Chapter 3

AN EVIDENCE-INFORMED MODEL FOR THE MODERN PRACTICE OF ELECTROCONVULSIVE THERAPY

Amer M. Burhan*, Sarah Jarmain and Verinder Sharma

Regional Mental Health CareLondon/St Joseph’s Health Care, London
Department of Psychiatry, Western University, London, ON

ABSTRACT

After nearly a century of use and despite its many controversies, Electroconvulsive therapy (ECT) continues to be practiced and remains a viable treatment option in modern day psychiatry. Arriving on the scene in the 1930s when limited therapeutic options were available, ECT transformed psychiatry and created hope for the severely mentally ill. Unfortunately, it rapidly was seen as a panacea and was used for a wide variety of psychiatric disorders, many of which did not respond to it. The reputation of ECT suffered due to its inappropriate application and a negative public portrayal of the treatment by the anti-psychiatry movement and in Hollywood movies. The arrival of effective psychotropic medications created an exaggerated hope that ECT and

* Corresponding author: Amer M Burhan, MBChB, FRCPC (Psychiatry and Geriatric Psychiatry); Clinical Lead, Electroconvulsive Therapy and Neuromodulation; Assistant Professor and Chair of the Division of Geriatric Psychiatry; 850 Highbury Ave, P.O.Box 5532, Stn. B; London, ON, N6A-4H1.
other “invasive” procedures used to treat the severely mentally ill would be eliminated. However, ECT continues to be practiced – it stood the test of time because evidence suggests that there is no other treatment in psychiatry that approaches its efficacy in the management of certain psychiatric illnesses like severe depression. Modern day psychiatry is turning back to ECT when needing a quick response or faced with the history of medication resistance. However, ECT is not always practiced in accordance with available treatment guidelines and many health centers do not even have a policy for the safe and evidence-informed practice of ECT. In this chapter, we summarize the current evidence for the use of ECT in psychiatric illnesses, including benefits and risks, and outline a model of ECT service delivery informed by current published evidence and consensus guidelines.

INTRODUCTION

Electroconvulsive therapy (ECT) is one of the most demonized currently practiced interventions in medicine. Applying electrical current to treat mental disorders is perceived by the public quite differently from applying electric current to revive the heart despite the fact that the former is a very effective intervention while the later has limited success rate. Hollywood movies have portrayed ECT in a variety of ways but have trended towards the negative as depicted in movies such as “One Flew Over the Cuckoo’s Nest”. In that movie a rebellious sociopath was treated with ECT more or less as a punishment and he was “zombified” as a result! [1].

Psychiatry is not without blame when it comes to the negative image of ECT. In addition to using ECT as a method of punishment and control in some settings, a prominent academic psychiatrist out of McGill University in Montreal used ECT in the infamous “depatterning” therapy where several sessions of ECT were given in the same day to induce an organic mental state and reset the brain [2, 3]. Despite the issues surrounding the perception of ECT by the public, and the fervent anti-ECT movement, ECT continues to be practiced in modern day psychiatry. What makes ECT such a resilient treatment? To answer this question we need to look at both the story of the evolution of ECT and the current evidence for its efficacy and risks. There is a large body of literature around ECT including excellent reviews, metaanalyses, book chapters and several books devoted to ECT. The intent for this chapter is not to replicate the already established literature but rather describe a practical, evidence-informed model for ECT that can be used as a template to assist those planning to develop ECT services and will orient
health practitioners and the public to the rationale behind and elements necessary for the ethical practice of ECT in modern day psychiatry. The chapter will briefly describe the history and evolution of ECT, then summarize current evidence regarding benefit and risk of ECT and finally describe an evidence informed model for the modern practice of ECT.

**HISTORY AND EVOLUTION OF ECT**

In this section we summarize the history and evolution of ECT based on reviewing several excellent resources that the reader is referred to for more details [3,4,5,6-9]. ECT is the product of a complex set of circumstances. It came on the tail of several attempts by psychiatrists to find a remedy for mental disorders, a set of illnesses that had no effective treatments at the time. While neuroses were treated in private clinics and spas with talk therapy, hydrotherapy and other tools, major mood and psychotic disorders were typically managed within psychiatric asylums, which were established to house the mentally ill and treat them humanely. By the 1900’s, psychiatric asylums were crowded with hopeless cases of manic depressive illness, catatonic schizophrenia, dementia, general paresis of the insane (neurosyphilis) and other major mental illnesses. The care in the asylums was largely custodial with little chance for remission. Early 20th century witnessed several attempts by psychiatrists to find biological remedies for major mental illnesses. The first breakthrough came from Vienna, when psychiatry professor Julius Wagner-Jauregg inoculated patients with generalized paralysis of the insane with blood drawn from malaria patients resulting in the “febrile cure”. Other physical therapies followed; notably frontal lobotomy performed initially on 20 patients between 1935 and 1936 by neurosurgeon Almeida Lima under the direction of Egas Moniz, a Portuguese neurologist. This procedure, and with little scientific validation, was spread widely by the American neurologist Walter Freedman who performed over 18,000 modified (transphenoidal) frontal lobotomy procedures by the 1950’s. Also in the late 19th and early 20th century, induction of sleep and later prolonged sleep therapy was evolving. This included using several chemical alkaloids such as morphine, hyoscyamine, choral hydrate and barbiturates among other chemicals used in cocktails to induce sleep. In 1933, Manfred Sakel discovered insulin coma therapy, which was initially used to help with morphine addiction, but was also found to help agitation in the mentally ill.
Some of these therapies, like the insulin coma therapy, produced mixed and at times exaggerated results based on limited scientific validation.

The story of using a seizure as a therapeutic intervention in psychiatry started when Ladislas Von Meduna, a Hungarian neuropathologist/psychiatrist, proposed ameliorating symptoms of schizophrenia with a chemically induced convulsion. Meduna was intrigued by the notion that patients with schizophrenia who develop epilepsy show improvement in their psychoses. From his neuropathology work he reported that among patients with schizophrenia, those with epilepsy have a higher count of glia cells than those without, which convinced him further of the concept of therapeutic antagonism between seizures and psychosis. He treated his first patient in January 23, 1934, a 33-year-old man from Budapest who was dealing with psychosis and became catatonic by the time he received the first injection of camphor, a chemical that was known to induce a seizure in those that consumed it accidentally or at will. The patient received 6 injections in total over 2 weeks and was in remission by February 10th. Meduna went on to treat more patients and published his first report on this therapy in January 1935 showing close to a 50% remission rate (10 out of 26 patients treated). Because of the unreliability of camphor in producing seizures, and the long agonizing wait by patients for a seizure to happen, a synthetic alternative (Cardizol in Europe and Metrazol in the US) was used instead. Chemical seizure therapy was used in Europe and even more in the US until the early 1940s when ECT arrived on the scene.

When Italian psychiatry professor Ugo Cerletti was a student in the university of Genoa, he worked on a method of inducing sleep in lab animals using electricity. Some of these animals developed convulsions but these appeared benign and did not result in any major complications. He later became a professor in the university of Rome. Motivated by Meduna’s success, and the difficulties associated with chemical convulsive therapy, he continued to experiment inducing convulsions in lab animals using electricity. However, he was unsure how safe it would be to electrically induce convulsions in humans (something that would improve the process of therapeutic convulsion compared to the unreliable and agonizing chemical induction that Meduna invented). Initially, he placed electrodes in the animal’s oral and anal cavities, which resulted in several fatalities in lab animals due to the passage of electricity through the heart. Cerletti instructed his team members to work on different aspects of seizure and related therapies. Luciano Bini was one of Cerletti’s team and was given the task of investigating the use of electricity to induce a convulsion. Bini was aware that the Rome
slaughterhouses used electricity to induce convulsion in pigs to stun them before slaughter. Using a device he assembled, he was able to induce convulsions in animal dogs by delivering the charge to the temples and these animals survived the convulsions unharmed.

In April 1938, a 39-year old engineer from Milan was admitted to Cerletti’s Rome clinic in a disorganized psychotic state. He was the first human to receive ECT, or “electroshock” therapy as Cerletti et al called it in their first report. The patient received 11 sessions in total and was discharged well from the clinic. Cerletti and Bini did extensive medical and physiological profiling of this patient and they eventually published their “experiment” in a 500 page report in a special issue of Rivista Sperimentale Di Freniatria (Journal of Experimental Psychiatry) in December of 1940. Lothar Kalinowsky, who attended Cerletti’s clinic in the early days of ECT, was instrumental in spreading ECT first to Paris, then England and finally to the United States where he established an ECT service at the New York State Psychiatric Institute (Columbia University affiliated). Other psychiatrists that were instrumental in bringing ECT to America were Renato Almansi who brought Bini’s machine to the United States and collaborated with David Impastato to give ECT in their clinic in New York. ECT brought hope to psychiatry and especially to those treating major mental illness and severe persistent psychoses. The 1940s and 1950s witnessed wide spread use of ECT and brought therapeutic excitement to psychiatrists in psychiatric institutes treating severe mental illness and by 1959 it was considered the treatment of choice for manic-depressive and major depressive illnesses. Initially, ECT was practiced “unmodified”, i.e. without sedation or muscle relaxation. It wasn’t until 1952 that succinylcholine, a muscle depolarizing non-reversible muscarinic receptor blocker, was introduced to the practice of ECT, which reduced orthopedic complications. Soon after general anesthesia was introduced with an intravenous injection of an ultra-short acting barbiturate Methohexidal to eliminate the sense of suffocation patients felt with the paralysis of the breathing muscles.

The resistance to ECT initially came from psychoanalysis, which dominated American psychiatry in the 1940s and 1950s. The idea that a physical therapy can cure mental disorders is conceptually challenging to psychodynamic principles. But the more damaging resistance came from the antipsychiatry movement that intensified in the 1960s. Initially, this movement was not necessarily an anti-ECT movement but rather against the state and psychiatric control over those who didn’t obey the rules. ECT was an easy target because culturally it was associated with severe forms of punishments
like the use of electrocution for the death penalty. The antipsychiatry movement declared ECT as a barbaric treatment that damages the brain and purported that psychiatric hospitals were using it as a punishment for patients who misbehave. The over use of ECT for a broad range of conditions, the use of ECT as a threat for patients who repeatedly challenged authority, and an early poorly designed single author report claiming brain damage secondary to ECT [10] partially confirmed the anti-ECT claim. The Scientology movement in the United States with its wealthy and influential members campaigned against ECT and forced several jurisdictions to limit and in some cases ban the practice of ECT.

![Image of the history and evolution of ECT.

Psychiatry ECT advocates started fighting back in the early 1970s when the Massachusetts State Commissioner of Mental Health in early 1972, and the American Psychiatric Association in 1974, formed a task force on ECT lead by Fred Frankel out of Harvard. Further advocacy came with the efforts of academic psychiatrists such as Max Fink who wrote extensively on ECT and its efficacy. The APA formed another task force in 1987 that published a report in 1990 bringing ECT back to mainstream psychiatry including the reintroduction of ECT into psychiatric training programs. It was established
that ECT is a safe and effective treatment for depression (unipolar or bipolar), mania and psychosis of schizophrenia and that there is no credible evidence of brain damage. The issue with memory loss was also profiled further and it was clear that in the majority of patients’ memory loss is temporary and insignificant when it comes to day-to-day function.

Today, ECT is a core therapy in psychiatry, it is practiced in its modified form under careful medical and psychiatric monitoring and is actively being researched to enhance its efficacy and reduce its cognitive side effects. Figure 1 highlights key points in the history and evolution of ECT from pre-convulsive therapy through the development of ECT and the post-development course.

**CURRENT EVIDENCE FOR ECT**

**Indication and Efficacy of ECT**

Clinical guidelines from the United States, Canada, and the United Kingdom [11-14] agree on the clinical efficacy of ECT as a short-term treatment for:

- Major depressive episode
- Acute psychotic episode of schizophrenia
- Acute mania
- Catatonia

The evidence is the strongest for acute major depressive episodes of different types. Both Unipolar and bipolar depression are responsive to ECT although there is more established evidence for unipolar depression. Therefore, while ECT is among first line options for unipolar depression, it is considered third line to treat depression in bipolar patient unless there is an urgent need for quick response like in cases of acute suicidality or limited intake of fluids and food or in psychotic depression. ECT is second line in treating acute mania but can be used first line in delirious mania [15]. The UK ECT Review Group published a review and meta-analysis in the Lancet in 2003 summarizing the evidence. Based on metaanalysis of six sham controlled trials, including 256 patients (mainly inpatients bellow age 70), the standard effect size (SES) of real compared to simulated ECT is -0.91 (95% CI = −1.27 to −0.54), indicating a mean difference in the Hamilton Rating Scale for
Depression (HRSD) of 9.67 (95% CI = 5.72 to 13.53) in favor of ECT. Despite some methodological concerns, and the fact that ECT was compared mainly to tricyclic antidepressants rather than SSRIs, there is also a clear signal of superiority of ECT compared to pharmacotherapy in the treatment of major depressive episodes of different types. There were 18 randomized controlled trials (RCTs) with 1144 patients in total comparing ECT with pharmacotherapy in the short-term treatment of major depressive episodes. The SES based on pooled data from 13 of these trials was –0.80 (95% CI= –1.29 to –0.29). This translates to a mean difference of 5.2 (95% CI = 1.37 to 8.87) on the HRSD in favor of ECT. All sub-types of major depressive episodes are responsive to ECT and the notion that psychotic or retarded depression being more responsive has not been fully confirmed.

ECT is used in treatment resistant depression, which has been defined in several ways, but the generally accepted definition is failure to respond to successive adequate trials (dose and duration) of two antidepressants from different classes. Although initially it was estimated that 15-20% of patients with depression fall into the category of treatment resistant depression, the STAR-D naturalistic trial showed a rate as high as 50 % [16]. In treatment resistant depression, ECT provides fair efficacy and can help more than half of these patients [17-21].

ECT should be considered as a first line treatment when:

- There is an urgent need for response such as in cases with severe suicidal state, or poor food and fluid intake.
- History of pharmacotherapy resistance or intolerance to pharmacotherapy.
- Medical status requires an urgent response or dictates that medications are not safe.

ECT in special patient populations:

- Pregnancy: ECT is considered relatively safe to treat depression in pregnancy in all trimesters. There is no evidence of teratogenicity or other harm to the fetus. Obstetric consultation and fetal monitoring is recommended and the procedure needs to be done where there is immediate access to obstetrical emergency services [22].
- Elderly: ECT is safe and effective for older adults and is highly utilized in this population [20, 23].
• Children and adolescents: there is very limited evidence to guide the practice of ECT in this population. In general, it is accepted that adolescents may respond the same as adults but careful consideration of the diagnosis, risk and benefit and a second opinion is warranted [24, 25].

Bilateral ECT is more effective than unilateral ECT (effect size -0.32 in favor of bilateral). High electric charge is more effective than low electric charge (mean difference on HDRS of 5.24 in favor of high dose) [26, 27]. When it comes to frequency of ECT there is no significant difference between once, twice or thrice a week but the response might be faster for thrice a week. The trend suggests that twice a week might be the best compromise for elective cases and thrice a week for urgent cases [28]. The evidence suggests that higher electric dose relative to the individual seizure threshold is needed for unilateral lead placement compared to bilateral lead placement. It is estimated that unilateral lead placement dose should be 2.5 times the threshold or higher though more cognitive side effects are likely as the charge increases. In bilateral lead placement, 1.5-2 times the threshold is adequate [29].

**Charge Characteristics and Lead Placement**

The electric stimulus used to induce the seizure in ECT has evolved over the years since Bini used his prototype machine. In modern day practice of ECT the following characteristics are usually specified:

1) Pulse width (PW): this is the width of the rectangular-wave delivered and it ranges from 0.25-2 milliseconds (mSec). In the past a sine-wave stimulus was used, and was found to cause more cognitive impairment due to excessive electric charge being delivered when neurons are refractory to stimulation. The abrupt rise of the rectangular-wave seems to count for higher efficiency compared to the sine-wave, which rises slowly and increases the action potential of neurons. Since the invention of rectangular-wave (or pulse wave) stimulus, the issue with duration or width of the pulse came into focus. It is now established that briefer pulse results in less cognitive adverse events due mainly to less energy needed to induce a seizure. How brief the pulse should be is still a matter of debate and depends on lead placement.
2) Frequency (FQ): measured by number of pulses per second or Hertz (Hz). This is another stimulus parameter that has been considered when it comes to cognitive adverse events. Knowing the characteristics of the normal action potential of neurons help to understand this issue. A frequency above 80 Hz was thought to result in stimulus crowding and may deliver excessive charge, hence increasing the risk of cognitive adverse events [30]. On the other hand, the pursuit of briefer pulse resulted in the need for higher frequency to reach the desired charge at maximum duration of the stimulus. This will be described below to clarify this issue further.

3) Duration: this is the total duration in seconds of the stimulus being delivered as a train of pulses at a certain frequency. Most machines deliver up to an 8 second duration. This is one way of increasing the charge without causing a specific increase in the risk of cognitive adverse events.

4) Energy: measured in Joules, this is not under the operator’s control, as it will vary to maintain the current constant, a desirable feature that will allow more charge to reach brain tissue. Impedance (resistance) affects the energy used to maintain the current constant (according to Ohm’s law: \( E=IR \) where \( E= \) energy or voltage, \( I= \) intensity or current, and \( R= \) resistance or impedance (website: http://www.physics.uoguelph.ca/tutorials/ohm/Q.ohm.intro.html accessed July 25, 14).

5) Current (pulse amplitude): measured in Amp; this is kept constant and it ranges from 0.5-0.9 Amp (or 500-900 mili-Amp) depending on the machine used.

6) Charge: this is the sum of electricity delivered and is measured in milicolomb (mCol). This can be calculated using the following formula:

\[
\text{Charge (mCol)} = \text{PW (msec)} \times \text{current (Amp)} \times \text{frequency (Hz)} \times \text{duration (second)} \times 2
\]

The reason for multiplying the total by two is that the stimulus in modern day machines is bi-phasic. For a detailed discussion of the stimulus characteristics, and future directions, see Peterchev et al 2010 [31].

There are three main lead placements for ECT electrodes in modern day practice [32]:
• Bitemporal (BT): the center of each lead is placed 2-3 cm above the midpoint connecting the outer canthus of the eye to the tragus of the ear in each side
• Bifrontal (BF): the center of each lead is placed 4-5 cm above the outer side of the canthus of the eyes
• Unilateral (UL): one lead is placed similar to bitemporal lead on the temple and the other is 2-3 cm lateral to the vertex. Most commonly a right unilateral placement is used (RUL) given that the right brain is the non-dominant side in vast majority of people this can be reversed for rare occasions of “true” right brain dominant individuals, when left unilateral is used. Methods to establish brain dominance are available including “handedness” questionnaires, but in general it is safe to assume left sided brain dominance in all right handed people and the vast majority of left handed people. As such, starting with RUL placement as a default option is relatively safe. In cases that significant confusion result from RUL placement treatment, a trial of left unilateral placement (LUL) would be warranted.

The largest amount of literature is available for BT lead placement, which is the classic placement for ECT. This is followed by RUL placement and finally by BF placement.

A recent large study compared the three placements and found that they are equally efficacious, with BT placement being faster acting; cognitive profile was not significantly different between placements. Of note, this study used electric stimulation that is 6 times the seizure threshold for RUL placement, did not specify the pulse width used for the different lead placements, and was under-powered for cognitive outcome, which limits conclusions regarding cognitive advantage between lead placements [33]. Other studies that addressed the issue of lead placement and stimulus characteristics include a study by Sackiem et al that showed that RUL placement at 2.5 times the seizure threshold might be as efficacious as BT with less cognitive side effects [29].

This was followed by another study showing that ultra brief pulse right unilateral treatment (UB/RUL) is especially effective and memory sparing [34]. Cognitive advantage was confirmed by Loo et al but with some evidence of slower onset of response in UB/RUL [35].

Please refer to figure 2 for illustration of electric stimulus characteristics and lead placement.
Figure 2. ECT parameters including stimulus characteristics and standard lead placement options. A) Sine-wave stimulus used in older ECT machines. It delivered excessive amount of electrical charge and caused more cognitive adverse effects. B) Rectangular-shape stimulus used in modern ECT machines delivers more efficient stimulus in “pulses”. Briefer pulse is considered more cognitive sparing. C) Bitemporal (BT): center of each lead is 2-3 cm above the midpoint between the outer canthus and tragus; Bifrontal (BF): Bifrontal: center of each lead is 4-5 cm above the outer canthus; Unilateral (UL): center of temporal as in bitemporal, second lead is 2-3 cm lateral to the vertex.

The goal for ECT practitioners is to produce symptomatic relief with the least amount of risk including the risk for cognitive adverse effects. As such, there are several factors that affect the decision to choose one lead placement over the other, and with the choice of lead placement comes the choice of stimulus characteristics that are suitable for that lead placement. Therefore we have adopted the following strategy:
• For cases where there is an urgent need for symptomatic relief outweighing the risk of cognitive adverse events:
  • We start with BT lead placement at 150% seizure threshold, or at the half age method if seizure threshold determination is deemed risky or undesirable (cardiovascular risk or risk of delaying benefit)
  • After a few treatments and when the clinical condition improves enough, we establish the seizure threshold and treat with UB/RUL lead placement at 250% the seizure threshold and based on clinical and EEG response
  • Elective cases normally start with UB/RUL at 250% individual seizure threshold with subsequent adjustments based on clinical and EEG response.

Figure 3 outlines starting parameter strategy

![Figure 3. Parameter selection for ECT.](image)

**Risks of ECT**

ECT can have psychiatric, cognitive, medical and anesthesia related risks.
Psychiatric risks: this includes pre-treatment insomnia and anxiety, risk of manic switch in bipolar disorder patients treated for depression, and post-ictal agitation. Risk of suicide was also highlighted in patients with mood disorders receiving ECT but further study is needed to clarify selection bias issues like severity and bipolarity [36, 37].

Cognitive risks: adverse effects of ECT will be discussed in more detail in a separate section but includes immediate post-ictal confusion, and anterograde and retrograde amnestic symptoms (which are usually short term but in a small percentage of cases can be long term).

Medical and anesthesia related risks: to understand this issue we need first to explain the physiological changes associated with ECT and the agents used for anesthesia, and how these might interact with medical risks [38, 39].

Risks from the delivery of ECT:

- Electric charge risk: this can be increased with skull defects, communicating gel or other inadvertent shunting of the electric current that impose electric shock or fire risk. Implanted electric devices such as a deep brain stimulator, cardiac pacemaker and the like need to be taken into account to avoid short circuits.
- After delivery of the charge, and before the occurrence of a generalized seizure, there is an initial parasympathetic phase when bradycardia and even asystole can happen. Usually this is short lasting unless there is a pre-ECT cardiac illness that can exaggerate this response (e.g. atrial-ventricular block). Repeated non-convulsive stimuli can increase this risk further.
- With the occurrence of a seizure, a surge of norepinephrine in the brain and epinephrine from the adrenal gland happens, which will create a cardiovascular response including stimulation of Beta1 receptors in the head (resulting in tachycardia), and alpha-receptors in the blood vessels (resulting in vasospasm). The result is transient tachycardia and hypertension. This is usually benign but can become a concern in those with pre-ECT hypertension, cardiac arrhythmia or ischemic heart disease. It can be modified with beta and alpha blocking agents.
- There is an increase in cerebral and ocular blood flow and increase in intracranial and intraocular pressure.
- There is increased secretions and increased intra-abdominal pressure.
Medical conditions that increase the risk from ECT include:

- Obesity, chronic obstructive pulmonary disease (COPD), ischemic heart disease (especially recent myocardial infarction), recent stroke, increased intracranial pressure, increased intraocular pressure (glaucoma), fragile bones, loose teeth, hereditary anesthesia issues (malignant hyperthermia, pseudoacetylcholine esterase deficiency). Every effort is made to identify prior risks and try to mitigate them to reduce risk from the procedure.

- Mortality rate is thought to be very low for ECT procedure. Old estimates puts it at 4 deaths per 100,000 treatments, which is close to the risk of anesthesia induction alone. The rate of mortality likely declined steadily with the improvement in medical screening and anesthesia practices [40].

Anesthesia for ECT involves induction of brief deep sleep with an intravenous general anesthetic followed by induction of brief generalized muscle paralysis. This imposes risks of hypoxia, hypercapnea (carbon dioxide retention), and aspiration. Special attention is given to the airway, ease of intubation and the risk of cardio-pulmonary failure. Patency of upper airways, air entry to lungs, body mass index, gastro-esophageal reflux risk, “nil per os” (NPO=nothing per mouth) status are important issues to take into account before ECT.

For induction of sleep, agents that are intravenous, fast acting and have the least effect on the seizure induction and quality are preferred. Agents used for this purpose include:

- Methohexital: a barbiturate anesthesia induction agent; has the longest track record among anesthesia agents for ECT; 0.5-1.5 mg/kg; least suppressant to the seizure; but unfortunately not always available.
- Thiopental: a barbiturate anesthesia induction agent, 2-3 mg/kg; a reasonable alternative to Methohexital but may suppress the seizure more. Some issue with availability recently (e.g. in Canada since 2009).
- Propofol: an anesthesia induction agent of unclear mechanism; 0.5-1 mg/kg; a reasonable alternative to barbiturates but may suppress the seizure more, though the quality of the seizure and efficacy of ECT is maintained with some trend of better cognitive and hemodynamic outcome [41, 42].
- Ketamine: a glutamate blocking agent acting on NMDA receptors; an induction agent; 0.7-2.8 mg/kg, resulting in a longer seizure; has its own potential antidepressant effect in recent studies [43, 44].
Ketamine seems to have an advantage over Methohexital in terms of less seizure suppression and cognitive adverse effects; post anesthesia psychotic states have been reported but seem to be mild and manageable [45].

- Etomidate: induction agent; 0.15-0.3 mg/kg; advantage in prolonging the seizure but with some increased post-ictal confusion compared to Methohexital and Propofol [46].

There are other agents used as adjuvant with propofol such as alfentanil and remifentanil but they can cause significant nausea. Also, with the availability of an anesthesia machine, induction with anesthesia gas such as sevoflurane can be done safely with some cognitive advantage over intravenous agents like thiopental [39].

The most commonly used muscle relaxant for ECT is succinylcholine, a depolarizing acetylcholine analogue. It has the advantage of inducing muscle relaxation quickly (within a minute), which matches the anesthetic induction agent well. Dosage is variable and depends on the risk of fractures but in general around 0.5-1 mg/kg. Risks involved with the use of succinylcholine include anticholinergic effects like sinus bradycardia and asystole; increased intraocular, intragastric and intracranial pressure. Myalgia is common and can be treated with analgesia, but at times a pre-treatment with a small dose of curare can help. Rare but important adverse events with succinylcholine include malignant hyperthermia (tachycardia, hyperthermia, acidosis and hypercarbia), which can occur especially in those with familial tendency or those with muscular dystrophies. Neuroleptic Malignant Syndrome, despite its phenomenological similarity to malignant hyperthermia is not a contraindication for succinylcholine. Another important consideration is a genetic deficiency in pseudocholineesterase, a plasma enzyme that metabolizes succinylcholine and limits the duration of its action. Families with this deficiency tend to have prolonged paralysis, which will require airway and breathing support. For those with contraindication to succinylcholine, curare agents are considered based on anesthesia’s assessment. [47]

**Modified vs. Unmodified ECT**

In most countries around the world, it is accepted that modified ECT is the standard of care. As mentioned in the history of ECT section, it was in the late 50s that muscle paralysis was recommended for ECT to prevent fractures and
reduce the intensity of motor manifestation of seizures induced by ECT. Induction of sleep followed to protect patients from the sense of suffocation [3].

Despite recommendation by the World Health Organization (WHO) to use modified ECT as a standard of practice [48], not all countries around the world follows those recommendations mainly due to limited anesthesia resources. The evidence suggests that unmodified ECT (without anesthesia and induced reversible muscle paralysis) is as effective as modified ECT. The main indication for modified ECT is to reduce fractures, anxiety and fear, post ECT agitation and confusion and stigma from ECT [49].

**Cognitive Issues with ECT:** the cognitive sequelae of ECT have been an area of considerable debate and have contributed significantly to the negative image of the treatment. Anti-ECT groups considered these issues a confirmation that ECT damages the brain. As stated above, there is no credible evidence of brain damage from ECT, but cognitive adverse effects of this treatment remain a concern to patients and the public, practitioners and scientists in the field. Several good reviews and metaanalyses are published on this issue [50-54]. The current evidence suggests the following cognitive effects from ECT:

- Acute reversible confusion usually manifests in the form of disorientation and recovers within 90 minutes on average in the majority of patients. Those who continue to have disorientation after 90 minutes tend to suffer more retrograde amnesia later on [55].
- Acute reversible anterograde (learning new information) memory impairment. This includes difficulty remembering events during the ECT treatment course and soon after and tends to recover within weeks and up to 6 months after discontinuation of ECT.
- Acute retrograde (backward) memory impairment. This includes difficulty in remembering events before the start of ECT and presents in a temporal gradient fashion (more severe impairment for events closer to the start of ECT compared to events further back from the start of ECT); this is also usually reversible within weeks to 6 months after ECT.
- Chronic (persistent change) in memory. Here we have relatively small percentage of patients with one of the followings:
  - Subjective memory impairment: tends to be longer lasting than objectively measured cognitive function and might be related to mood.
• Retrograde: some patients complain about loss of memories formed prior to the start of ECT that lasts even years after the course of treatment. There has been some debate in the literature about this. Rose et al did a review of patient’s experience and reported no less than a third dealing with permanent loss of memory [56]. Recently Bergsholm [57] examined the report by Rose and concluded that the results are seriously flawed given that patients who received bilateral and sine-wave treatment were mixed with those who received briefer pulse and unilateral ECT; what was referred to as “permanent” impairment referred to the gaps in memory created during and for the period before ECT and not as an ongoing problems with memory formation; and that several anti-ECT groups were included in the analysis creating a negative bias towards inflating blame towards ECT.

The mechanism of memory impairment with ECT is poorly understood but does not seem to be related to structural changes in the brain but rather a temporary functional alteration of learning and retrieval mechanism. The medial temporal lobe and diencephalon involved in memory consolidation via long-term potentiation seem to be relevant to this issue. The temporal gradient retrograde impairment supports the possibility that a functional change in the memory consolidation process is at play [53]. Risk for cognitive impairment was investigated in a prospective study over 6 months [58] and showed higher risk with:

• Advanced Age
• Female gender (most likely due to lower seizure threshold)
• Lower pre-morbid IQ
• Bilateral lead placement
• Stimulus characteristics like using sine-wave form

Mechanism of Action

The search for the mechanism of action of ECT is ongoing. There are several proposed mechanisms based on measurable chemical, physiological, and even structural changes in the brain seen in animal models and in post-mortem and in-vivo electrophysiological and imaging human studies. Earlier proposed mechanisms fell out of favor due to the speculative nature (like the
psychoanalytic hypothesis that patients with depression derive benefit from the sense of punishment that counters their inward directed anger). Other unfounded hypotheses include patient’s getting rid of symptoms at the cost of brain damage and/or permanent amnesia. There are several well-documented changes that bring forward more credible proposed mechanisms including:

- **Anti-convulsant mechanism**: there is evidence that with successive sessions of ECT, the brain become more resistant to the induction of a seizure (increased seizure threshold), shorten the seizure and have more slow-waves especially in the frontal areas.
- **Neurotransmitters/neurohormonal**: there is evidence of significant change in serotonin, norepinephrine and dopamine signalling similar to antidepressant therapy but on a larger scale and with the correction of hypothalamic-pituitary-adrenal axis abnormalities
- **Neuroplasticity**: one of the more recent and exciting areas of research is related to the induction of neuroplastic changes such as increased arborization, neurogenesis and firing in animal models of ECT.

For recent conceptual reviews on this topic see [59, 60].

**Cost-Effectiveness of ECT**

Despite the cost of personnel, equipment and medications used for anesthesia with ECT, the efficacy of the treatment might be cost saving in the long run. Few studies have looked at the cost-effectiveness of ECT and compared it to medications and more recent therapies such as transcranial magnetic stimulation (TMS). The report by the Health Technology Assessment Committee commissioned by the National Institute of Health and Clinical Excellence (NICE) indicates that ECT is not more cost effective compared to pharmacotherapy for depression [61]. This report was criticized for focusing only on the cost of the direct management of depression, and not taking into account indirect costs such as lost productivity, lost economic status due to disability or suicide. It also compared average treatment naive patients to those who are treatment refractory patients usually referred to ECT, which questioned the validity of conclusions [62]. There is evidence that maintenance ECT can be quite cost effective compared to maintenance medications, especially when used for old age depression [63]. Comparison between ECT and rTMS is less clear with a report from the US that indicates
that rTMS is more cost effective [64] while another from the UK that considered ECT more cost effective [65]. This issue will likely be clarified as rTMS parameters become clearer. Based on the current evidence ECT seems to be cost-effective, especially for those with treatment resistant depression, and those with severe depression in old age [66].

**AN EVIDENCE INFORMED MODEL FOR ECT**

**The ECT Administrative Structure**

ECT services are typically provided by psychiatric services within general or specialized (or tertiary care) hospitals. Patients who are deemed to be at higher than average medical risk from the procedure are typically treated in a general hospital setting with access to acute medical services and intensive care units. Typically an ECT service would have a manager, a service coordinator (usually a registered nurse), a medical director or lead (typically a psychiatrist with added expertise in ECT) and clerical support. The service can be a stand-alone service with or without other forms of brain modulation, or a joint service among several programs within psychiatry (mood, psychosis and/or geriatric programs). The service needs budget to cover cost of equipment purchase and maintenance, supplies, medications and personnel. Depending on the setting, ECT can generate funds through fee for service or hospital funding from government sources.

The ECT Accreditation Service (ETAS) [67, 68] formed as part of a quality improvement initiative through the Royal College of Psychiatrists in the UK, have developed a set of standards for the administration of ECT that clearly describes the different components of an evidence based ECT service. The Standards are broken down into the following components:

- The ECT Clinic and Facilities
- Staff and Training
- Assessment and Preparation
- Consent and Information Giving
- Anesthetic Practice
- The Administration of ECT
- Recovery, Monitoring and Follow UP
- Special Precautions
We will not replicate the details of the standards within this chapter but instead will highlight some key considerations that we have incorporated into our own ECT service.

ECT Clinic and Equipment

ECT Clinic Design

The ECT Clinic should consist (at a minimum) of three rooms/areas – a waiting room, a treatment room, and a recovery area. Visual and sound separation between the different areas is important, as is the consideration of patient privacy while waiting for and recovering from the ECT procedure. The location should have easy access from both the inpatient units of the hospital, as well as access for ambulatory patients. The treatment and recovery room needs to be of sufficient size to allow for easy movement of stretchers in and out of the space, and access to the ECT, anesthesia and emergency related equipment.

ECT Clinic Equipment

ECT machine and parameters: The function of the ECT machine is to deliver the planned electric stimulus to induce a seizure safely. Since Bini’s first prototype machine there have been several companies that have manufactured and distributed ECT machines.

Modern ECT machines deliver a range of electrical stimuli varying the stimulus characteristics within safety limits, and based on current evidence for the most efficient and memory sparing characteristics. Older machines delivered what is termed the sine-wave stimulus, which was found to deliver excessive charge causing more cognitive difficulties. Modern ECT machines deliver what is termed “rectangular or square-wave” stimulus. The width of rectangular-wave stimulus has been an area of extensive research. It is proposed that “brief” wave (0.5-2 msec.) is effective and memory sparing. Some groups proposed that “ultra-brief” wave (0.25-0.37) gives even further advantages with respect to memory sparing. This is still debatable but there is evidence that at least unilateral lead placement using ultra-brief stimulus maintains benefit, while sparing memory function.

The two most commonly used machines in North America are MECTA machines (MECTA Corp. https://www.mectacorp.com/home.html) and Thymatron machines (Somatics, LLC http://www.thymatron.com/main_home.asp). These machines basically offer similar advantages given that they
both provide constant current, a range of ultra-brief and brief pulse width, an impedance (resistance) test and auto-abort of the stimulus if it is outside the accepted impedance range, and both are equipped with EEG and ECG monitors and print outs. Thymatron machines start with lower ultra-brief pulses (0.25 compared to 0.3 for MECTA) and increase the current to 0.9 Amp across all suites (compared to maximum of 0.8 Amp for MECTA although in 2011 models MECTA introduced 0.5-0.9 Amp current range). Some countries, notably the USA, impose limits on the total charge. This is why the maximum charge for machines marketed in the USA is a 576 mCol charge, while in the UK this can be almost doubled to 1008.

**Anesthesia Machine and Parameters**

It is a standard practice in modern day anesthesia to have a machine that provides detailed monitoring of blood pressure, oxygen saturation, and breathing cycle. Carbon dioxide monitoring is not always required, though useful for those with obstructive lung disease. Oxygen delivery via a mask, a secretion suctioning machine, disposable airway support, and bite blocks are essential to assure safe maintenance of airway and oxygenation and to prevent injury with the seizure.

**ECT Personnel Training, Roles and Responsibilities**

As previously described, ECT has evolved into a highly technical and complex treatment procedure that has demonstrated efficacy in treating specific disorders. Several professional organizations have published position papers and clinical practice guidelines for ECT use in the various patient populations [11-14]. These guidelines have also stipulated that the physicians who prescribe and/or administer this modality of treatment should ensure that their knowledge and practical skills are kept up to date by engaging in continuing professional development and review of the literature pertaining to ECT. Furthermore, they have outlined that the hospital/ institution/ facility and the department heads ensure that the physicians are given appropriate training on an ongoing basis, and to ensure that they have the appropriate credentials/ privileges to undertake this procedure.

Within our ECT service, we have delineated that physicians who administer ECT must meet the following knowledge/skill requirements to be credentialled:
Physicians who administer ECT must have an understanding of the following:

- Anaesthetics and muscle relaxants
- Airway management and oxygenation
- Bite-blocks and nerve stimulators
- Electrophysiological monitoring of heart rhythms and blood pressure
- Management of medical emergencies during ECT

Physicians who administer ECT must be proficient in the following:

- Indications and appropriate selection of patients, including risk-benefit assessments, for ECT
- Consent procedures for ECT (treatment capable and incapable patients)
- Pre ECT work up or preparation of patients for ECT (e.g., medications to hold prior to ECT)
- Types and use of ECT equipment and techniques of ECT administration (e.g. stimulus parameters and dosing, concept of threshold, electrode/lead placements)
- Management of seizures including the monitoring of motor and EEG convulsions during ECT, management of missed and prolonged seizures, and management of an increase in seizure threshold (concept of inadequate seizure) during a course of ECT
- Emergency use of ECT
- Documentation of clinical condition following individual ECT treatments (inter-ECT interval progress)
- Evaluation of therapeutic outcomes and side effects during a course of ECT including:
  - Cognitive changes
  - Management of behavioural complications (post ECT agitation, etc.)
  - Monitoring of clinical efficacy (using objective scales to monitor response such as mood scales)
- Duration of a course of ECT and frequency of treatments (inter-ECT interval)
- Use of Maintenance ECT
- Post-ECT medication management, particularly to prevent relapse and recurrence
In addition to this, in order to maintain these privileges they must:

- Keep their knowledge of ECT up to date (minimum - attend a biannual ECT Workshop or equivalent)
  - Participate in a Pre-test and Post-test Evaluation as part of the Workshop
  - Should maintain a PASS mark on this evaluation
- Under SUPERVISION - Performance of a minimum number of 10 ECT treatments involving at least three different patients annually
- Under SUPERVISION - Performance of a minimum number of three ECT workups, course of ECT and post ECT management
- Attend ECT/ Neuropsychiatry Journal Club which will be held 4 x/year, or demonstrate equivalent level of review of literature e.g. Personal Learning Project
- Must keep a dossier of their ECT profile that should be evaluated annually

Nursing staff who are involved in ECT similarly should go through regular training/updating of their knowledge related to both ECT, assessment and recovery of patients, and emergency management should a critical incident arise.

ECT trainees: in academic centers like ours, medical students, psychiatry and other specialty residents, nursing and other allied health professionals like psychology, and paramedics attend the service for observation and skill training. Psychiatry residents are required to perform ECT assessments, preparation and procedure as part of their curriculum to reach proficiency in the practice of ECT prior to completing their residency.

The particular roles and responsibilities of the ECT interprofessional team are outlined in Appendix A. Standardization of practice, clarity in roles and responsibilities and the incorporation of key practices such as a treatment pause help to ensure that ECT continues to be delivered in evidence based, quality and safe manner.

ECT Process

The ECT service can be divided into three main phases thought they are overlapping:
- Pre-ECT assessment and preparation
- ECT procedure
- Post ECT recovery

Figures 4-7 summarizes the ECT process and responsible personnel

Figure 4. Selection of the appropriate patient for ECT.

Figure 5. Pre-ECT process.
Figure 6. Summary of ECT procedure.

- Confirm NPO, consent, and special medication instructions
- Change patient to gown and place on stretcher
- Bring chart to treatment room with patient

Treatment Panel (Psych)

- Confirm patient identity, consent, treatment number, lead placement, anesthetic agents and dosages, any pre and/or post ECT medications to be given

Setting up equipment (Psych)

- Acquire EEG recording (bilateral 10-20 montages), set stimulus details, monitor patient’s mental state, vital signs and O2 sat, level of sedation and paralysis (after anesthesia is given), assure patient

Setting up equipment (Anesth)

- Acquire vital signs and O2 sat reading, monitor ECG, assure open airway, establish IV access, give general anesthetic then muscle paralysis agent after sleep induction, place bite block, support airway and give patient any pre-ECT medications ordered or needed to keep vital signs stable

Delivery of stimulus

- Once optimum anesthesia and paralysis is achieved, the psychiatrist announce stimulus to be delivered
- Ensure optimum impedance and clearance to avoid current shunting
- Deliver the stimulus fully
- Monitor respiration clinically and through EEG recording while monitoring patient for any medical risk such as syncope, stridor, prolonged seizure etc.
- Announce the end of the seizure

Figure 7. Summary of post-ECT recovery.

- Clear airway
- Adequate breathing effort
- Vital signs stable
- No acute medical changes such as seizure
- No agitation

Immediate recovery (Anesth, RN)

- Awake and regained baseline orientation
- No nausea or vomiting
- No pain
- Back to baseline mobility
- Good urine output
- Free of acute psychiatric symptoms

Secondary recovery (Psych, RN)

- Continue the course? (when and what parameters?)
- Space treatment?
- Discontinue treatment? (what is the follow-up plan?)

Course (Psych, RN)
ECT Procedure: Challenges and Solutions

- **Difficulty inducing a seizure**: there are several factors that can interfere with seizure induction [69]:
  - advanced age
  - male gender
  - skull and tissue density
  - medications like benzodiazepines, hypnotics, anticonvulsants and some anti-arrhythmia agents
  - bilateral lead placement
  - number of ECT treatments (threshold increase with the increase in number of treatment)
  - stimulus characteristics (briefer pulse is better for induction of a seizure)
  - electrolyte imbalance

The inability to induce a seizure results in anxiety, unnecessary exposure to anesthesia risk, bradycardia and asystole and lack of benefit. Effort to augment the chance of establishing a therapeutic seizure starts with correcting factors such as electrolyte imbalance and medications that raise seizure threshold. Other strategies have been investigated including:

- adjustment of anesthesia induction agents (safely lower the dose, add augmentation agent such as remifentanil, use etomidate or ketamine induction)
- changing lead placement to unilateral
- changing stimulus to briefer pulse
- hyperventilating prior to the delivery of stimulus
- pre-medicating with xanthines like caffeine, theophylline and aminophylline

For a more extensive review on seizure augmentation strategies refer to [70]

- **Managing medications during and after ECT**

It is essential to review the patient’s medication list and decide on what needs to be modified before, during and after ECT. The relationship of medications with ECT can fall in one of the following three categories:
Medications that create risk when combined with ECT
2) Medications that affect seizure threshold (amount of electricity needed to induce a therapeutic seizure) or seizure duration
3) Medications that can be helpful with ECT by controlling medical risk factors or potentially facilitating therapeutic response.

The general rule is to continue to administer medications used to treat medical risk factors unless there is a specific caution regarding the combination with ECT. Usually oral medications are given with small amount of clear water up to 2 hours prior to ECT. Some medications can be given intravenously at the time of the treatment by the anesthetist. Considering pharmacokinetic characteristics such as half-life is essential; for example monoamine oxidase inhibitors (MAOIs) need to be discontinued at least 2 weeks before ECT can be given safely due to the potential for malignant hypertension and tachyarrhythmia with the sudden, several folds surge of nor-epinephrine/epinephrine with ECT [11]. Combination of antidepressants like tricyclic antidepressants, selective serotonin or serotonin and norepinephrine reuptake inhibitors (SSRIs, SNRIs), or antipsychotics with ECT to facilitate and/or maintain response/remission needs further validation but it is generally safe [71].

Table 1 summarizes medications and their effect on ECT.

ECT Course Management

- Acute: this is defined as the course of ECT required to treat an episode of mental illness. Number of ECT sessions needed to result in remission of an episode of mental illness varies depending mainly on the syndrome being treated, treatment parameters used and the success in inducing an optimum seizure. Most patients seem to show some response by treatment number 6, though some may respond earlier and others later. In general 8-12 sessions would represent a full course of acute ECT. It is important to make sure that all sessions counted are optimum in quality.
Table 1. Summary of medications effects on ECT

<table>
<thead>
<tr>
<th>Medications that are risky with ECT</th>
<th>Medications that affect seizure threshold or quality</th>
<th>Medications that are helpful with ECT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Med.</strong></td>
<td><strong>Effect</strong></td>
<td><strong>Med.</strong></td>
</tr>
<tr>
<td>MAOIs</td>
<td>Hypertensive crisis</td>
<td>Benzodiazepines</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lithium</td>
<td>Delirium</td>
<td>Hypnotics</td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>Insulin</td>
<td>Hypoglycemia</td>
<td>anticonvulsants</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Choline esterase inhibitors</td>
<td>Prolong paralysis from succinylcholine</td>
<td>Xanthines (Theophylline, caffeine,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>aminophylline)</td>
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<tr>
<td></td>
<td></td>
<td>TCA, SSRIs, SNRIs, Clozapine and</td>
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<td></td>
<td></td>
<td>Bupropion</td>
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<tr>
<td>Choline</td>
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<td>Choline esterase inhibitors</td>
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<td>esterase inhibitors</td>
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<td>inhibitors</td>
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<tr>
<td>Prolong paralysis from succinylcholine</td>
<td></td>
<td>Xanthines (Theophylline, caffeine, aminophylline)</td>
</tr>
<tr>
<td>TCA, SSRIs, SNRIs, Clozapine and Bupropion)</td>
<td>Reduce seizure threshold, facilitate, prolong seizure</td>
<td>Anti-angina Anti-arrhythmia</td>
</tr>
<tr>
<td>Key:</td>
<td><strong>MAOIs= monoamine oxidase inhibitors</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>TCA= tricyclic antidepressants</strong></td>
<td></td>
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<tr>
<td></td>
<td><strong>SSRIs: selective serotonin reuptake inhibitors</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>SNRIs: selective serotonin and norepinephrine reuptake inhibitors</strong></td>
<td></td>
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<tr>
<td></td>
<td><strong>Bronchodilators</strong></td>
<td></td>
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<tr>
<td></td>
<td><strong>Prevent broncospasm, improve air-entry to the lungs</strong></td>
<td></td>
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<tr>
<td></td>
<td><strong>Antipsychotics</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Facilitate and maintain ECT effect</strong></td>
<td></td>
</tr>
</tbody>
</table>
Continuation and maintenance: when a patient responds to an acute course of ECT it is important to consider the issue of continuation and maintenance. The risk of relapse after discontinuation of ECT is very high. Pharmacotherapy following ECT has low chance of maintaining remission [72]. There are no good systematic studies to confirm the details of a continuation or maintenance ECT but it is generally accepted that some patients who repeatedly failed medication maintenance and who responded to ECT have a high likelihood of needing ECT on ongoing basis. Some advocated continuation for several months and try to taper ECT off while others thought that indefinite maintenance is needed. Most of the literature on maintenance ECT is derived from patients with unipolar depression though there are some case reports for bipolar depression patients [73]. For a detailed recent review see Rabheru 2012 [74].

CONCLUSION

ECT continues to have a significant role in treating some forms of mental disorders including acute depression, mania, acute psychosis and catatonia, among other indications such as medication resistant depression. There is a negative perception of the treatment due to several factors including its somewhat indiscriminate use in the past, the use of fixed high dose bitemporal treatment rather than individualizing the dose and lead placement, at times suggesting that it is a treatment option for those who misbehave or resist authority of the hospital, abuse of its use in some centers based on false and unfounded assumptions, and the biased and often powerful anti-psychiatry movement that found ECT an easy target given its apparent invasive nature especially when it was used unmodified in the past.

There was a considerable amount of work that went into validating the efficacy and safety of ECT and since the 1980s there is a resurging interest in the treatment due to its unmatched efficacy for some of the most severe mental health presentations. The issue of cognitive consequences of ECT has received significant attention over the last few decades and although more work is needed, it has provided the field with guidance on how to modify treatment parameters to reduce the risk and severity of adverse cognitive effects of ECT. In our model we balance between the need for sure and rapid response and risk of cognitive adverse events. We also define the roles of team members involved in the delivery of ECT in order to assure clear
accountability to our patients and their families. While other modalities of treatment are evolving such as magnetic seizure therapy, deep brain stimulation and trans-cranial magnetic stimulation, ECT maintains its position in psychiatric treatments. Therefore, a clear and ethical application of the treatment is the responsibility of all health and medical practitioners involved. We hope that this chapter will be used by practicing physicians and trainees to learn about the evidence-informed practical aspects of ECT, and those trying to establish an ECT service to use this model as template.

REFERENCES


APPENDIX A: ECT PERSONNEL ROLES AND RESPONSIBILITIES

1. ECT Psychiatrist:
   - Selection of individuals for the treatment: this will be based on an ECT assessment by a qualified ECT consultant psychiatrist based on a referral from the most responsible physician (MRP) of the individual. The assessment will include the following elements:
     - Review of the data provided by the referring MRP using “ECT referral template” (see Appendix B)
     - Take psychiatric and medical history
     - Confirm psychiatric diagnosis
     - Confirm details of treatment history
     - Confirm completion of appropriate rating scales (Clinical Global Impression Scale, Hamilton Depression Scale-7, Montreal Cognitive Assessment Scale)
     - Completion of the “ECT Assessment Template” (see Appendix C), which include parameter selection.
     - Confirm/obtain informed consent
     - Complete ECT tracking sheet
   - Selection of electrical stimulus parameters: based on individual case needs the treatment can be routine or urgent.
     - Routine: using “ECT Parameter Selection Guide” (see Appendix D), the ECT psychiatrist will try to establish seizure threshold using the recommended level based on age and gender. The default starting stimulus is Right Unilateral Ultra-brief pulse. If the seizure did not occur with the first stimulation, re-stimulation at 10% higher dose can be tried after 40 second, a further re-stimulation with additional 10% increment can be tried if the anesthetist allows based on level of sedation and muscle relaxation of the individual.
     - Once the individual seizure threshold is established, ECT psychiatrist will calculate the dose of subsequent treatment based on the current evidence
Applying ECT: the psychiatrist holds the ultimate responsibility for the ECT procedure. In the treatment suite, the treating psychiatrist have the following responsibilities:

- Confirm valid consent, number of treatment, orders written
- Treatment pause
- Set the parameters on the machine and log the individual’s data into the computer attached to the machine, record on the treatment sheet and apply the leads to the head of the individual.
- A pre-stimulus EEG sample will be acquired, lead applied, and once contact is deemed adequate and safe as indicated by the green indicator on the machine, the psychiatrist will notify the team about the imminent delivery of the stimulus assuring that the patient is in deep sedation with adequately relaxed muscles and that no one is touching the patient. The stimulus will be delivered using remote control equipped leads held by the psychiatrist on the appropriate lead location decided on for the patient. The energy is delivered fully before the leads are removed from the head.
- Seizure monitoring:
  - EEG evidence of a generalized seizure with spikes and waves, in left and right leads, with adequate duration and amplitude and good post-ictal suppression (see Appendix D)
  - Motor seizure will be monitored for tonic-clonic characteristics, rigorousness and duration
  - Vital signs will be monitored for changes in heart rate and blood pressure
  - Recovery of patient from the seizure will be monitored jointly with the anesthetist including recovery of spontaneous breathing and the cessation of the seizure and post-ictal confusion and/or agitation. The
anesthetist will then move the patient with RN to the anesthesia recovery room where vital signs are continuously monitored for 5 minutes.

- Secondary recovery room is staffed with RNs. Alertness, vital signs, comfort will be monitored per protocol.
- Once patient is alert, has stable vital signs, with no nausea or vomiting and is back to baseline mobility he can be discharged by a physician (anesthetist or psychiatrist)
- Patient will be accompanied by a responsible adult

Follow-up of ECT outpatients:

- ECT consultants will be responsible for monitoring the course of ECT for patients accepted by him/her. This follow-up is focused on ECT parameters and is not assuming the outpatient psychiatric care of the patient. As such, reviewing treatment logs to decide of adjusting parameters and lead placement, frequency of ECT and when to terminate ECT will be within the scope of this follow-up. ECT consultants are not required to discharge patients from ECT after each ECT session, this can be done by anesthesia based on medical clearance, and instead the ECT consultant will review treatment logs and EEG and communicate with ECT RN verbal or writer orders to plan next steps.

2. ECT Anesthetist:

- Pre-ECT screening: for medical and anesthesia risks
- Assign a risk level based on medical/anesthesia history, physical examination and investigations.
- Order further investigations or other consultations based on the patient medical status (e.g. general internal medicine for better blood pressure or diabetic control, vascular surgery if aortic aneurysm is present, neurosurgery if there is intracranial mass or vascular anomaly etc.)
- Instruct on pre-procedure preparation, which is mainly nothing by mouth 8 hours prior to treatment but also any
medications to be taken to reduce risk (e.g. beta blockers, pump inhibitors, bronco-inhales)
- Calculate dosage of muscle relaxant and hypnotic.
- Assure adequate sedation and muscle paralysis while maintaining optimum ventilation and oxygen saturation and vital sign stability.
- Assist patient’s airway and breathing while patient is sedated and paralyzed.
- Administer and pre or post ECT medications planned or as the need arises, e.g. if high blood pressure may use an beta blocking agent such as labetalol, if seizure is prolonged may use IV benzodiazepine such as midazolam.
- Assure safe recovery from anesthesia

3. ECT Nurses
   - Ambulatory ECT Nurse:
     - Receive all ECT clinic referrals and ensure all referral documents are complete
     - Book and schedule all Pre-ECT Psychiatric, Medical and Anesthesiology consultations as well as any follow up appointments
     - Book and schedule all treatments
     - Contact patient and advise of date and time of treatment
     - Provide education and support to outpatients and families as required
     - Assemble charts for outpatients and check inpatients chart to ensure completeness
     - Arrange the list of patients for the day of treatment and provide current information on each patient receiving treatment (name, age, type of treatment, previous medications used, number of treatments, special considerations) and make copies for other team members.
     - Facilitate and organize student orientation to the ECT and observation
     - Register and schedules all patients into the system using Cerner
     - Complete the billing for Psychiatrists
     - Assist with ordering supplies
     - Coordinating staff number according to number of outpatient having treatments
• Assist with gathering and completing statistical information
• Ensure that all information on policy and protocols is kept up to date both in manuals and on bulletin boards in collaboration with the leader
• Coordinate with LHSC (ward clerk that book ECT) for high-risk patient to have treatment there. Must have all other work ups completed at RMHC prior to booking the appointment.
• Monitor and check the drug and equipment expiry date on a monthly basis
• Monitor and check the AED on the 4th floor just outside of OHSS on a monthly basis.
• Email booked anesthetists number for following day
• Fax current treatment list to wards
• Call kitchen and order juices

- Treatment Room Nurse:
  • Pick up crash cart, and mask from Health services
  • Post the individual patient identification sheet, for the patient that is receiving treatment and update when needed, revise reference cards (date, number and medication change)
  • Ensure all equipment is available and ready in the treatment room
  • Check all Ensure cords, hoses are in good condition
  • Ensure all gas tanks are turned on
  • Ensure that all safety check are completed on the gas machine if concerns arise bring to the attention of anesthetist
  • Ensure treatment medications and emergency medications are available and current.
  • Set up the treatment at the beginning of ECT and between patients
  • Participate in the treatment pause
  • Clean all equipment between patients

As per the Standard Wiping Protocol for Disinfecting Mobile Patient Equipment please see http://intra.sjhc.london.on.ca/depts/icontrol/ for more information.

All monitoring cords EEG, ECG (both machines) oximeter, Cord for Blood pressure cuff, Cord for electrodes, and Ambu bag
- Provide emergency interventions as required
- Facilitate the orientation of new staff to the area
- Facilitate student orientation and observation to the ECT treatment room
- Assist with making appointments and arranging consultation when necessary
- Assist with ordering of supplies
- Assist with the billing for Psychiatrists
- Make yellow OHIP card for anesthetists and arm bands
- Check patient file day of treatment for order
- Take out anesthetic sheets and pink sheet and place in order of treatment
- Set up anesthetists consults

  - **Electrode Nurse** (same as ambulatory clinic nurse)
    - Assist to ensure that all equipment is available and ready in the treatment room. Ensure that all cords are in good condition. Ensure that those electrodes do not touch to prevent any shock
    - Assist with setting up and cleaning of equipment prior to the start of ECT and between patients.

  As per the Standard Wiping Protocol for Disinfecting Mobile Patient Equipment please see http://intra.sjhc.london.on.ca/depts/icontrol/ for more information.

  All cords form EEG, ECG, (both machines) oximeter, Blood pressure cords, ambu bag
  - Ensure all supplies required for the treatment of each patient are available
  - Bring the patient into the room
  - Apply all monitoring equipment from both the ECT and anesthesia machine (BP cuff, ECG, EEG, oximeter ) applying stimulus and monitoring electrodes, and monitoring vital signs.
  - Ensure the electrodes are appropriate for the type of treatment patient is receiving (flat or concave)
  - Prep the patients head for treatment (cleanse area if needed)
  - Participate in the treatment pause
  - Apply electrodes to the patient when instructed by the Dr., in correct ordered placement, prior stimulus being delivered
  - Ensure that the contact gel doesn’t touch to prevent shock
  - Remove electrodes when stimulus as been delivered
  - Assist Anesthesiologist when needed
– Remove all monitoring devices
– Assist with transfer of patient to the recovery room
– Provide emergency interventions as required
– Facilitate and assist with training of new staff to the area

– **Recovery Room Nurse**
– Ensure the room and equipment are ready for use

Inspect all machines Power cords, cables
Inspect all Oxygen tanks for proper functioning

- DO NOT USE AN ACCESSORY OR EQUIPMENT WHICH SHOWS PHYSICAL DAMAGE. Please notify coordinator. Ensure that arrangements are made for this machine to be check ASAP by Biomedical department. https://www.lhsc.on.ca/priv/bme/index.htm
- Review the crash cart
- Review the recovery room Oxygen therapy practices guidelines
- Review Malignant Hyperthermia with the kit and the protocol and literature provided
- Receive report form Anesthesiologist on each patient
- Monitor patients post anesthetic responsibilities include monitoring of vital signs, pulse oximetry, ECG( if requested by the anesthesiologist), and mental status, administering oxygen and intravenous as needed
- Assist the patient to expel the artificial airway if needed
- Assess vital signs upon arrival and every 5 minutes for a total of 3 checks
- Re-orientate the patient to person, time, and place
- Gently and repeatedly reassure the patient that their treatment is finished
- Discontinue intravenous access
- Provide emergency interventions as necessary
- Determine when to notify anesthetist or the treating physician if concerns arise
- Arrange for the patient to transfer back to the unit or to the ambulatory ECT clinic
- Provide reassurance and support to the patient
- Ensure all documentation is complete
- Clear patient information from the monitor between patients
- Clean all equipment between patients as per the Standard Wiping Protocol for Disinfecting Mobile Patient Equipment please see http://intra.sjhc.london.on.ca/depts/icontrol/ for more information.
All cords from ECG (if used), oximeter, Blood pressure cords, stretchers.

- Clean and tidy recovery room when ECT is completed

4. ECT Clerk: Assist ECT ambulatory nurse in setting up files, contact patients, scheduling and collecting statistics in addition to other office support.

APPENDIX B

ECT Clinic Referral

Thank you for referring your patient to our ECT service.

Please read the following follow-up requirements:

Our ECT services require that the referring physician see their patient weekly while they are receiving ECT. The follow-up should be scheduled a day before or after each treatment to allow for better assessment. Global impression, cognitive and mood scales should be completed and faxed to our ECT services immediately to aid us in making decisions about changes in the course of treatment.

Please complete this referral form and use the scales attached for initial and follow-up assessments.

- Patient diagnoses (list all):
  
  Axis I:
  
  Axis II:
  
  Target syndrome for ECT:
  
  - Acute Major depressive episode
  - Acute Mania
  - Acute Psychosis
  - Other (specify):
  
  - Is this a Primary or Secondary indication for ECT?
  
  - Primary indication because:
    
    - A rapid, definitive response needed because:
      
    - Risks of alternative treatments outweigh ECT risk (describe):
      
    - History of good response to ECT (describe):
      
    - Patient preference:
      
  - Secondary indication because:
    
    - Treatment resistance or intolerance (describe medications trials including medication, dose and duration):
      
  Medication(s) | Dose and duration | Combination with | Response/side effects

  - Adherence to medications is difficult (describe):
Previous course of ECT:  □ No □ Yes □ (describe)

<table>
<thead>
<tr>
<th>Date(s)</th>
<th>Lead placement</th>
<th>Stimulus parameters</th>
<th>Response/adverse effects</th>
</tr>
</thead>
<tbody>
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<td></td>
</tr>
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<td></td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>

In what Setting ECT Should Be provided?

□ In-patient  □ Out-patient  □ Either

Medical, Medications and Lab Information

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<thead>
<tr>
<th>Medical problems:</th>
<th>Stability</th>
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<table>
<thead>
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<th>Medications:</th>
<th>Dosages</th>
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<td>3-</td>
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</tr>
<tr>
<td>4-</td>
<td></td>
</tr>
</tbody>
</table>

History of complications with anesthesia?  □ No □ Yes □ (please explain):

Labs done within the last month? (please attach)

Any specialist consultations? (please attach)

Signed informed consent obtained?

• □ No □ Yes □ (date:______)
Clinical Global Impression (CGI) (Guy 2000)

Initial on date............

Severity of Illness: compared to your total experience with this population, how mentally ill is this patient?
0 = not assessed
1 = normal, not at all ill
2 = borderline mentally ill
3 = mildly ill
4 = moderately ill
5 = markedly ill
6 = extremely ill
7 = among the most extremely ill patients

Global Improvement (rate only in follow-up visits)
Rate total improvement whether or not, in your judgment, it is due entirely to ECT. Compared to his/her condition before ECT, how much has he/she changed?
0 = Not Assessed
1 = Very Much Improved
2 = Much Improved
3 = minimally improved
4 = no change
5 = minimally worse
6 = much worse
7 = very much worse
F/U on date ........ (___), F/U on date ........ (___), F/U on date ........ (___), F/U on date ........ (___)
Hamilton Depression Scale-7 items (McIntyre et al 2005)

For each item that best characterizes the patient during the past week, write the number in the appropriate score box.

Note: when scoring, rater is to combine patient replies with their observations

1. DEPRESSION MOOD (badness, the blues, weepy)
   "Have you been feeling down or depressed this past week? How often have you felt this way, and for how long?"
   0. Absent
   1. Indicated only on questioning
   2. Spontaneously reported verbally
   3. Communicated nonverbally (facial expression, Posture, voice, weeping tendency)
   4. Patient reports VIRTUALLY ONLY these feeling states in spontaneous verbal and nonverbal communications
   Note length of time if Depressed Mood present
   # weeks

2. FEELINGS OF GUILT (self-criticism, self-reproach)
   "In the past week, have you felt guilty about something you've done, or that you've let others down?"
   0. Absent
   1. Self-reproach (letting people down)
   2. Ideas of guilt or ruminating about past errors about sinful deeds
   3. Present distress is a punishment. Delusions of guilt
   4. Hears accusatory or denunciatory voices and/or experiences threatening visual hallucinations

3. INTEREST, PLEASURE, LEVEL OF ACTIVITIES (work and activities)
   "Are you as productive at work and at home as usual? Have you felt interested in doing the things that usually interest you?"
   0. No difficulty
   1. Fatigue, weakness or thoughts/feelings of incapacity (related to work, activities, hobbies)
   2. Loss of interest (directly reported or indirectly through listlessness, indecision and vacillation)
   3. Decrease in actual time spent in activities or decrease in productivity
   4. Stopped working due to current illness

4. TENSION, NERVOUSNESS (psychological anxiety)
   "Have you been feeling more tense or nervous than usual this past week? Have you been worrying a lot?"
   0. No difficulty
   1. Subjective tension and irritability
   2. Worrying about minor matters
   3. Apprehensive attitude apparent in face or speech
   4. Fears expressed without being questioned

5. PHYSICAL SYMPTOMS OF ANXIETY (somatic anxiety)
   "In the past week, have you had any of these symptoms?"
   0. No difficulty
   1. GI-dry mouth, gas, indigestion, diarrhea, cramps, bloating
   2. Resp.-hyperventilation, sighing
   3. Urinary frequency
   4. Sweating
   5. HAM-D score ≤3 indicates Full Remission.
   HAM-D score ≥4 indicates Non/Partial Response. TOTAL SCORE
   Total:
   Initial assessment Date ……….(__)
   F/U Date……….___, Date ……….(__), Date ……….(__), Date ……….(__)
Cognitive screening

Please refer to [www.mocatest.org](http://www.mocatest.org) to obtain MOCA test with instructions, the test is available in several languages and you can use different versions for repeated testing to avoid practice effect.

APPENDIX C

ECT Assessment Template (Initial)

- Date of assessment:
  - Not suitable for ECT (explain):

  - Suitable for ECT:
    - Target symptom(s):
      - Acute Major depressive episode
      - Acute Mania
      - Acute Psychosis
      - Other (specify):
    - Type of ECT:
      - Right Unilateral Placement (explain):
      - Bi-Frontal Placement (explain):
      - Bi-Temporal Placement (explain):
    - Acute
    - Continuation/maintenance
      - Frequency of treatment:
      - Electricity (may refer to titration tables):
        - Pulse width:
          - Frequency:
          - Duration:
          - Current:
          - Total charge:
      - Setting:
        - In-patient
        - Outpatient

Summary and rationale:
APPENDIX D

ECT Parameter Selection Guide, London Ontario City-wide ECT service for spECTrum5000Q machine

For urgent cases: start with Bitemporal lead placement, use % energy- half age method (see bellow) to estimate starting charge, re-stimulat at 50-75 mC charge if no seizure. Once patient is out of the acuity (after 2-3 treatments) establish seizure threshold using suggested titration tables.

For Elective Cases:
1- decide on Unilateral, Bifrontal or Bitemporal based on preference and cognitive status
2- establish seizure threshold using titration table, start at the suggested level re-stimulate after 20 second if no seizure or after 40 second if partial seizure at the next level
3- use 1.5-2.5 seizure threshold, brief pulse for bilateral treatment or, use 3-6 times the seizure threshold, ultra brief pulse for UL

Half age method:
%energy (joules)-1/2 age method (Petridges &Fink 1998)(e.g. age 60=1000x60/2x0.8x200= 187 mC starting charge)
An approximate method is 3xage=charge (e.g. 60x3= 180 mC starting charge)
(this is the best approximation for %energy-1/2age as it is within 10 mC up to age 80 and within 12 mC up to age 95). For UL placement, can use 6 x age

Bench Mark Method:
Start with a high charge to make sure there will be a rigorous seizure, measure change in heart rate (beat per minute, BPM) and EEG as “bench mark” then adjust charge to lower level keeping the bench mark within 6 BPM (e.g. start 4 times the age to get a vigorous seizure, record HR, then go down to lower charge and monitor HR). This can be used for monitoring the stimulus dosing, if the seizure EEG quality is not optimum the charge can increase by 50-75 mC. (Swartz 2002)
ECT Parameter Selection Guide, London Ontario City-wide ECT service for spECTrum5000Q machine

Titration to seizure threshold: Bifrontal and Bitemporal

<table>
<thead>
<tr>
<th>% total charge</th>
<th>PW</th>
<th>Freq</th>
<th>Duration</th>
<th>Current</th>
<th>Charge</th>
<th>Titration</th>
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<tbody>
<tr>
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<td>0.5</td>
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<td>3.5</td>
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*UL=unilateral, BL=bilateral, F=female, M=Male, S=senior >65

ECT Parameter Selection Guide, London Ontario City-wide ECT service for spECTrum5000Q machine

Titration to seizure threshold: Unilateral (usually Right)

<table>
<thead>
<tr>
<th>% total charge</th>
<th>PW</th>
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</table>

*UL=unilateral, BL=bilateral, F=female, M=Male, S=senior >65

SPECTRUM 5000Q machine (a MECTA machine) used at RMHC, London
ECT Parameter Selection Guide, London Ontario City-wide ECT service for spECTrum5000Q machine

EEG output

- Recruitment
- Tonic phase
- Clonic phase
- Post Motor Seizure
- Postictal Suppression

References: