



Electroconvulsive Therapy

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This position paper was reviewed and approved for republication with major revisions by the Canadian Psychiatric Association's Board of Directors on June 2, 2009. This is the third position paper issued by the CPA on electroconvulsive therapy (ECT). Previous position papers were published in 1980¹ and 1992.² Substantial new findings related to the use of ECT have resulted in the need to update the position of the CPA on ECT. The position paper was developed by the CPA's Standing Committee on Professional Standards and Practice.

Introduction

Electroconvulsive therapy (ECT) remains an important therapeutic option in contemporary psychiatric practice. The mechanism of action of ECT remains incompletely understood; however, extensive research, and 70 years of clinical experience with the treatment, supports the CPA's current recommendation that ECT should remain readily available as a treatment option.

ECT has well defined indications, demonstrated efficacy and safety, well characterized side-effects, and established standards for practice. The decision to use ECT in the treatment of a patient is a medical one, based on the psychiatrist's assessment of the patient's illness and an evaluation of the merits and risks of ECT, compared with alternative treatments. Similar to all medical procedures, the use of ECT requires informed consent from the patient or a substituted decision maker.

Optimal contemporary use of ECT requires considerable expertise on the part of psychiatrists, anesthesiologists,

and, in the case of patients with significant medical illness, other medical specialists. As knowledge about mental illness accumulates, the specific treatments offered by psychiatrists, and the technique of those treatments, will necessarily evolve. The modern psychiatrist must remain abreast of this evolution.

Definition

ECT is a medical procedure in which a brief electrical stimulus is used to induce a cerebral seizure under controlled conditions. Its purpose is to treat specific types of major mental disorders.

History

ECT was developed as a treatment for schizophrenia in 1938 by Ugo Cerletti and Lucio Bini.³ Prior to the 1930s, there were few treatments to offer severely ill psychiatric patients, other than custodial care, sedation, and some social support.⁴ During the 1930s, 4 somatic therapies for schizophrenia were developed including insulin coma,⁵

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prefrontal lobotomy,⁶ pharmacological convulsive therapy,⁷ and ECT. Convulsive treatment for schizophrenia was based on a hypothesized biological antagonism between epilepsy and schizophrenia. The development of a method for using electrical stimuli to induce seizures represented an improvement on pharmacological convulsive treatment, which was associated with technical problems and markedly uncomfortable sensations experienced by conscious patients, pre-ictally. ECT is the only somatic treatment from that era that remains in common use today.

While ECT was originally developed for the treatment of schizophrenia, it was not long after its introduction that it became widely recognized that the best results were obtained in patients with major mood disorders rather than schizophrenia. This observation has been borne out by numerous randomized controlled trials (RCTs), and is reflected in the contemporary diagnostic indications for ECT.^{8,9}

The technique of ECT has evolved and been refined considerably since its introduction. The improvements and refinements have included routine use of brief monitored anesthesia with muscular relaxation and preoxygenation, the use of more efficient brief-pulse electrical stimulation, alternative stimulus electrode placements (unilateral and bifrontal), and the use of electroencephalogram (EEG) seizure monitoring. Despite these improvements, there was a significant decline in the use of ECT in the 1960s and 1970s. The introduction of several effective pharmacological treatments and a vocal anti-ECT lobby were likely important causes of this decline. Recognition of the limitations of pharmacological treatments, unequivocal clinical trial data demonstrating the efficacy of the treatment, and the publication of several comprehensive and objective reports on ECT^{10,11} resulted in a sustained return of clinical and academic interest in the treatment during the following decades.

Although unanswered questions about ECT remain, systematic research and study has clearly established an ongoing role for ECT in the treatment of serious mental disorders.^{8,9}

Indications

The principal diagnostic indications for ECT include major depressive disorder, bipolar disorder (manic, depressed, or mixed phase), and schizophrenia and related conditions (schizoaffective disorder, schizophreniform disorder). In the case of schizophrenia, the effectiveness of ECT is greater when duration of illness is relatively brief or when catatonic or affective symptoms are

prominent. Evidence for the efficacy of ECT for these principal diagnostic indications is robust. Reviews of this evidence are available elsewhere.^{8,9,12}

Under exceptional circumstances, ECT may also be considered as a treatment option for mental disorders not included above. Some primary medical disorders may also respond to ECT; for example, Parkinson's disease,¹³ neuroleptic malignant syndrome,¹⁴ and refractory epilepsy.¹⁵ When considering ECT for unusual indications, the treating psychiatrist should be aware that compelling evidence of the effectiveness of ECT is lacking. The available standard treatments for these other disorders should be pursued before considering the use of ECT.

The decision to use ECT in the treatment of a patient is based on numerous considerations in addition to diagnosis. Some of the important factors include the severity and impairment associated with the condition, the relative need for rapid response to treatment, previous treatment responsiveness, the risks and benefits of ECT, compared with other appropriate treatments, and patient preferences. Although ECT is frequently used as a second- or third-line treatment after psychotropic medications have failed, its use need not be restricted to this setting. Consideration of the factors outlined above may lead the psychiatrist to offer ECT as a primary treatment modality.

Contraindications

There are no absolute contraindications to the use of ECT. However, there are numerous medical conditions that may increase the otherwise low level of risk associated with ECT.^{8,9} Diseases of the cardiovascular, respiratory, and central nervous systems are of greatest importance in this regard. When ECT is considered for a patient with a concurrent medical illness that increases the risk associated with treatment, care must be taken to optimize the patient's medical condition before the administration of the treatment, and to modify the anesthetic approach as required. This preparation will require consultation with an anesthesiologist and other specialist physicians as dictated by the nature of the underlying illness. As in all applications of ECT, the decision to treat a patient in the presence of one of these conditions should be made only after careful consideration of the potential risks and benefits of ECT, alternative treatments, or no treatment.

Efficacy

A full review of the studies demonstrating the effectiveness of ECT is beyond the scope of this paper. Numerous comprehensive reviews of this issue are available elsewhere.^{8,9,12} The following is a summary of the main

conclusions regarding the efficacy of ECT for the principal diagnostic indications.

Numerous RCTs have demonstrated that ECT is efficacious in the treatment of major depression. Genuine ECT is substantially more effective in the acute treatment of depression than sham ECT.¹⁶⁻¹⁸ In addition, studies evaluating the impact of ECT treatment technique have yielded clear evidence that certain forms of ECT (specifically, high stimulus dose unilateral ECT or moderate stimulus dose bilateral ECT) are much more efficacious than low stimulus dose unilateral ECT.^{19,20} ECT is more efficacious than standard antidepressant medication treatments²¹⁻²³; however, clinical trials directly comparing ECT with optimal contemporary pharmacotherapy have not been conducted.

ECT is efficacious in the treatment of manic, depressed, and mixed states in bipolar disorder. Comparative clinical studies of ECT and lithium,²⁴ the combination of lithium plus haloperidol,²⁵ and sham ECT²⁶ have concluded that ECT is efficacious and generally yields superior results than pharmacotherapy. Studies comparing ECT with contemporary combination pharmacotherapy for mania have not been conducted.

Evidence for the efficacy of ECT in schizophrenia is less clear than for mood disorders. Genuine ECT has been shown to be more effective than sham ECT in reducing symptoms of schizophrenia.²⁷⁻²⁹ All of these studies excluded patients with chronic symptoms of schizophrenia and permitted significant affective symptoms. Genuine ECT does not appear to have greater efficacy than sham ECT in patients with chronic schizophrenia.^{30,31} Studies comparing ECT with antipsychotic medication have found no advantage for ECT,^{29,32,33} although the combination of ECT and antipsychotic medication may yield more rapid improvement than antipsychotics alone in nonchronic schizophrenia.^{34,35} More recent studies found that the combination of ECT and an antipsychotic was a more effective maintenance treatment for patients with medication-resistant schizophrenia than either ECT or antipsychotics alone.^{36,37} Clinical experience and case series data also suggest that a significant number of medication resistant patients with schizophrenia may benefit from the combination of ECT and antipsychotic medication.⁹ Taken together, study data suggest that ECT has a more limited role in the treatment of schizophrenia than in the treatment of mood disorders. ECT should rarely be considered as a first-line treatment for schizophrenia. Catatonia, which can occur in the course of severe mood disorders or in schizophrenia, is responsive to treatment with ECT. The presence of catatonia,

regardless of underlying diagnosis, may be a favourable prognostic indicator for acute response to ECT.^{38,39}

The number of treatments required for an effective course of ECT varies substantially between people. Typically, patients with mood disorders require 6 to 12 treatments, while patients with schizophrenia may require a somewhat higher number. A subgroup of patients may show dramatic improvement with only a few treatments. When patients show a slow or insignificant response to a series of treatments, it requires the clinical judgment of the psychiatrist to determine when to recommend continuation, termination, or modification of the treatments. This judgment takes account of such factors as the nature and severity of the patient's symptoms, the extent of cognitive side effects, history of response to ECT, and the degree of response obtained thus far. Treatment modifications in response to nonimprovement may include a switch from unilateral to bilateral ECT, or an increase in the ECT stimulus.

Specific guidelines for a maximum allowable number of treatments are difficult to develop. When patients with mood disorders do not show substantial improvement after 12 to 14 treatments, consultation with a psychiatric colleague should be considered before further extending the course of treatment. Similarly, when patients with schizophrenia show little improvement after 14 to 16 treatments, a second opinion should be considered before proceeding further.

ECT is usually administered at a frequency of 2 or 3 times per week. Comparative studies indicate that ECT administration at a frequency of twice per week results in the same degree of clinical improvement, with a slightly slower speed of response.^{40,41} The twice weekly treatment schedule may also yield a lesser level of short-term cognitive side effects.^{40,41} In clinical practice it may also be necessary to reduce treatment frequency if excessive cognitive side effects occur. Practices such as prolonged daily administrations of ECT or multiple seizure inductions per treatment session are not supported by scientific evidence, and are not recommended.⁹ However, brief periods of daily treatment or the induction of 2 treatments per session may sometimes be appropriate early in the course of treatment if a particularly rapid treatment response is required.

The mental disorders for which ECT is ordinarily prescribed are commonly recurrent in nature. Longitudinal studies of patients treated for severe forms of depression (the most common indication for ECT) show high relapse rates in the year following acute treatment.^{42,43} For these reasons, continuation therapy

(a relapse prevention strategy) following successful treatment with ECT is indicated. Continuation therapy may consist of medication treatment, continuation of ECT at a reduced treatment frequency (for example, a single ECT treatment every 1 to 4 weeks), or both. Two large RCTs provide evidence that both continuation ECT and continuation pharmacotherapy with nortriptyline and lithium are effective in preventing relapse of depression following response to ECT.^{42,44} A smaller RCT provides evidence that the combination of ECT and nortriptyline is a more effective continuation and maintenance treatment than nortriptyline alone in late life psychotic depression.⁴⁵ Case series data also suggest that continuation ECT may be effective for relapse prevention in schizophrenia.^{46,47} Maintenance therapy (a recurrence prevention strategy) is most clearly indicated in patients who already have an established pattern of recurrent illness, and for patients who experience return of symptoms when attempting to discontinue continuation therapy⁹; however, most patients who receive ECT are candidates for maintenance therapy. Maintenance therapy, such as continuation therapy, may consist of medication treatment, ongoing ECT at a reduced treatment frequency or both. The required duration of maintenance therapy is not established and must be individualized based on clinical judgment.

Adverse Effects

ECT may be accompanied by potentially significant medical complications including prolonged seizures, prolonged apnea, or cardiac or pulmonary complications. However, with appropriate pre-ECT assessment, optimization of overall medical care, appropriate anesthetic management, and prompt attention to emerging medical concerns, such complications can generally be avoided or successfully managed.^{8,9} Contemporary use of ECT including preoxygenation, brief anesthesia, muscular relaxation, use of bite block, and physiological monitoring is associated with a very low rate of morbidity and mortality. Recent estimates of the mortality rate associated with ECT are about 2 deaths per 100 000 treatments.^{48,49} Reports from several countries indicate that mortality following hospitalization is lower for ECT treated patients than for depressed patients who did not receive ECT.^{50,51}

Common complaints following ECT include headache, muscle aches, nausea, and occasionally vomiting. These self-limited symptoms are not generally severe, and typically respond favourably to symptomatic treatments.

The adverse effect of ECT that arouses the greatest concern for patients and families is memory dysfunction. Immediately following ECT there is a period of postictal confusion. After clearing of the postictal confusion, retrograde amnesia (forgetting of events before the seizure) and anterograde amnesia (forgetting of events after the seizure) can be demonstrated using various neuropsychological tests. Patients may also experience subjective memory dysfunction that does not correlate with objectively measured memory function. A few patients experience longer-lasting subjective memory impairment. A comprehensive review of these memory effects is available elsewhere.^{8,9} Although experts differ in their interpretation of the results of objective studies (for example, Abrams⁸ and Sackeim et al¹⁹), there is agreement that the degree of measurable amnesia is significantly related to the specific technique of ECT that is used. Higher treatment number, more frequent treatment schedule, bilateral electrode placement, high electrical stimulus intensity, and sine-wave stimuli have been associated with more marked memory-related adverse effects.^{52–55} Anterograde amnesia has not been shown to persist for more than a few weeks following ECT.^{20,54} Retrograde amnesia is most pronounced immediately following ECT and with the passage of time the degree of retrograde amnesia subsides. The time interval most likely to be affected with enduring memory loss is the period beginning several months before and ending several weeks after ECT.⁹ Although both autobiographical and public events may be affected, retrograde amnesia is generally greater for public information than autobiographical information.⁵⁵

Claims have been made that ECT causes brain damage⁵⁶ Such claims lack validity; comprehensive and objective reviews of available data do not find credible evidence that ECT causes structural brain damage.^{57,58}

Assessment and Documentation

Before the administration of ECT, a thorough evaluation of the patient's psychiatric and medical status is required, including physical examination. Conditions of particular concern that would prompt consultation with an anesthesiologist include unstable angina, recent myocardial infarction, recent cerebrovascular accident, valvular heart disease, congestive heart failure, aneurysm, significant pulmonary disease, diabetes, hypertension, hiatus hernia, and adverse reactions to previous anesthetics^{9,59} Laboratory investigations are guided by clinical suspicions or findings, but these will often include

a complete blood count, serum electrolytes, and renal function tests. All patients aged 50 years and older should receive an electrocardiogram, as should those who have known cardiovascular disease, hypertension, diabetes, renal disease, or pulmonary disease.⁵⁹ With coexisting medical conditions or advanced age, other specialty consultations may be required. Ideally, the patient's American Society of Anesthesiologists (ASA) physical status class⁶⁰ will be determined ahead of time as this could decide whether the patient should be admitted as an inpatient and (or) what location ECT would be best performed. The results of this complete evaluation should be documented in the patient's record before commencing ECT. The record must also contain the signed consent, the indication(s) for ECT, baseline mental status exam, and recent vital signs. The psychiatric team is responsible for ordering the holding, discontinuing, or tapering of psychotropics affecting ECT, when required. According to the Canadian Anesthesiologists' Society,⁶¹ patients should fast for 8 hours after a meal that includes meat, or fried or fatty foods, 6 hours after a light meal (such as toast and a clear fluid), and 2 hours after clear fluids.

Between ECT treatments, the attending psychiatrist should reassess and document changes (at least weekly for an index acute series) in target symptoms and the occurrence of adverse effects, particularly, cognitive dysfunction. The physician who administers the ECT should keep a record of each treatment including stimulus parameters and electrode placement, the doses of anaesthetic and muscle relaxant used, and the quality and duration of seizure activity elicited. After the course of ECT has been terminated, a summary note should be completed for the medical record.

The ECT Stimulus

Since Sackeim et al's⁶² landmark study demonstrating that generating a generalized seizure of sufficient duration is not, in itself, sufficient for a therapeutic response, a wealth of research has been directed at refining a technique that can produce an efficacious seizure with less cognitive impairment risk. It is beyond the scope of this paper to discuss details of the technique as complete descriptions are available elsewhere.^{8,9,59} However, it is worth emphasizing that the specific technique used in ECT is a matter of considerable consequence. Electrode placement, stimulus intensity and waveform, treatment frequency, and concomitant psychotropic drugs have been mentioned previously as factors affecting the severity of cognitive side effects.

These parameters can also affect its antidepressant efficacy and (or) speed of response. In contrast, cumulative seizure duration over an ECT series is not associated with outcome.^{20,63-65}

The d'Elia placement is the preferred standard for unilateral ECT⁹ and, since its development,⁶⁶ there has been considerable controversy over the merits of unilateral, compared with bilateral electrode placements regarding efficacy and memory impairment. Bitemporal ECT has been clearly associated with greater anterograde and retrograde amnesia.^{20,55,66} Earlier reports on the relative efficacy of the 2 electrode placements have been much more divided, but later reports on markedly supra-threshold right unilateral ECT^{19,67-69} have shown comparable responses. Bifrontal ECT⁷⁰ is an alternative technique that can be as efficacious as bitemporal ECT,^{12,71} but may be cognitively advantageous,^{71,72} although more data are needed to support this assertion. Left unilateral ECT can also be cognitive sparing and may be of consideration for those who rely on right hemispheric (visual-spatial) function for their livelihood.⁸ In summary, there are various options when initiating ECT. Unilateral ECT may be advantageous when it is especially important to reduce cognitive side effects. Traditionally, practitioners tend to favour bilateral ECT when there is a greater urgency for improvement,^{9,59} although some studies show similar speeds of antidepressant response between bitemporal ECT and markedly suprathreshold unilateral ECT in non-life-threatening situations.^{19,69} If unilateral ECT fails after 6 to 10 treatments, or there is a history of failure, one could consider bilateral ECT.⁹ Bilateral ECT is preferred for acute mania⁵⁹ and probably for severe catatonia.³⁹

The intensity of the stimulus and the stimulus pulse width waveform are other important parameters for consideration, and dosing for ECT is analogous to a therapeutic window. Moderately to markedly suprathreshold ECT is the goal for unilateral placement and entails applying a stimulus dose anywhere from 2.5 to 6 times above seizure threshold.⁹ In contrast, modestly to moderately suprathreshold ECT is the goal for bilateral placement, which implies 1.5 to 2.5 times threshold.⁹ Barely suprathreshold brief-pulse unilateral ECT is remarkably ineffective.^{62,70} Increasing the stimulus intensity increases the efficacy of brief-pulsewidth unilateral ECT.^{19,20,67} Ultrabrief-pulse, markedly suprathreshold (6 times), unilateral ECT appears to confer similar responses as brief-pulse bilateral or unilateral ECT, but with significantly less amnesic effects, compared with brief-pulse unilateral ECT.⁶⁹ This important study requires

replication, particularly the finding that ultrabrief-pulse bitemporal ECT was inferior to unilateral ECT in efficacy. Suprathreshold stimuli above 2.5 times for bilateral ECT can lead to greater cognitive impairment out of proportion to efficacy.^{9,59} For brief-pulsewidth unilateral ECT, this likely results when stimuli are above 8 times threshold.⁶⁸

Since stimulus intensity relative to seizure threshold (ST) has been identified as an important determinant of both the efficacy and memory side effects of ECT, empirical titration has been advocated as the most precise way to administer the ECT stimulus.^{9,59} Empirical titration involves identifying ST at the first ECT session by increasing the intensity incrementally in a systematic way until seizure activity is observed.⁷³ Age-based formulas^{8,74} to determine the initial stimulus intensity are reasonable alternatives. For unilateral ECT, a fixed high-dose strategy with low-pulsewidth (≤ 0.5 ms) and long stimulus duration (≥ 6 s) is another alternative technique.⁸ In contrast, for bilateral ECT, a high fixed dose strategy should only be used rarely and reserved for emergency patients (for example, patients with catatonia). Stimulus dose titration may be particularly useful for those who are at higher risk to develop amnesia during a course of ECT (for example, concomitant dementia, or history of severe and persistent post-ECT cognitive impairment). A seizure should be monitored both by the motor ictal response and by, at least, single-channel EEG monitoring.⁹ The challenge in maintaining therapeutic response is that the ST can unpredictably rise for some patients during the treatment course.^{71,75,76} Therefore, adjusting the stimulus intensity may be necessary over an ECT series, as guided by the patient's clinical and cognitive status, whether the seizure meets the minimum EEG seizure duration of 15 seconds,⁹ and possibly by ictal and postictal EEG parameters.⁷⁷ STs approaching or exceeding the maximum charge output of the ECT device should prompt a review of anesthetic factors, concomitant psychotropics, use of deliberate hyperventilation, and hydration status.⁹ The earliest ECT devices employed a sine wave form stimulus.³ This delivered a charge in excess to the amount needed to efficiently elicit a seizure, leading to substantially greater amnesia.^{52,78} For this reason, the continued use of sine wave devices is not justified, and a brief-pulse device is currently recommended.^{9,59}

The question of what constitutes the optimal technique for administering ECT is not completely answered. Therefore, the clinician administering or prescribing ECT must make decisions about various aspects of the

treatment technique based on an ongoing individualized assessment of the relative risks and benefits. The ECT practitioner needs to be skilled and flexible in performing the various techniques that can enhance outcome.

Mechanism of Action

The mechanism of action of ECT remains uncertain. There is no unifying hypothesis for the remarkable efficacy of ECT in treating major depression. However, there are many contemporary theories that may explain its biological effects. These include increases in neurotrophins, changes in the regional metabolism of relevant brain areas, and its physiological anticonvulsant effects.

Hypotheses surrounding changes in transmission of central monoamines, some of which are similar to antidepressants and some which differ,^{79–81} have been superseded by theories involving the increase in secondary messengers, which can enduringly increase the gene expression of neurotrophic factors, such as brain-derived neurotrophic factor (BDNF) and others.^{82–84} These changes in neurotrophic factors can lead to neurogenesis, neuroplasticity, and dendritic sprouting in key areas such as the hippocampus in animal models involving electroconvulsive stimulation (ECS), which can translate into changes in monoamine transmission.⁸⁵ Importantly, this finding supports the observation that there is no structural brain damage with contemporary ECT.^{57,58} The rise in BDNF with chronic ECS has been the most studied to date.⁸⁶ There are data to suggest limbic BDNF levels diminish in patients with depression correlating with decreased hippocampal and prefrontal volumes in the brains of patients with depression.⁸⁷ Antidepressants also appear to increase BDNF levels. Indirect evidence for this theory in humans is that ECT can produce a rise in plasma BDNF levels that correlate with clinical response.⁸⁸

Some studies have shown a course of ECT will reduce regional cerebral blood flows (rCBFs) or cerebral metabolic rates.^{89,90} However, this is an inconsistent finding.⁹¹ The significance of reduced cortical rCBF is unclear, but cortical circuits do have reciprocal connections to subcortical areas implicated in depression. It is not clear if ECT has similar or different effects, compared with other brain stimulation techniques approved for treating depression.^{92,93} The anticonvulsant hypothesis for ECT postulates that reductions in rCBF with ECT may relate to the strength of seizure inhibition and the rise in seizure threshold that accompanies clinical response, especially in right unilateral ECT.⁸⁹ Anticonvulsant effects of ECT can be inferred from

ictal, postictal, and interictal EEGs.^{94,95} Such anticonvulsant effects may be due to increased gamma amino butyric acidergic (GABAergic) transmission resulting from ECT^{96,97} as there is evidence for reduced central GABA levels in patients with depression. However, higher seizure thresholds correlating with outcome in ECT responders may not necessarily apply for bilateral ECT.⁹⁸

To date, there are no reliable biomarkers that are widely available, such as BDNF levels or EEG spectral analysis, which are recommended for predicting or tracking responders to ECT with depression. The mechanisms for ECT's efficacy in mania, schizophrenia, catatonia, and Parkinson disease have not been extensively investigated. What is clear is that there is no support for psychodynamic theories that have generally emphasized the role of ECT as punishment for imagined wrongdoing and amnesia for the causes of depression.⁹⁹

Consent to Treatment

Although obtaining patient consent before any therapeutic intervention is recommended, perhaps nowhere else in contemporary psychiatric practice is it as important as before initiating a course of ECT. This is due, in part, to the regrettable degree of misperception surrounding the procedure, especially as it is currently practiced. Written consent is required but a patient's mere signature on a piece of paper is not sufficient. A recent British paper¹⁰⁰ identified that about a third of patients who had signed a form did not feel as if they had freely consented to the procedure. Hence the attending psychiatrist or other designated medical personnel on the patient care team must be directly involved in obtaining informed consent. Further, for the consent to be truly informed, the patient needs to be apprised about the nature of the illness being treated, the expected benefits and possible adverse effects of ECT, as well as the benefits and risks of other alternatives, including providing no treatment, in addition to time being provided for answering patient questions.¹⁰¹

Additionally, an assessment of the patient's competence to give consent to the treatment is required. When this assessment reveals a diminished capacity, an inquiry into the presence of an advance care directive is appropriate, especially as an expressed wish to refuse ECT is conceivable.^{102,103} However, it is interesting to note that an equally high response rate has been demonstrated in consenting and nonconsenting (compulsory treatment by substituted consent) patients with severe depression.¹⁰⁴

Special care should be taken in the obtaining of consent in special patient populations where the capacity to give consent to treatment may be compromised, for example, in patients with medical illness, elderly patients, or patients with severe depression.¹⁰⁵ However, findings from recent literature would suggest that not only is decisional capacity often intact in the latter 2 groups, but that appropriate education further enhances their decisional capacity.^{106,107}

When a patient is determined to be incompetent to properly give consent and there is no advance care directive in place, substituted consent should be obtained in accordance with local requirements and provincial legislation. It is often prudent to obtain a second psychiatric opinion under these circumstances, when possible.

Although typically a written form is only signed once, informed consent is a continuous process that begins before the initiation of ECT and continues throughout the course of treatment. Consequently, the treatment team needs to remain cognizant that consent may be withdrawn at any time.

Education

While all psychiatrists should be knowledgeable of the indications and relative contraindications of ECT, those who administer the treatment need to maintain a high level of knowledge and familiarity with advances in the technique. The full spectrum of education begins in medical school, continues during residency training, and progresses with the continued professional development of the practising psychiatrist. Adequate educational opportunities are paramount as the training characteristics of psychiatrists appear to influence their tendency to use the procedure in practice.¹⁰⁸

Both live demonstration and viewing an educational videotape of ECT appear to be effective methods for teaching medical students about the procedure.¹⁰⁹ However, more robust and standardized curricula need to be in place in psychiatric residency programs.^{110,111} More recently, the American Psychiatric Association's Task Force on ECT has made recommendations for the comprehensive training of residents in ECT administration.⁹ Notwithstanding, there are diverse educational experiences, at least in American residency programs, ranging from some programs having brief, elective observation to others with progressive formal didactic sessions and readings, informed observation, and hands-on supervised learning, complete with formalized evaluative processes.¹¹² It is incumbent on the psychiatrist

who administers ECT to remain up-to-date in the field as part of maintaining their Canadian Medical Educational Directions for Specialists (CanMEDS) competencies.¹¹³

Research

Despite Salzman's¹¹⁴ clarion call, about 10 years ago, for more research to be conducted relating to various aspects of ECT, relatively little has been done, especially if one considers research on pharmacological treatments by comparison. This is unfortunate as the relative paucity of academic interest likely plays a role in the wide geographic variation in usage of this treatment modality,¹¹⁵ thereby potentially depriving some patients of its clinical utility.

While not attempting to be all-inclusive, examples of the types of investigations and answers that are needed follow. Many of the extant efficacy studies are dated and generally limited to patients with depression. Modern, rigorously designed, and powered multisite studies of people with depression and of other clinical populations are needed. In particular, clinical trials directly comparing ECT to current-state pharmacotherapy to assess short- and long-term efficacy, cost-effectiveness, safety, and quality of life are needed. We need to better understand that the predictors of response and relapse have better evidence to advise us on technique issues, as well as to establish biomarkers for tracking the response to treatment. Although progress is being made in the areas of brain imaging and molecular biology, more work remains on delineating ECT's mechanisms of action, particularly in nondepressed states.

Summary and Recommendations

When used according to contemporary standards, ECT is a safe and effective treatment that should continue to be readily available as a treatment option for mental disorders.

ECT should be used only on the recommendation of a psychiatrist, and should be administered by a physician, preferably a psychiatrist.

The main conditions for which ECT is indicated are major depressive disorder, bipolar disorder, and nonchronic episodes of schizophrenia and related psychoses, particularly when affective or catatonic features are prominent. A second psychiatric opinion is recommended if ECT is to be used for an unusual indication.

A thorough evaluation, including medical history, physical examination, and appropriate laboratory investigations, is necessary to detect and assess significant

medical illnesses before the administration of ECT. If an active medical disorder is present, steps should be taken to treat the disorder and (or) to further modify the technique of ECT to minimize the possible risks. Consultation(s) from an anesthesiologist and other medical specialists should be obtained, if indicated.

During a course of ECT, the psychiatrist should regularly reassess and document changes in target symptoms and the occurrence of adverse effects. Evaluation of cognitive functions is part of this process.

Modification of ECT with brief anesthesia, muscle relaxants, and preoxygenation should be accomplished by a suitably qualified physician, unless specific contraindications to the use of these modifications are present. The seizure needs to be appropriately monitored, and equipment must be available for emergency resuscitation during or after the treatment if needed.

The decision to use ECT requires informed consent from the patient or a substituted decision maker. If the patient is incompetent to give this consent, a consultation from another psychiatrist regarding the use of ECT should be obtained.

ECT is a complex medical intervention. Practitioners require considerable knowledge and practical skills to optimally perform the procedure. Psychiatric residencies should include training in ECT and psychiatrists should keep up to date with advances in the theory and practice of ECT.

Continuing basic and clinical research is encouraged to delineate the mechanism of action of ECT and to further improve the clinical application of the treatment.

The position of the CPA on ECT should be periodically reviewed, as dictated by changes in our knowledge and understanding of the treatment of major mental illness.

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