Electroconvulsive Therapy

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Electroconvulsive Therapy

Electroconvulsive therapy (ECT) is one of the most controversial treatments in modern psychiatry. It has been the subject of movies, documentaries, protests, and political referendums. Few treatments have come under such scrutiny or vilification. ECT’s protagonists emphasize its safety and efficacy. ECT’s antagonists claim that it causes irreversible brain damage and severe memory loss. Researchers in the field of ECT are enabling clinicians to address these issues in objective and meaningful ways. Apart from the rhetoric and polemics, a powerful science has emerged for the ECT practitioner and patient. The breadth of this work provides for a comprehensive discussion about efficacy, safety, side effects, patient selection, and treatment methods.

MECHANISM OF ACTION

• What causes the ameliorative action of electroconvulsive therapy?

There is a wide array of ideas regarding ECT’s action. However, because there are multiple, simultaneous changes induced by each ECT treatment and over a course of treatment, it is difficult to separate one change from another to determine a single locus of action. Currently, no unified theory exists as to how ECT exerts its action; a myriad of work looking at diverse systems exists in the literature. Contemporary theories regarding ECT’s mode of action fall into 3 primary groups: 1) enhancement of monoamine transmission (eg, dopamine, norepinephrine, serotonin), 2) neurotrophic effects, and 3) anticonvulsant effect.

MONOAMINE TRANSMISSION

A variety of serotonin receptor changes have been reported in studies measuring the effects of long-term ECT. Many or most of these involve animal studies, where applicability to the human brain is far from certain. These changes are shown in Table 1. Studies have suggested that the actual concentration of serotonin is not as critical or central a locus of action as in antidepressants. Some studies indicate a relationship between the serotonergic system and the opiate system, suggesting that ECT may exert its action by affecting this system. A number of studies fail to show any convincing effect of ECT on norepinephrine turnover. Plasma catecholamines increase in response to ECT but are not altered with repeated ECT. Mann et al found that the degree of catecholamine increase correlated with clinical response. This suggests that ECT’s antidepressant action may be related to its effect on catecholamine release via the sympathetic nervous system. However, this finding does not mean this effect is part of ECT’s antidepressant action.

ECT is useful in treating patients with Parkinson’s disease, suggesting that it increases dopaminergic transmission in the basal ganglia. Indeed, most evidence does indicate that ECT causes an increase in dopaminergic activity. This seems to be mostly due to an increase in dopamine turnover, although a receptor effect cannot be excluded.

NEUROTROPIC EFFECTS

Using animal models, Duman et al have suggested that ECT works by activating the adenylyl cyclase system and thereby increasing brain-derived neurotrophic factor (BDNF) and its receptor, TrkB, in the hippocampus and cerebral cortex. BDNF has been shown to increase synaptic strength, survival, and growth of adult neurons. BDNF has been shown to increase norepinephrine and serotonin turnover and sprouting of serotonergic terminals. Duman et al hypothesize that ECT reverses the atrophy of stress-vulnerable neurons or protects them from any further damage by regulation of these neurotropic factors.

ANTICONVULSANT EFFECT

Sackeim et al report that clinical response to ECT is related to an increase in seizure threshold throughout a course of treatments. Using data from animal models, at least 3 possible mechanisms have been suggested: 1) ECT enhances GABAergic transmission that contributes to the increase in seizure threshold; 2) antagonism of GABAergic transmission is a key factor in seizure induction with ECT; and 3) ECT produces endogenous anticonvulsant substances, at least one of which has opioid-like properties. The magnitude of the change in seizure threshold varies directly with the degree of clinical improvement.

Positron emission tomography scanning studies in conjunction with ECT also demonstrate that the increase in seizure threshold throughout a course of ECT varies directly with decreases in regional cerebral blood flow.
and cerebral metabolic rate in anterior frontal regions. Sackeim et al also found that the electroencephalograms (EEGs) of patients undergoing ECT showed the largest increases in slow-wave (ie, delta and theta) activity in those patients who had the best clinical response to ECT. The authors state that the efficacy of ECT is linked to reductions in cerebral blood flow and increases in EEG slow-wave activity in a network of prefrontal cortical regions, suggesting antidepressant response depends on diminished functional activity in a specific brain region.

**EFFICACY**

- **What evidence of electroconvulsive therapy’s efficacy exists?**

  The clinical literature establishing the efficacy of ECT in treating specific disorders is among the most substantial for any medical treatment. In 1985 the National Institute of Mental Health determined that ECT was efficacious in treating mania, delusional and severe endogenous depression, and certain schizophrenic syndromes. A recent summary by Herman et al showed the greatest evidence base for using ECT to treat major depressive disorder. Other diagnostic groups or behaviors with favorable study efficacy included bipolar disorder, mania, and Parkinson’s disease. Few studies have shown efficacy of ECT as a primary treatment for schizophrenia. More studies show its efficacy in treating schizoaffective disorder. Case reports demonstrate ECT’s efficacy in treating catatonia, delirium, neuroleptic malignant syndrome (NMS), tardive dyskinesia, tardive dystonia, neuroleptic-induced parkinsonism, akathisia, and substance-induced psychosis, among others.

**SAFETY AND SIDE EFFECTS**

**MORBIDITY AND MORTALITY**

A reasonable estimate of the mortality associated with ECT is 1 per 10,000 patients or 1 per 80,000 treatments. Patients with severe medical illness may have higher rates. This is on par with the risk of death from minor surgery or child birth. When mortality occurs with ECT, it usually happens immediately after the seizure or during the postictal recovery period. ECT mortality and morbidity are usually of cardiovascular or pulmonary origin. Other complications that can occur with ECT include prolonged seizures, status epilepticus, tardive dyskinesia, and neuroleptic-induced parkinsonism, akathisia, and substance-induced psychosis, among others.

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**Table 1. Selective Biochemical Effects of Electroconvulsive Therapy**

<table>
<thead>
<tr>
<th>Neurotransmitter/neuropeptide concentrations</th>
<th>Increased</th>
<th>Decreased</th>
</tr>
</thead>
<tbody>
<tr>
<td>Norepinephrine</td>
<td></td>
<td>Acetylcholine</td>
</tr>
<tr>
<td>Dopamine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serotonin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GABA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>β-Endorphin</td>
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<table>
<thead>
<tr>
<th>Receptor density</th>
<th>Increased</th>
<th>Decreased</th>
</tr>
</thead>
<tbody>
<tr>
<td>5-HT2</td>
<td></td>
<td>β2-Adrenergic</td>
</tr>
<tr>
<td>D1</td>
<td></td>
<td>α2-Adrenergic</td>
</tr>
<tr>
<td>GABA B</td>
<td></td>
<td>5-HT1A</td>
</tr>
<tr>
<td>Adenosine A1</td>
<td></td>
<td>Muscarinic</td>
</tr>
<tr>
<td>δ-Opioid</td>
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</table>

<table>
<thead>
<tr>
<th>Second messenger systems</th>
<th>Increased and decreased:</th>
<th>Decreased:</th>
</tr>
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<tbody>
<tr>
<td>cAMP</td>
<td></td>
<td>PKC</td>
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<table>
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<tr>
<th>Gene products</th>
<th>Increased mRNA:</th>
<th>Decreased mRNA:</th>
</tr>
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<tr>
<td>GABA-A (α1, γ2)</td>
<td>Peptidylglycine</td>
<td>Prodynorphia</td>
</tr>
<tr>
<td>β1-Adrenergic</td>
<td>α-amidating</td>
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<tr>
<td>5-HT1</td>
<td>monooxygenase</td>
<td></td>
</tr>
<tr>
<td>Tyrosine hydroxylase</td>
<td>Preprocholecysto-</td>
<td></td>
</tr>
<tr>
<td>Ylase</td>
<td>kinin</td>
<td></td>
</tr>
<tr>
<td>Neuropeptide Y</td>
<td>Preprotachykinin-A</td>
<td></td>
</tr>
<tr>
<td>Cerebral ornithine decarboxylase</td>
<td>Corticotropin-</td>
<td></td>
</tr>
<tr>
<td>Somatostatin</td>
<td>releasing</td>
<td>hormone</td>
</tr>
<tr>
<td>Preproenkephalin-A</td>
<td>Arginine vaso-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>pressin</td>
<td></td>
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<table>
<thead>
<tr>
<th>Proto-oncogenes</th>
<th>Increased:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>c-fos</td>
<td></td>
<td></td>
</tr>
<tr>
<td>c-jun</td>
<td></td>
<td></td>
</tr>
<tr>
<td>jun-B</td>
<td></td>
<td></td>
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<tr>
<td>zif/268</td>
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</tr>
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</table>

seizures, prolonged apnea, headache (typically frontal and throbbing), muscle soreness, nausea, skin burns, treatment emergent mania, delirium, and memory loss.

**COGNITIVE SIDE EFFECTS**

- **How extensive are the cognitive side effects of electroconvulsive therapy?**

  The cognitive side effects are the more enduring and problematic aspects of ECT. These side effects are related to the methods of ECT administration. Methods that increase the likelihood, severity, and persistence of cognitive side effects include bilateral electrode placement, sine wave current, high electrical energy, high dosage of barbiturate anesthetic, frequent treatments, and a high number of treatments. Methods that diminish cognitive dysfunction include right unilateral electrode placement, brief pulse current, lower electrical energy (confounded by effects on efficacy, as discussed below), lowest possible doses of barbiturate anesthesia and adjuvant anticholinergics, longer intervals between treatments, and fewer treatments. Cognitive side effects are also influenced by the severity of psychiatric illness. Sobin et al reported that depressed patients with no known neurologic disorder, who had greater retrograde amnesia at long-term follow-up, also had greater pre-ECT global cognitive impairment. Patients with preexisting neurologic disease or insult may also be at greater risk for ECT-induced delirium and memory deficits.

  Many patients who receive ECT have decreased attention and concentration as a product of their mental illness. These symptoms typically improve with response of the psychiatric illness to ECT. Consequently, improvement can be measured in several cognitive domains upon completion of ECT. It has not been proven that ECT will result in enduring damage to executive functions (eg, the capacity to shift mental sets), abstract reasoning, creativity, semantic memory, implicit memory, or skill acquisition or retention.

**MEMORY IMPAIRMENT**

- **Does electroconvulsive therapy cause impairment of anterograde or retrograde memory?**

  ECT does cause impairment of anterograde and retrograde memory. Anterograde memory is the ability to retain newly learned information. Anterograde amnesia resolves rapidly upon completion of ECT. No study has documented anterograde amnesia for more than a few weeks after completing ECT.

  Retrograde memory encompasses recall of past events, including personal and public information. Retrograde amnesia is usually densest for events that occurred closest to the time of ECT treatment. The degree of retrograde amnesia decreases with increasing time following completion of a course of ECT. This improvement begins within days of finishing ECT. Retrograde amnesia is rarely continuous but is characterized by gaps in recall. Memory of remotest events tends to return first, with those events closest to the time of ECT returning later. The time course for recovery from retrograde amnesia is typically longer than that for recovery from anterograde amnesia. Some patients can experience permanent retrograde memory loss as a consequence of treatment with ECT. At the current time, the rate of this permanent impairment is not clear, presumably because the incidence is so rare.

  There is little association between memory self-ratings and objective neuropsychological testing in patients who have received ECT, but strong associations have been reported between the severity of subjective memory complaints and mood state. The reader is cautioned, however, that the inability to verify objective neuropsychological deficits in patients with subjective memory complaints may reflect both the low incidence of these effects and the limitations of current testing methods. It is prudent to address these issues carefully in an informed consent process.

**INDICATIONS**

The most recent American Psychiatric Association (APA) Task Force Report on ECT emphasizes that ECT has a place as primary therapy, prior to a course of psychotropic medications, for some mental disorders. The report adjures that ECT should not be reserved for use only as a last resort. ECT may work faster and more effectively than other modes of treatment. In depression and acute mania, substantial improvement is often seen after just a few treatments, and the time to achieve maximal symptom remission is more rapid with ECT than with psychotropics. In major depression, ECT is more likely to be effective than antidepressants if the index episode of the illness is more chronic. Thus, factors that influence first-line use of ECT include the severity of a patient’s psychiatric or medical status (ie, prompting a need for rapid, definitive response), lethality (ie, suicidality, homicidality), and treatment history (ie, a history of poor medication response or a good response to ECT in prior illness episodes). In patients with certain medical conditions (eg, pregnant women, the elderly, the physically debilitated) ECT may be safer than psychotropics. ECT is also useful in treating certain medical conditions (Table 2).
**MAJOR DEPRESSION**

ECT is effective for both unipolar and bipolar depressive episodes. ECT has an 80% to 90% response rate in patients who are experiencing their first index episode of illness.14,15 Among patients who have not responded to 1 or more adequate antidepressant trials, ECT has a 50% to 60% response rate.15,16 Catatonia or catatonic symptoms may be a particularly favorable prognostic sign.7

- Are antidepressant drugs more effective than electroconvulsive therapy?

No trial has ever found an antidepressant medication regimen to be more effective than ECT. However, several factors influence a patient’s response to ECT. Older patients are more likely than younger patients to show marked benefit.11,17 Electrode placement and stimulus dosage profoundly affect the efficacy of the treatment; thus, technical factors in ECT administration strongly influence effectiveness. ECT confers no advantage in reducing the risk of relapse or recurrence of illness.

**Combination Therapy**

Tricyclic antidepressants have been given safely with ECT, and some improvement in response rates has been noted with the combination. More studies need to be done to determine the effectiveness of newer antidepressants (eg, selective serotonin reuptake inhibitors) combined with ECT. Starting an antidepressant after a course of ECT is necessary for adequate prophylaxis. Some practitioners may begin this during ECT. Maintenance ECT in lieu of medication is also a viable prophylactic strategy.

**MANIA**

Mania responds well to ECT. One study reported an 80% improvement in 589 patients with acute mania.18 Prospective studies have been done comparing ECT to lithium,19 to lithium plus haloperidol,18,20 and to sham ECT in patients receiving neuroleptic treatment.21 All of these studies indicated that ECT was effective and likely to provide a better short-term outcome than pharmacotherapy.

Because there are very effective antimanic medications, most manic patients are referred to ECT only because they have failed to respond to medication. Studies have shown that a substantial number of medication-resistant manic patients respond favorably to ECT.20,22 ECT response rates for medication-resistant manic patients are lower than for manic patients who receive ECT as first-line treatment (ie, have not demonstrated medication resistance). This is not surprising, as it is true for other disorders (eg, major depression). Manic patients who rapidly cycle can be especially difficult to treat with medication. Studies show that ECT may be helpful in this population.23

**SCHIZOPHRENIA**

In schizophrenia, ECT is usually used only after medications have been unsuccessful. The critical clinical question, therefore, regards its use in medication-resistant schizophrenic patients. Patients with a shorter duration of illness who experienced an abrupt onset are more likely to respond to ECT than patients with chronic disease. Catatonic type schizophrenia is more likely to respond favorably to ECT as well. Although patients with unremitting, chronic schizophrenia are least likely to benefit from ECT, Fink and Sackei24 argue that such patients should not be denied a trial of ECT because they have a low likelihood of responding to alternative treatments as well. ECT is an efficacious treatment for schizophreniform disorder and schizoaffective disorder.7

- Is a combination of antipsychotic drugs and electroconvulsive therapy indicated in schizophrenia?

**Combination Therapy**

The combination of ECT with an antipsychotic agent seems to be more effective than ECT alone. ECT appears to be safe when administered in combination with traditional and atypical antipsychotic medications, including clozapine. One recent study found that the
combination of ECT and antipsychotic medication was more effective as continuation therapy than either treatment alone in patients who responded to the combination treatment in the acute phase.²⁵,²⁶

MENTAL DISORDERS DUE TO MEDICAL CONDITIONS

The APA Task Force notes that ECT should be used in treating these conditions in patients who are intolerant or resistant to more standard medical treatments and who require an urgent response.⁷ Evidence for this use exists primarily at the level of case reports and has included illnesses such as mental syndromes secondary to lupus erythematosus,²⁷,²⁸ catatonia secondary to medical conditions,²⁹ delirium, and some chronic pain syndromes with concomitant mood disorders.³⁰

RELATIVE CONTRAINDICATIONS TO ELECTROCONVULSIVE THERAPY

As the practice of ECT has become more refined, it has become the case that there are no absolute contraindications to ECT. The decision to employ ECT is always based upon a weighing of risk versus benefit and the particular clinical indication for which ECT would be used. Special precautions may be necessary to administer ECT to patients who pose a particular risk because of the physiologic changes that occur with ECT.

As the electrical stimulus is applied to the patient, there is an initial vagal and intense parasympathetic outflow that causes transient bradycardia and potentially asystole. During this period there is a decrease in cardiac output with potential hypotension. As the seizure develops, sympathetic outflow occurs. Tachycardia and hypertension then ensue. During the clonic phase of the seizure, cerebral blood flow and blood-brain permeability increase. The seizure causes some cerebral edema, and intracranial pressure rises.

Special considerations include the use of medications before, during, and after ECT to control these physiologic changes, or changes in ECT technique itself. Table 3 lists some conditions that require additional patient monitoring. Other medical conditions that may require specific adjustments when using ECT are listed in Table 4.

**ADMINISTRATION**

PRELIMINARY EVALUATION

Any patient considered for ECT should be evaluated by both an ECT psychiatrist and an anesthesiologist qualified to administer anesthesia for ECT. These evaluations should consist of an assessment of any prior ECT, the indications for ECT, a thorough medical history, and a physical examination with attention paid to the teeth and mouth. Both of these evaluations should address risk factors and suggest any additional evaluations, changes in prescribed medications, or modifications to ECT technique.

- **What laboratory tests should be obtained before administering electroconvulsive therapy?**

  No specific laboratory tests are required for ECT. At a minimum, it is prudent to obtain a complete blood count, serum potassium and sodium levels, and an electrocardiogram (ECG). A pregnancy test should be considered in women of child-bearing age. Because muscle relaxation diminishes the chance of fracture in the current practice of ECT, radiographs of the spine are not routinely performed as part of a preliminary workup. They might be indicated, however, in patients with a preexisting history of spinal disease or trauma. If preliminary spinal films are done, equal consideration should be given to obtaining them post-treatment. EEGs, computed axial tomography scans, or magnetic resonance imaging scans are not required preliminary laboratory tests but should be considered if a brain abnormality is suspected. Pseudocholinesterase testing is not recommended unless a patient has a family history or past personal history suggesting a pseudocholinesterase deficiency. It may be necessary to involve a

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**Table 3. Conditions Associated with Increased Risk in Electroconvulsive Therapy**

Unstable or severe cardiovascular conditions (eg, recent myocardial infarction, unstable angina, poorly compensated congestive heart failure, clinically significant cardiac arrhythmias, severe valvular cardiac disease)

Aneurysm or vascular malformation that might be susceptible to rupture with increased blood pressure

Increased intracranial pressure, as may occur with some brain tumors or other space-occupying cerebral lesions

Recent cerebral infarction

Pulmonary conditions (eg, severe chronic obstructive pulmonary disease, asthma, pneumonia)

Patient status rated as ASA level 4 or 5


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6 Hospital Physician Board Review Manual
variety of consultants in the preliminary evaluation of a patient, depending upon a patient’s concurrent medical illnesses.

INFORMED CONSENT

Informed consent should be obtained as part of the preliminary evaluation. Informed consent for ECT is obtained in the same manner as other consents are obtained and documented in routine medical care. Care must be taken to adhere to any unique state regulations regarding the practice of ECT. If questions exist regarding the patient’s capacity to participate in an informed consent process, they should be addressed in accordance with local guidelines, regulations, and laws. Because ECT involves a series of treatments, consent is usually obtained for the entire series, rather than for each individual treatment. Some practitioners specify a maximum number of treatments. If further treatment is required, a new consent should be obtained. It is important that patients understand they may withdraw consent at any time, so that consenting to a series of treatments in no way abridges their right to change their mind at any point in the series. The ECT practitioner should review this matter with patients at each and every treatment session. The APA Task Force report recommends that consent to ECT include at least the following items:

- The reasons for ECT and who is recommending it
- A review of treatment alternatives
- A description of the procedure, including times and location
- Electrode placement options with specific recommendation for the patient and reasons
- A discussion of the number of treatments that are likely to be required, and the maximum number before formal reconsent is necessary
- A statement that there is no guarantee that ECT will be effective, just as with any other medical procedure or treatment
- A review of the relapse risk and the need for some type of continuation or maintenance treatment
- Adverse effects of the treatment, including effects on the cardiovascular and central nervous systems (eg, cognitive effects that may be transient or chronic) and possible death
- A consent for ECT includes consent for any emergency treatment that might be needed in the course of the treatment
- A description of behavioral restrictions that might be necessary through the course of ECT
- An offer to answer questions at any time or provision of names of people who can answer the patient’s questions
- A statement that consent can be withdrawn at any point

TREATMENT SETTING AND TECHNIQUE

ECT is administered on both an inpatient and outpatient basis. The choice of inpatient or outpatient

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### Table 4. Adjustments for Specific Medical Conditions in Electroconvulsive Therapy

<table>
<thead>
<tr>
<th>Condition</th>
<th>Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epilepsy</td>
<td>Epileptic patients may be at a slightly increased risk for prolonged or spontaneous seizures during ECT. Anticonvulsants should be adjusted to the lowest necessary dose during ECT.</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Electrically induced seizures have a hyperglycemic effect. Also, since patients are typically NPO prior to ECT treatments, the glucose should be carefully monitored in this fasting state. Adjustments may need to be made to hypoglycemic agents, including insulin.</td>
</tr>
<tr>
<td>Hyperthyroidism</td>
<td>ECT increases the risk of thyroid storm in patients with hyperthyroidism. Endocrinologic consultation should be obtained and β-blocking agents should be used during ECT.</td>
</tr>
<tr>
<td>Osteoporosis</td>
<td>Radiographs of the spine should be obtained before and after ECT. An increased dose of muscle relaxant may be considered.</td>
</tr>
<tr>
<td>Glaucoma</td>
<td>ECT causes a transient increase in intraocular pressure, but there are no known cases of complications from ECT in patients with narrow angle glaucoma. Glaucoma medications are administered before each treatment for these patients. Demecarium and echothiophate should not be used during ECT because they may greatly prolong the effects of succinylcholine.</td>
</tr>
</tbody>
</table>

ECT = electroconvulsive therapy; NPO = nothing by mouth.
treatment depends on prior ECT history, medical factors, and the lethality or morbidity of the patient’s psychiatric condition. It is possible that one might begin ECT as an inpatient and then switch to outpatient treatment during the course of treatment.

- **What equipment is necessary in the administration of electroconvulsive therapy?**

The ECT suite should be equipped to monitor heart rate, ECG, blood pressure, and pulse oximetry continuously. Equipment should be at hand to provide advanced cardiac life support immediately, should the need arise. The treatment area should also afford management of any medical complications that occur (e.g., hypertension, status epilepticus). The patient should be supplied 100% oxygen to breathe before and during treatment. The patient’s airway is typically maintained with a mask and bag, but there should be provision for rapid endotracheal intubation should any airway compromise occur during treatment. Some patients, particularly obese patients who represent American Society of Anesthesiologists level 4 or 5 risk, may be routinely intubated for ECT. Patients with severe gastroesophageal reflux or pregnant patients may also be intubated if there is an increased risk for aspiration.

- **How is the patient’s seizure monitored?**

During treatment, the patient’s seizure is monitored in 2 ways: by EEG measurement and by watching the motor seizure in an extremity. A blood pressure cuff is typically applied to an arm or above an ankle and inflated to higher than systolic blood pressure prior to infusion of the paralyzing agent. The hand or foot distal to the inflated cuff remains unparalyzed, and both the tonic and clonic morphology of the seizure and its duration can be observed and directly timed. If unilateral ECT is administered, the cuff should be placed on the side ipsilateral to the stimulus electrodes. This provides evidence that the seizure has generalized.

Intravenous access is obtained to administer anesthesia. The anesthesia for ECT usually consists of a short-acting barbiturate to put the patient to sleep. Methohexital is one of the most popular agents because of its relatively brief half-life. The choice of agent is weighed against effects on seizure threshold and duration, arrhythmogenicity, ease of wakening, or a medical contraindication to barbiturate use (e.g., porphyria). Etomidate is an alternative to methohexitol in patients who have brief, abortive, or no seizures at all when the stimulus is delivered.

The patient may be premedicated with an anticholinergic to minimize the risks of bradyarrhythmias or asystole during the first seconds of the seizure. Bradyarrhythmias may also develop in the postictal period. Any number of other medications may be given prior to the ECT or during the treatment, once the seizure has begun, to treat concomitant medical or physiologic problems.

Once the patient is asleep, a paralyzing agent is infused, typically succinylcholine, which is a depolarizing agent. Considerations that can influence choice of the paralyzing agent include pseudocholinesterase deficiency (very rare), history of NMS, or the possibility of hyperkalemia, all of which would direct the use of a nondepolarizing paralyzing agent. Once fasciculations are observed in the toes, the patient’s paralysis should be assessed. The degree of paralysis can be assessed by using a pulse stimulator or by observing the extinction of the Babinski reflex.

At this point, a bite block is placed between the patient’s upper and lower teeth. It is imperative that the patient be well oxygenated prior to passing the electrical current, and oxygenation should continue throughout the seizure and until the patient begins to breathe again without assistance. Hyperventilation prior to stimulating the patient is absolutely essential to successful treatment. Hyperventilation with 100% oxygen reduces the partial pressure of arterial carbon dioxide, lowering seizure threshold. This facilitates seizure elicitation at lower electrical energies and allows for better propagation of the seizure as well. Prior to inducing the seizure, the patient’s chin should be manually lifted to keep it in tight opposition to the bite block.

Once the patient is asleep, paralyzed, well oxygenated, hyperventilated, properly monitored, fitted with the bite block, and chin supported, the electrical stimulus is delivered. The seizure begins almost immediately, develops into the tonic phase, then fairly quickly evolves into the clonic phase and then ends. The timing of the motor seizure begins with the first movement of the cuffed extremity and ends when the clonic phase ceases. The electrical seizure is measured by 2 EEG electrodes that were placed as part of patient preparation. The consensus now is that an adequate seizure needs to last at least 20 seconds by motor measurement and 25 seconds by EEG measurement. Most patients awaken within minutes of the seizure and then are recovered in a monitored environment until fully awake.

**ELECTRODE PLACEMENT**

- **What is the effect of electrode placement in electroconvulsive therapy?**

One of the most intensively scrutinized questions in modern ECT practice is the question of bilateral or
unilateral electrode placement. Both the electrode placement and the amount of current delivered are critical to treatment efficacy and memory side effects. Unilateral ECT causes fewer cognitive side effects, but bilateral ECT typically has had better efficacy.

In a recent study, Sackeim et al demonstrated that markedly suprathreshold (defined as 6 times threshold) right unilateral ECT was equal in efficacy to moderately suprathreshold (defined as 1½ to 2½ times threshold) bilateral ECT, while still having a more favorable memory side effect profile compared to bilateral ECT. Markedly suprathreshold right unilateral ECT retains significant cognitive advantages over bilateral ECT regarding the breadth, magnitude, and persistence of adverse cognitive effects. When treating major depression, the modern ECT practitioner now chooses between bilateral ECT, using no more than moderately suprathreshold energy (defined as 1½ to 2½ times threshold), and right unilateral ECT, using moderately to markedly suprathreshold energy (defined as 2½ to 6 times threshold).

When bilateral electrode placement is used, the electrodes are typically applied in a bitemporal fashion (Figure). When unilateral electrode placement is used, the preferred positioning is the d’Elia configuration, as shown in the Figure.

FREQUENCY AND NUMBER OF TREATMENTS

The decision about the frequency of treatments is based on the desire to produce the greatest and quickest treatment benefit with the least side effects. The more frequently ECT is administered, the more likely a patient will experience greater cognitive side effects, even if transitory. Some patients may even develop delirium. Dense cognitive side effects or delirium may make it difficult to determine the improvement in target symptoms from treatment to treatment, and hence complicate the determination of the appropriate endpoint of treatment. It may be true that there is a consolidation period after each treatment, so that a minimum interval needs to elapse in order to recruit the fullest effect of a given ECT treatment. In the United States, most patients receive 3 ECT treatments per week, spaced evenly apart. However, it has been shown that twice weekly ECT produces fewer cognitive side effects with equal treatment efficacy, although more time may be needed to complete treatment.

• How many treatments are necessary?

The number of treatments required for a given illness varies from patient to patient. Treatment duration also varies from diagnosis to diagnosis, with major depression usually responding in 6 to 12 treatments; acute mania may respond in fewer treatments, and schizophrenia frequently requires more than 6 to 12 treatments. The clinician should identify target symptoms at the outset of treatment. Treatment is continued until all of these symptoms are successfully treated, or until no evidence of change is noted between successive treatments. If a patient plateaus and continues to show significant target symptoms, consideration should be given to changes in technique, such as electrode placement, stimulus energy, or concurrent medications.

USE OF MEDICATIONS DURING TREATMENT

The patient’s medications should be reviewed to determine which drugs need to be stopped, which continued,
and if continued whether an altered schedule is necessary to optimize ECT. Medications that help manage the physiologic changes associated with ECT may be given before the treatment. Such agents include antihypertensives, antianginals, antiarrhythmics (with the exception of lidocaine and its derivatives), antireflux agents, bronchodilators (except theophylline), glaucoma medications (except long-acting cholinesterase inhibitors), and corticosteroids. Some medications, such as diuretics, should be withheld until after ECT treatment is completed. Hypoglycemics may be given in split doses before and after each treatment. In general, psychotropic medications are withheld until after treatment, particularly anticonvulsants, benzodiazepines, and lithium. Table 5 summarizes some specific medications and recommended use or discontinuation during ECT.

**CONTINUATION AND MAINTENANCE THERAPY**

Although ECT is extremely useful in treating acute episodes of illness, it confers no advantage in preventing relapse or recurrence of illness. Therefore, it is imperative that a patient who is treated with ECT receives some type of continuation therapy upon completion of the acute course of ECT. This continuation therapy may consist of ECT, medication, or a combination of the two. It is also possible to taper ECT while initiating pharmacotherapy, treating with ECT at some reduced schedule (eg, once per week) for a few weeks until the pharmacologic agent is titrated to a full therapeutic dose.

- **When should the practitioner consider electroconvulsive therapy as part of a prophylactic strategy?**

Any patient who responds to ECT may be considered for ECT as continuation therapy. ECT alone, or a combination of ECT and pharmacotherapy, may be especially compelling when the patient has failed to respond to pharmacotherapy alone for treatment of the index illness episode, or when the patient has relapsed while on pharmacotherapy. Other indications for continuation ECT include patient preference or situations in which ECT is safer than the side effects associated with pharmacotherapy.

Continuation ECT is typically administered on an outpatient basis. There is no standard regimen for this treatment. Continuation therapy begins immediately after completing an acute course of ECT with weekly or more frequent treatments, and then slowly decreases in frequency of treatments until perhaps 1 treatment is given per month. The tapering of frequency typically occurs over weeks to months. Patient response, the appearance of prodromal signs or symptoms suggesting relapse, and side effects (including cognitive impairment), are all used by the ECT practitioner in determining the frequency of treatments and the duration of continuation or maintenance ECT.

By definition, any treatments lasting 6 months beyond remission are maintenance ECT. There are no data to guide the clinician in determining how long maintenance ECT should continue past remission of the index episode. Studies of pharmacotherapy indicate that medication maintenance in major depression can decrease recurrence rates out to at least 5 years after an episode. Factors that guide the ECT practitioner in continuing or discontinuing maintenance therapy are essentially the same ones used to determine how continuation therapy should be administered. Rapid discontinuation of maintenance therapy is fraught with the potential for recurrence of illness. No evidence indicates that a lifetime maximum number of ECT treatments exists.

**SUMMARY**

Modern ECT is safe and effective for a variety of indications including certain mental illnesses (especially major depression and mania) and some mental disorders due to medical conditions. Certain medical conditions also respond to ECT, particularly Parkinson’s disease. There are no absolute contraindications to ECT, although medications or technique may need adjustment to maximize the safety of ECT. When administered for major depression, markedly suprathreshold electrical stimulus energy with right unilateral d’Elia electrode placement provides efficacy equivalent to that of bilateral ECT but with less cognitive side effects. It is assumed that this holds true for other diagnostic indications as well, although this has not been proven. The risks of ECT are essentially the risks of anesthesia, except for cognitive side effects, which remain the most problematic aspect of modern ECT treatment. Most objective studies fail to show enduring cognitive impairment, but some patients report persistent loss of autobiographical memories in the years preceding their illness and treatment, as well as difficulty with new memory formation.

The mechanism by which ECT exerts its healing effects is not yet known. A myriad of changes occur in response to ECT, and these are being exhaustively catalogued by researchers. They include changes in neurotransmitters, receptors, peptides, cerebral blood flow, neural plasticity, and seizure threshold, among
others. Which of these actions are necessary and sufficient for ECT to exert its therapeutic effect is not known. The brisk pace of research in ECT has enabled clinicians to refine the technique of administration, offer greater efficacy with fewer side effects, and provide safe and effective treatment to patients.

**REFERENCES**


### Table 5. Recommended Medication Changes in Electroconvulsive Therapy

<table>
<thead>
<tr>
<th>Medication</th>
<th>Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Theophylline</td>
<td>Theophylline has been linked to status epilepticus during ECT. It should be stopped, or reduced to a lowest possible dose. Newer agents do not appear to have the same risk for status epilepticus.</td>
</tr>
<tr>
<td>Lithium</td>
<td>Lithium may increase the risk for delirium or prolonged seizures during ECT. If lithium is continued during ECT, its dose should be reduced and levels maintained in the low- to mid-therapeutic range.</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>Benzodiazepines raise seizure threshold and should be avoided as much as possible during ECT. If a benzodiazepine is needed it should have a relatively short half-life and be withheld at least 8 hours before an ECT treatment. Flumazenil can be used at the time of treatment to reverse the benzodiazepine. Care should be given to avoid withdrawal effects in the postictal state.</td>
</tr>
<tr>
<td>Anticonvulsant medications</td>
<td>Doses of anticonvulsants may be decreased prior to ECT, or one could adjust anticonvulsant dosages only if difficulty arises in obtaining a seizure with adequate expression and therapeutic properties. When prescribed for a seizure disorder, anticonvulsants should not be administered prior to an ECT treatment session, but given afterwards. Because ECT has anticonvulsant properties, the anticonvulsant dose may be reduced during an ECT course.</td>
</tr>
<tr>
<td>MAOIs</td>
<td>The bulk of literature actually shows that MAOIs are safely prescribed in the setting of general anesthesia. However, many practitioners will advocate stopping MAOIs 7 to 14 days prior to any anesthesia. Selective MAOIs are not likely to pose a risk during ECT, but there is little data on their concurrent use in ECT.</td>
</tr>
<tr>
<td>Reserpine</td>
<td>Reserpine should not be given with ECT; concurrent use with ECT has resulted in death.</td>
</tr>
</tbody>
</table>

ECT = electroconvulsive therapy; MAOI = monoamine oxidase inhibitor.