The Use of Electroconvulsive Therapy for Depression in the Elderly

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Electroconvulsive therapy (ECT) is an effective and relatively safe procedure for treating severe, treatment-refractory mood disorders in special populations, particularly in the elderly. The frail elderly are especially good candidates for ECT because they are often unresponsive to, or intolerant of, psychotropic medications. The central nervous, cardiovascular and respiratory systems require special attention during the course of ECT. With modern anesthesia techniques and careful medical management of each high-risk patient, most elderly can successfully complete a course of ECT. Obtaining informed consent for ECT from incapable patients is a challenging issue with few concrete guidelines.

Key words: electroconvulsive therapy, elderly, depression, special patient population.

Until the 1930s, little could be done to help those suffering from major mental disorders (MMDs) apart from custodial care and sedation. In 1934, Meduna first noted a reduction of psychotic symptoms with chemically-induced seizures and, a few years later, Cerletti and Bini introduced electroconvulsive therapy (ECT), which quickly became the main somatic treatment for MMDs. By the mid-1950s, the use of ECT declined dramatically due to the discovery of psychopharmacologic agents, which were hoped to replace ECT. Negative public image aided by popular movies and fear of inhumane and punitive treatment led to the stigma of ECT that still persists today. Over the past 60 years, ECT has evolved and proven to be an effective and potentially life-saving intervention for certain devastating illnesses, often when other treatments have little or no effect.

Indications for ECT

ECT is used most often in patients who have not responded to other treatments. However, it should not always be used as a "last resort", as this may prolong suffering, delay response or contribute to treatment resistance. Reasons to consider ECT include treatment resistance, medication intolerance, suicidality, inanition, or deterioration of the patient's condition requiring rapid, definitive response.[1] Both principal and other diagnostic groups for which ECT is considered are presented in Table 1. Other diagnostic groups for which ECT is used occasionally include other psychiatric disorders, medical disorders with severe secondary affective or psychotic symptoms, catatonic states, delirium, Parkinson's disease, neuroleptic malignant syndrome and intractable seizure disorder.

ECT Use in the Elderly

ECT is used most frequently in the elderly for treatment of medication-resistant or medication-intolerant depression, especially in late-onset depression. A course of ECT is effective in 70–80% of elderly depressed patients who have failed drug therapy,[2] and has a more reliable and rapid response than drug therapy among the elderly and adult population.[3,4] ECT is increasingly being used as first-line therapy for severely depressed patients who are refusing to eat or drink, are psychotic or are at high risk for suicide. Coexisting medical conditions that frequently occur in the elderly also may make ECT the treatment of choice for some patients due to its speed of action and safety profile. ECT should be considered a first-line treatment for elderly patients whose hepatic, renal or cardiac function prevents the use of pharmacotherapy for depression.[5]

In general, the short-term outcome of ECT treatment of depression in the elderly appears to be more favourable than in younger adults.[6,7] This may be due in part to the fact that the elderly often receive ECT earlier in the course of illness than younger adults due to medication intolerance, medical complications or psychotic depression, thereby reducing the duration of the index episode and medication resistance, both of which are positive predictors of response to ECT.[7] However, the elderly may not respond as well to unilateral ECT as younger patients, and more often require bilateral ECT[8] and a longer course of treatment to achieve remission.[9]

Older patients and those with medical illnesses are at greater risk for ECT-

Table 1
Diagnostic Groups Considered for ECT

Principal
Major depression
– unipolar or bipolar
Mania
Psychotic symptoms in:
– schizophrenia
– schizoaffective disorder
– schizophreniform disorder
Other
Psychiatric disorders
Medical disorders with severe secondary affective or psychotic symptoms
Catatonic states
Delirium
Parkinson's disease
Neuroleptic malignant syndrome
Intractable seizure disorder

related persistent confusion and memory deficits,[10] amnesia of autobiographic information, and anterograde and retrograde amnesia, particularly with bilateral ECT. Baseline pre-ECT cognitive scores and postictal disorientation have been shown to predict the degree of long-term retrograde amnesia in patients who are not neurologically impaired.[11] However, cognitive impairment associated with depression may actually improve with ECT, even when it is associated with underlying cerebrovascular disease.[12] Therefore, the potential risk of exacerbating cognitive impairment must be weighed against the risk of not treating a severely depressed older person.

Elderly patients experience stress to their cardiovascular system during ECT and must be evaluated carefully for their ability to tolerate such changes, including bradycardia, tachycardia, hypertension or arrhythmia.[5] The specific indications and relative contraindications are well described in the APA Task Force Report.[1]

Patients who reside in nursing homes have higher rates of depression—as much as 40% higher in some studies[13]—as well as high rates of medical comorbidity and disability over long periods of time.[14] These patients are at high risk for mortality, especially the "old-old" who are cognitively impaired, less functional and in poorer health. The treatment of depression in the institutionalised, ill, elderly patient may or may not improve survival, but likely improves quality of life and outcomes of other comorbid medical conditions.[15]

Use of ECT in Patients with Medical Conditions

Patients being considered for ECT, especially the elderly, often have coexisting central nervous system, cardiovascular or respiratory conditions that may cause concern (Table 2). The pre-ECT medical evaluation should identify these conditions, assess their contribution to the overall risk during ECT and suggest ways to reduce this risk. In consideration of these medical risks, the treating psychiatrist, in collaboration with the anesthetist, should arrive at a final recommendation for ECT in addition to any procedural modifications needed to administer the ECT safely. The medical conditions that should be of concern during ECT are outlined here.

Central Nervous System Conditions

Space Occupying Lesions (SOLs)

The APA's Task Force on ECT[16] concluded that there was no absolute contraindication to ECT, although patients with cerebral SOLs are at a substantially increased risk for intracranial pressure and its related problems. These include the possibility of coning, rupture of aneurysms or AV malformations, or worsening of other types of lesions causing increase in mass effect within the cranium during ECT. Several case reports have noted safe and successful ECT in the presence of intracranial tumours, particularly meningomas.[17,18] Recently, the first published report was made of successful ECT administered to a depressed patient with a brain tumour who had increased intracranial pressure.[19] With increased clinical experience and development of newer pharmacological agents pre-, intra- and post-anesthesia, ECT may be used to treat profound depression even in the presence of brain edema. Even though the risks remain high, they must be weighed carefully against the severity and persistence of the psychiatric manifestations for each case.

Intracranial Vascular Masses (IVMs)

Patients being considered for ECT who have IVMs are at increased risk for serious morbidity and mortality due to the possibility of rupture as well as their mass effect. Cardiovascular side effects during ECT, including arterial hypertension, increased cerebral blood flow and increased intracranial pressure, are of concern among these patients. A recent review of the literature and case reports outlines eight cases of ECT performed in patients with IVMs, none of which had an adverse outcome.[20] While these numbers do not establish unequivocal safety in this population, the individual practitioner must con-

Table 2
Medical Conditions of Concern During ECT

ECT should be considered if benefit outweighs risk, which can generally be managed with careful anesthesia technique.

Central Nervous System

Space occupying lesions:
- risk is higher due to raised intracranial pressure

Intracranial vascular masses:
- risk of rupture and mass effect

Stroke:
- hemorrhagic more likely to rebleed vs. ischemic
- no specific guide for waiting period
- generally well tolerated

Dementia:
- greater risk of post-ECT confusion

Cardiovascular
- uncompensated congestive heart failure
- severe valvular disease
- unstable angina
- uncontrolled hypertension
- fragile vascular aneurysms
- clinically significant cardiac arrythmias
- low ejection fraction
- stable patients can be managed medically during ECT with relatively low risk of complications
- recent MI is a risk for re-infarction: clinical decision of when to administer ECT after MI

Respiratory
- careful attention to ensure adequate oxygen delivery during anesthesia and ECT is important

tinue to perform risk versus benefit analysis on a case-by-case basis.

Stroke

Depression develops in 30–60% of patients within two years after a stroke,[21] and impedes recovery and rehabilitation of stroke patients.[22] For patients resistant to or intolerant of antidepressants, ECT can be effective, although there is no generally accepted recommendations on how long to wait after a stroke before ECT is administered. Although the risk in patients with stable lesions is thought to be small, fresh lesions, particularly hemorrhagic lesions, may be more likely to rebleed with ECT than ischemic infarctions.[23] ECT is very effective and generally well tolerated in post-stroke patients, but they are at high risk of relapse despite a robust ECT response and maintenance antidepressant therapy.[24] Caution must be used and treatment should be carefully monitored and administered in the acute post-stroke period, and only in settings where adequate medical, neurologic and radiologic consultations are available.[23]

Patients with MRI evidence of subcortical hyperintensities, but without cognitive impairment (silent cerebrovascular disease), generally respond as well as depressed patients without dementia and do not appear to be at greater long-term cognitive risk with use of ECT.[25] However, these patients, as well as others with stroke and dementia, may be at increased risk for confusion or transient cognitive worsening post-ECT.

Epilepsy

ECT has marked anticonvulsant effects, for which it has been used since the 1940s. It may improve seizure control in epilepsy, especially with intractable epilepsy or status epilepticus that is unresponsive to standard medications.[26]

Dementia

Pre-existing dementia puts a patient at higher risk for transient post-ECT confusion,[27] but the affective benefits of ECT in demented patients are comparable to non-demented patients.[28] An extensive literature review of ECT use in organic dementia and depressive "pseudodementia" found that 73% of patients had a positive response with respect to depression. Patients with subcortical dementias (Parkinson's disease, Huntington's disease, normal pressure hydrocephalus) responded somewhat better than those with cortical dementias (Alzheimer and Pick's diseases). Transient post-ECT confusion was noted in 21% of patients and remitted spontaneously in virtually all cases. Cognitive improvement was noted in 29% of patients, presumably due to alleviation of depression-related cognitive effects.[29]

Parkinson's Disease

Parkinson's disease (PD) patients resistant to or intolerant of standard medications frequently benefit from ECT in the acute as well as continuation or maintenance phases of ECT (C/MECT).[30] Improvements are often in the motor domain, especially in those with "on-off" phenomenon, and are independent of the benefits observed for psychiatric symptoms. The neuromuscular benefits have been found to persist for days to months, allowing better management of refractory advanced PD with less dopaminergic medication. A series of studies were carried out on PD patients with "on-off" phenomenon treated specifically for motor symptoms.[31] These placebo-controlled studies randomised patients to active or sham ECT, and demonstrated substantial reduction of time in "off" periods among the active ECT-treated patients.[31] Among medication-refractory patients, advanced age, severe disability and long duration of treatment with levodopa were predictors of favourable response. Clinical guidelines for the treatment of PD with ECT[30] have been proposed based on these findings.

ECT increases dopaminergic tone, requiring careful reduction in dopaminergic medication to avoid worsening of psychosis or dyskinesias. There are some reports of greater cognitive dysfunction and delirium after ECT in PD,[32] which may be minimised by halving the dose of levodopa and starting the course of ECT with right unilateral, brief-pulse treatments, advancing to bilateral electrode placement if no definite response is obtained by the third treatment.

Cardiovascular Disorders

Prior to modern anesthetic techniques and effective medical management, the cardiovascular system caused the most frequent complications during ECT. These complications have been reduced dramatically in patients with and without prior cardiac history.[33] ECT is increasingly being considered for patients with medication-resistant or -intolerant and significant cardiovascular disease, many of whom are also elderly. Post-ECT changes of blood pressure, heart rate and rhythm in healthy patients are transient and safely managed. In the general population, the estimated mortality with ECT is about 1 per 10,000 patients or 3–4 per 100,000 treatments,[34] mostly due to cardiac complications. Anesthesia effects, muscle relaxants and hypoxia often contribute to the risk of mortality.

ECT has a biphasic effect on the cardiovascular system. Initially, there is a marked parasympathetic discharge coincident with the tonic phase, apnea and a forced expiration-induced Valsalva effect. The increased vagal tone may cause profound bradycardia and hypotension and increase the risk of sinus arrest or arrythmias. This vagal effect, including excessive oral secretions, may be attenuated or blocked by atropine or glycopyrrolate pretreatment. Subconvulsive stimuli (missed attempts at inducing seizures) may result in excess vagal outflow which, if not countered by seizure-induced sympathetic activation, may precipitate severe and life-threatening bradyarrhythmias and hypotension. Using suprathreshold stimuli, pretreatment with anticholinergic medication to prevent such complications should be considered in high-risk patients.

This initial phase is followed by sympathetic discharge causing a 15-fold increase in plasma epinephrine within seconds after the electrical stimulus,[35] with the opposite effect. It leads to increase in cardiac output, systemic vascular resistance, hypertension and tachyarrhythmias, thus increasing myocardial oxygen demand.

Administering short-acting beta-blockers, such as esmolol or labetalol, may attenuate this sympathetic effect. The rate-pressure product index (the product of the heart rate and systolic blood pressure) can be used to predict cardiac ischemia, which may occur when the index is over 20,000. Fortunately, the plasma half-life of norepinephrine is brief and its effects are transient during ECT, although they can lead to myocardial infarction (MI) or intracerebral hemorrhage in vulnerable, often elderly, patients with known risk factors.[36]

A controlled study found that pre-existing disease predicted the type of cardiac complication during ECT.[37] The severe cardiac patients were defined by ejection fraction > 50%, a QRS interval < 100ms, or ≥ 10 extrasystoles. Patients with pre-existing ischemic disease and conduction disorders were at risk for ischemia and arrhythmias, respectively. Despite having eight major and 14 minor cardiac complications among 40 patients, there were no deaths and 38 were able to complete the course of ECT. It was concluded that with close monitoring, ECT could be given with relative safety to patients with severe cardiovascular disease.

A retrospective study of 80 patients divided according to their degree of cardiac risk found the cardiac group to be more prone to developing minor but not major complications compared to case-controls, with no deaths or permanent cardiac morbidity during ECT.[33] Although these studies suggest a higher cardiac risk associated with ECT in patients with cardiovascular disease, the option of ECT should not be dismissed since most patients in both trials were able to complete the course of treatment.

Recent MI is a risk factor for re-infarction during ECT. Although not studied objectively, it is suggested that a three-month interval be allowed after MI prior to ECT. However, the clinical decision of when to administer ECT after MI should be based on the severity of and the time lapsed since the recent MI as well as other effective treatment options available for the psychiatric condition. Other significant cardiovascular risk factors that must be assessed include uncompensated congestive heart failure, severe valvular disease, unstable angina, uncontrolled hypertension, fragile vascular aneurysms and clinically significant cardiac arrythmias.[1]

Pre-ECT Medical Evaluation

A thorough medical evaluation is critical prior to considering ECT for all patients. In addition to a careful history, a physical examination, appropriate bloodwork, an electrocardiogram and a chest radiograph are useful. Optimal medical therapy in preparation for ECT is mandatory to minimise risk during and after treatment. Usual medication should be continued unless there are specific contraindications.[1] Particular attention to blood pressure and heart rate control by avoiding dehydration and hypotension must be made.

Medications to lower cardiovascular risk for anesthesia may be needed, including sympatholytics, other antihypertensives, anticholinergics and short-acting nitrates.[1] Anticoagulation with heparin or warfarin may be safely administered during ECT for patients at risk for embolisation. Cardiac pacemakers generally have a protective effect during ECT by improving the heart's rate and rhythm. A fixed-rate or demand pacemaker can be protective against asystole during excessive vagal tone with ECT.[38] Implanted defibrillators also are not problematic with ECT,[1] but a cardiac electrophysiologist should be consulted prior to treatment. Oxygen administration during ECT protects the myocardium from ischemia.

Consent Issues

Obtaining consent from patients for ECT involves ethical and legal obligations that have been described in the APA's conceptual framework.[1] Frequently, these are elderly patients with the significant extra burden of medical illness, cognitive dysfunction and impaired decision-making ability. Determination of capacity to consent is important in these patients who often are impaired due to their special clinical situation. However, ECT should not be considered different from other medical or surgical procedures with comparable risks and benefits. If incapable, appropriate substitute consent must be promptly obtained to prevent unnecessary suffering, physical morbidity and possible fatality.

Today, ECT is a well-researched medical procedure. It has been significantly refined over the years with little resemblance to the early days of its use. Innovations, including the use of oxygen, muscle relaxants, anesthesia, computerised stimulus delivery and seizure monitoring, have made ECT much safer and more acceptable to patients who require it, often as a life-saving procedure. ◆

No competing financial interests declared.

References

1. The practice of electroconvulsive therapy: Recommendations for treatment, training, and privileging. Second edition. A task force report of The American Psychiatric Association. 2001. American Psychiatric Association.
2. Stewart RB. Advances in pharmacotherapy: Depression in the elderly–issues and advances in treatment. J Clin Pharm Ther 1993;18:243-53.
3. Janicak PG, Davis JM, Gibbons RD, et al. Efficacy of ECT: a meta-analysis. Am J Psychiatry 1985;142:297-302.
4. Sackheim HA. The efficacy of electroconvulsive therapy in the treatment of major depressive disorder. In: Fisher S, Greenberg RP, editors. The limits of biological treatments for psychological distress: Comparisons with psychotherapy and placebo. Hillsdale: Erlbaum, 1989:275-307.
5. Greenberg L, Fink M. The use of electroconvulsive therapy in geriatric patients. Clin Geriatr Med 1992;8:349-54.
6. Tew JD JR, Mulsant BH, Haskett RF, et al. Acute efficacy of ECT in the treatment of major depression in the old-old. Am J Psychiatry 1999;156:1865-70.
7. Black DW, Winokur G, Nasrallah A. A multivariate analysis of the experience of 423 depressed inpatients treated with electroconvulsive therapy. Convuls Ther 1993;9:112-20.
8. Pettinati HM, Mathisen KS, Rosenberg J, et al. Meta-analytical approach to reconciling discrepancies in efficacy between bilateral and unilateral electroconvulsive therapy. Convuls Ther 1986;2:7-17.
9. Rich CL, Spiker DG, Jewell SW, et al. The efficacy of ECT, I: response rate in depressive episodes. Psychiatric Res 1984;11:167-76.
10. Alexopoulos GS, Shamoian CJ, Lucas J, et al. Medical problems of geriatric psychiatric patients and younger controls during electroconvulsive therapy. J Am Geriatr Soc 1984;32:651-4.
11. Sobin C, Sackeim HA, Prudic J et al: Predictors of retrograde amnesia following ECT. Am J Psychiatry. 1995. 152:995-1001
12. Baldwin RC, Benbow SM, Marriott A, et al. Depression in old age: A reconsideration of cerebral disease in relation to outcome. Br J Psychiatry 1993;163:82-90.

13. Katz IR, Lesher E, Kleban M, et al. Clinical features of depression in the nursing home. Int Psychogeriatr 1989;1:5-15.
14. Abrams RC, Teresi JA, Butin DN. Depression in nursing home residents. Clin Geriatr Med 1992;8:2:309-22.
15. Kroessler D, Fogel BS. Electroconvulsive therapy for major depression in the oldest old: Effects of medical comorbidity on post treatment survival . Am J Psychiatry 1993;1:30-7.
16. The American Psychiatric Association Task Force on ECT. The practice of electroconvlusive therapy: recommendations for treatment, training and privileging. Convuls Ther 1990;6:85-120.
17. Fried D, Mann J. Electroconvulsive treatment of a patient with known intracranial tumor. Biol Psychiatry 1988;23:176-80.
18. Starkstein SE, Migliorelli R. ECT in a patient with a frontal craniotomy and residual menginoma. J Neuropsychiatry Clin Neuroscience 1993;5:428-30.
19. Patkar AA, Hill KP, Weinstein SP, et al. ECT in the presence of brain tumor and increased intracranial pressure: evaluation and reduction of risk. J ECT 2000;16:2:189-97.
20. Salaris S, Szuba MP, Traber K. ECT and intracranial vascular masses. J ECT 2000;16:198-203.
21. Robinson RG, Bolduc PL, Price TR. Two year longitudinal study of post stroke mood disorder: diagnosis and outcome at one and two years. Stroke 1987;18:837-43.
22. Parikh RM, Robinson RG, Lipsey JR, et al: The impact of post-stroke depression on recovery in activities of daily living over a 2-year follow-up. Arch Neurol 1990;47:785-9.
23. Weintraub D, Lippmann SB. Electroconvulsive therapy in the acute post-stroke period. J ECT 2000;16:415-8.
24. Coffey CE, Hinckle PE, Weiner Rd, et al. Electroconvulsive therapy of depression in patients with white matter hyperintensity. Biol Psychiatry 1987;22:629-36.
25. Sackeim HA, Decina P, Prohovnik S, et al. Anticonvulsant and antidepressant properties of electroconvulsive therapy: A proposed mechanism of action. Biol Psychiatry 1983;18:1301-10.
26. Krystal AD, Coffey CE. Neuropsychiatric considerations in the use of electroconvulsive therapy. J Neuropsychiatry Clin Neurosci 1997;9:283-92.
27. Zwil AS, McAllister TW, Price TRP. Safety and efficacy of ECT in depressed patients with organic brain disease: review of a clinical experience. Convul Therapy 1992;8:103-9.
28. Nelson JP, Rosenberg DR. ECT treatment of demented elderly patients with major depression: a retrospective study of efficacy and safety. Convuls Therapy 1991;7:157-65.
29. Price TRP, McAllister TW. Safety and efficacy of ECT in depressed patients with dementia. A review of clinical experience. Convuls Therapy 1989;5:61-74.
30. Rasmussen K, Abrams R. Treatment of Parkinson's disease with electroconvulsive therapy. In: Kellner CH, editor. Electroconvulsive therapy, The Psychiatric Clinics of North America, 1991;14:925-33.
31. Anderson K, Balldin J, Gottfries CG, Granérus AK, et al: A double-blind evaluation of electroconvulsive therapy in Parkinson's disease with "on-off" phenomena. Acta Neurologica Scandanavica 1987;76:191-199.
32. Fiegel GS, Hassen MA, Zorumski C, et al. ECT-induced delirium in depressed patients with Parkinson's disease. J Neuropsychiatry Clin Neurosci 1991;3:405-11.
33. Rice EH, Sombrotto LB, Markowitz JC, et al. Cardiovascular morbidity in high-risk patients during ECT. Am J Psychiatry 1994;151:1637-41.
34. Abrams R: Electroconvulsive therapy, 2nd ed. New York, Oxford University Press, 1992.
35. Jones RM, Knight PR. Cardiovascular and hormonal responses to electroconvulsive therapy. Anaesthesia 1981;36: 795-9.
36. Cattan RA, Barry RP, Mead G, et al: Electroconvulsive therapy in octagenerians. J Am Geri Soc 1990;38:753-8.
37. Zielinski RJ, Roose SP, Devanand DP, et al. Cardiovascular complications of ECT in depressed patients with cardiac disease. Am J Psychiatry 1993;150:904-9.
38. Weiner RD, Coffey CE. Electroconvulsive therapy in the medical and neurological patient. In: Stoudemire A, Fogel BS, editors. Psychiatric care of the medical patient. New York: Oxford University Press, 1993.