral electrode placement, a larger number of treatments, and a higher stimulus charge,[40,41,50] and less impairment has been associated with right unilateral electrode placement, ultrabrief pulse widths, and shorter treatment courses.[40-43] It has not been possible to predict how an individual patient may be affected, but most patients have mild or moderate cognitive side effects, which usually resolve within days to weeks after the completion of the ECT course.[43,49-51] Anterograde amnesia typically resolves within 2 to 4 weeks. In contrast, retrograde amnesia — primarily gaps in autobiographical memory but not loss of semantic knowledge — develops gradually over a series of treatments and resolves more slowly, over a period of weeks to months after completion of the index course. For some patients, retrograde amnesia persists for more than a year.[50] The presence of ongoing depressive or other psychiatric symptoms may contribute to and correlate with persistent cognitive impairment that may be detected on neuropsychological testing.[49,50] In rare cases, an acute confusional state or delirium develops, requiring interruption or cessation of the treatment course. For many patients, including those undergoing maintenance ECT, as severe depression resolves, cognition in functions affected by depressive illness improves.[49,51,52] Neuropathological and neuroimaging studies have not shown evidence of structural brain damage after ECT.[53,54] In a Danish National Patient Registry cohort study involving 168,015 patients with depression, of whom 3.1% had at least one ECT treatment, over a median follow-up of 4.9 years, ECT was not associated with an increased risk of incident dementia.[55]

NEUROBIOLOGIC EFFECTS

The mechanism of action of ECT is not fully understood. Surrogates for the mechanism have been sought in studies of changes in brain structure and amine metabolism. In patients with depression, replicated imaging findings have shown that the gray-matter volume in the frontolimbic areas, particularly in the hippocampus and amygdala, is greater after a course of ECT

than before this treatment.[54,56,57] Diffusion tensor imaging studies have shown ECT-associated increases in the integrity of white-matter tracts in the frontal and temporal lobes.[54,56] Some studies have shown increases in monoamine neurotransmitters, normalization of the cortisol response to dexamethasone, and increased neurogenesis in the dentate gyrus, all of which putatively contribute to the mechanism of action. Large-scale studies using multiple types of analyses ("omics" studies, gene expression studies, and multimodal neuroimaging) are under way.[58,59]

STIGMA AND ECT

Sensationalist media portrayals, lack of knowledge among physicians about contemporary techniques of ECT, fear of electricity, and concern about memory effects have all contributed to the stigmatization of ECT.[1,21,22] Anti-ECT campaigns have long been part of an antipsychiatry movement, and Internet sources have promulgated misinformation about the procedure. Accurate, objective information is available from medical sources and leading academic organizations. Guidelines from major psychiatric associations[30,60-62] include ECT in treatment algorithms for severe and treatment-resistant mood and psychotic disorders (Table 2).

CONCLUSIONS

ECT is a valuable treatment for several severe psychiatric illnesses, particularly when a rapid response is critical and when other treatments have failed. Refinements in technique have reduced, but not eliminated, side effects. Research into the neurobiologic basis for the effects of ECT is ongoing, since the mechanism of action is not known. Stigma and lack of access to treatment have contributed to the underuse of ECT.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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