Guide to Electroconvulsive Therapy

Revised for 2020. v1
by

Conrad M. Swartz, Ph.D., M.D.

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In electroconvulsive therapy (ECT) a controlled seizure is produced with an amount of electricity near the minimum that can induce seizure. In the ECT seizure brain neurons fire in cyclical waves, releasing neurotransmitters. Normally this seizure is self limiting, concluding after 20-90 seconds. This seizure is largely responsible for the therapeutic benefits from ECT in major depression and other disorders. There is no definitive mechanism of action for ECT, or for any biological psychiatric treatment.

ECT has selective effects and is not a general purge. Perhaps the ECT seizure replaces pathological patterns of neurotransmitters with normal patterns. When given properly to suitable patients ECT usually brings improvement in specific psychiatric diseases. As with other clinical procedures, reliably good outcome for ECT with minimized adverse effects requires both substantial direct clinical experience and detailed technical information. This guide aims to provide the basic technical information needed by practitioners. Please check yearly for updates to this guide. Written materials alone are not sufficient preparation to perform the ECT procedure; professional instruction with in-person supervision is needed, along with receiving ECT privileges granted by a hospital after due consideration.

BASIC DOCUMENTATION FOR ECT

A written approved ECT policy document in place helps maintain straightforward communications among psychiatrists and anethesiologists. Such a policy describes ECT indications and procedures and typically cites the latest "Task Force Report on the Practice of Electroconvulsive Therapy" from the American Psychiatric Association (APA, 2001). The policy usually describes the credentials for ECT privileges and the respective responsibilities of psychiatrists, anesthesiologists, and nursing staff in the procedure. This policy can resemble this Guide to ECT.

Several forms are usually used in documenting ECT treatments, as for: 1) logging treatments and identifying medication doses and ECT stimuli, 2) medical record progress notes, 3) scheduling upcoming physical exams and lab tests for outpatients, 4) billing, and 5) informing patients of benefits and risks and recording consent.

PROCEDURES AND ARRANGEMENTS MADE FOR ECT

ECT Physical Setup

ECT is commonly given in either a post-surgery recovery area or a specific room. In a post-surgery area the Thymatron® device is usually kept on a wheeled cart that has drawers for supplies and forms. If a specific room is used for ECT, it should be adjacent to rooms for pre-ECT waiting and preparation, and for post-ECT monitoring and recovery.

For the ECT procedure an oxygen supply, suction, oximeter, laryngoscopes and laryngeal airways should be at hand. A nearby sink is convenient. A resuscitation cart with defibrillator should be quickly accessible, although its use should be extremely rare.

Patient Selection (Clinical Indications)

The official FDA indications for ECT are catatonia and severe major depression in patients at least 13 years old. The FDA has not recognized the use of ECT for other conditions mentioned in textbooks of ECT and textbooks of psychiatry, and they will not be discussed here. The term "severe" is traditionally applied when the depression resists medication, includes catatonia or psychosis, is mixed with mania, or requires urgent response because of suicidal behavior, inanition, medical instability, or agitation. In major depression completed suicide is associated with preoccupations or delusions involving hypochondria or hopelessness (Schneider et al., 2001). Effective responses to ECT are seen in psychotic depression, catatonic depression, or with signs of classical melancholia. Classical melancholia includes low psychomotor activity, little facial expression besides concern, apathy, virtually no initiation of new thought, diminished mental reactivity, impaired problem solving, an affect of sickness or abulia, and sometimes unprovoked agitation (e.g, Hamilton, 1989). Rating observable signs such as these before and along the course of ECT helps identify and describe clinical response.

Anxiety disorders, personality disorders, drug or alcohol abuse, somatization and dissociative disorders have not shown a useful response to a course of ECT.

The commonly mentioned alternative to ECT in catatonia is a benzodiazepine, e.g., lorazepam. However, no months-long followup study of benzodiazepine use for catatonia was found, i.e., there is no evidence of an effective or safe continuing response to benzodiazepines. If anyone takes at least 2 mg/day of lorazepam, learning, memory, personality and coordination are adversely affected, so achievement of full remission on such benzodiazepine doses is doubtful.

Efficient Informed Consent

Written informed consent for ECT on an approved form is necessary. In some states consent can be provided by court order or it can be implied by a life threatening emergency, but specific details vary. In providing informed consent the patient must be able to communicate choice, understand information including the situation, and rationally manipulate information. During the consent process opportunities should be provided for the patient to ask questions and display comprehension. Information provided should include the basic steps in the ECT procedure, e.g., anesthesia, starting a brain seizure with electricity, oxygen ventilation with a breathing bag, and gradual awakening over about 5 minutes. ECT should be identified as a treatment not a permanent cure. As with antibiotics given for an ear infection, ECT treats the episode, and further episodes can occur later. A progress note is written when informed consent is obtained.

Possible adverse effects mentioned should include headache, muscle ache, and tendency to fall and forgetfulness for a few weeks. Some patients have complained of persistent loss of some memories, mostly autobiographical. The APA Task Force Report notes that a few patients who receive ECT complain of severe unremitting memory loss and inability to work. To date many investigators have looked but there has been no clear evidence that ECT produces the brain imaging signs or neurological testing signs that accompany stroke, dementia or traumatic brain injury. Death is rare, about 1 per 40000 treatments on average, higher among older patients and males (Dennis et al., 2016). No deaths attributable to ECT occurred in Denmark during 2000-2007, when 99728 ECTs were given (Ostergaard et al., 2014). One reported patient had an MI along the course of ECT that was not detected and he did not survive the next ECT.

The FDA states the warnings noted below (as quotes) about ECT and ECT devices, with the expectation they be communicated to patients:

`"When used as intended this device provides short term relief of symptoms. The long-term safety and effectiveness of ECT treatment has not been demonstrated. ECT treatment may be associated with disorientation, confusion and memory loss, including short-term anterograde and long-term autobiographical memory loss following treatment. Based on the majority of clinical evidence, these side effects tend to go away with a few days to a few months after the last treatment with ECT. Although the incidence of permanent cognitive memory loss was not supported by the clinical literature, some patients have reported a permanent loss of memories of personal life events.

"Patients treated with ECT may experience manic symptoms (including euphoria and/or irritability, impulsivity, racing thoughts, distractibility, grandiosity, increased activity, talkativeness, and decreased need for sleep) or a worsening of the psychiatric symptoms they are being treated for.

"The physical risks of ECT may include the following (in order of frequency of occurrence):
1. Pain/somatic discomfort (including headache, muscle soreness, and nausea);
2. Skin burns;
3. Physical trauma (including fractures, contusions, injury from falls, dental and oral injury);
4. Prolonged or delayed onset seizures;
5. Pulmonary complications (hypoxemia, hypoventilation, aspiration, upper-airway obstruction);
6. Cardiovascular complications (cardiac arrhythmias, heart attack, high or low blood pressure, and stroke); and
7. Death."

The FDA stipulates that ECT patients be provided with written instructions stated in the User's Manual of the ECT device employed. For your reference, at the end of this User's Guide are the Instructions to Patients from the User's Manual for the Thymatron® System IV device (2019, by Somatics LLC, Venice, Florida).

The patient's concern about his appearance can help him consent to ECT. The patient is asked if his privacy is important and if he would like to appear normal to others instead of looking sick. The signs of illness you can see are then described to the patient, e.g., looking exhausted as if with the flu, resembling a statue by showing little movement or facial expression, appearing gray and pale, hardly talking, and showing no interest in others. Patients are often surprised to hear descriptions of visible signs of their illness and usually do not dispute them.

Obtaining verbal agreement from the patient's family for ECT usually helps them feel involved and not disempowered.

Pre-ECT Evaluation

Routine pre-ECT evaluation includes psychiatric history, mental status and physical exams, review of systems, ECT and labs. Labs typically inclue serum electrolytes, TSH, a liver function enzyme, and a hemoglobin level or complete blood count. Hyperkalemia is a concern because succinylcholine temporarily raises serum potassium levels. Hypomagnesemia can result from long-term proton pump inhibitors such as omeprazole or heavy drinking; it lowers seizure threshold and causes cardiac arrhythmias, anxiety and muscle spasms. Loose teeth and dentures should be removed before ECT, and the patient's mouth should be inspected for disease and peculiarities. Patients of black race should have a previous or current sickle cell test result.

Medical disorders that may involve specialty consultations include organ failure (e.g., cardiac, renal, hepatic, pulmonary, thyroid), recent MI or stroke, unstable medical condition (e.g., hypertension, arrhythmias, electrolyte abnormality, thrombi, sepsis), seizure disorder, dementia, porphyria, osteoporosis, increased intracranial pressure, cerebral neoplasm, and familial malignant hyperthermia. Other conditions with special precautions include retinal detachment, Harrington rods, gastric banding, and history of tardive seizure. Cardiac pacemakers are usually set to fixed rate mode during the ECT session. It has been routine to temporarily convert cardiac pacemakers to fixed mode instead of demand mode during the ECT procedure. With some modern pacemakers and under supervision by a cardiologist it may be possible to leave the pacemaker in demand mode. Warfarin is routinely continued during ECT.

Concurrent medications should be tapered out before ECT when possible because of possible interactions. Anticonvulsants including benzodiazepines inhibit seizure and obstruct ECT benefit. Because of extrapyramidal symptoms, antipsychotics increase aspiration risk, especially in older patients and with propofol anesthesia. Lithium often but not always exacerbates ECT cognitive side effects so at least partial tapering should be considered. L-DOPA doses are often halved during an ECT course because ECT appears to potentiate its effects. Anticholinergic medications can exacerbate cognitive dysfunction during ECT.

Aspiration pneumonia and regurgitation occurred during ECT in a patient with a gastric band implanted for weight control. Regurgitation was then prevented with these measures: liquid diet starting the afternoon before each ECT, NG tube placed and suctioned ASAP during anesthesia, and minimizing time between anesthesia steps (Lubit et al., 2016).

What about precautions for surgical implants? Any metal below the head should have no effect on the ECT stimulus. To prevent implant displacement by seizure muscle contractions consider raising succinylcholine dose. Metal between the two stimulus electrodes attracts and concentrates current. If metal crosses through the skull the electrodes should be relocated away from it, e.g., with a metal skull plate in the R-parietal bone L-unilateral ECT may be preferable.

The FDA has stated expectations that each ECT patient undergo cognitive status monitoring prior to beginning ECT and along the course of treatment via formal neuropsychological assessment. The FDA expects evaluation of specific cognitive functions including orientation, attention, memory and executive function. Both retrograde memory (e.g., recall of past events) and anterograde memory (i.e., learning) should be considered. The Montreal Cognitive Assessment test may be useful in cognitive status monitoring (see https://consultgeri.org/try-this/general-assessment/issue-3.2.pdf).

Sleep Medication

To decrease insomnia associated with apprehension about ECT consider sleep medication. Unfortunately, benzodiazepines impair ECT efficacy and exacerbate cognitive dysfunction. A medication that leaves patients feeling somewhat sedated during the next day is usually tolerable, and perhaps desirable, during an inpatient ECT course. One medication to consider is promethazine 25 or 50 mg at bedtime. Because promethazine is generally mildly proconvulsant it should not impair ECT treatment quality. Trazodone is another possibility, but is usually less sedating. Either drug can increase orthostasis. Zaleplon and zolpidem are other possibilities.

Common Anesthetic Agents and Doses

The ECT electrical stimulus immediately causes unconsciousness and is not painful. Anesthetic narcosis at ECT is intended only to prevent awareness and memory of the muscle paralysis. Anesthesia deeper than needed for this can diminish efficacy and delay recovery. For ECT narcosis a methohexital bolus is commonly used. A common initial dose is 0.67 mg/Kg, with 20 mg increments added as needed. Because methohexital has some anticonvulsant effects, a dose exceeding 1 mg/Kg total is usually undesirable. Such larger doses are needed only rarely.

Alternatives to methohexital are attractive when seizures are weak or difficult to obtain. They include etomidate 0.15 – 0.3 mg/Kg (Avramov et al., 1995), remifentanil 4 - 8 mcg/Kg (Sullivan et al., 2004), and a mixture of ketamine 0.5-0.7 mg/Kg and propofol 0.7-1 mg/Kg referred to as ketofol (Yalcin et al., 2012). Ketamine alone has been used but takes longer to awaken from, thereby lengthening the ECT session. Used alone, ketamine provokes cardiac arrhythmias and hypertension that persist after the ECT seizure, requires more time for anesthesia recovery, and can exacerbate epilepsy. Still, ketamine can be useful for patients who do not show ECT seizures of good quality or whose progress stalls with other narcosis agents. Propofol alone (1 mg/Kg) shortens ECT seizures markedly, but less so when combined (0.75 mg/Kg) with remifentanil (1 mcg/Kg; note mcg represents microgram not milligram) (Kadoi & Saito, 2015).

ECT muscle paralysis is given to prevent hypoxia and musculoskeletal injury. After the narcosis medication dose, a succinylcholine 0.67 mg/Kg bolus is typically given. Lean or muscular patients may need larger doses. Obese patients may receive lower doses. Various drugs such as lithium, quinidine, cyclophosphamide, and aminoglycosides potentiate succinylcholine effect, so less succinylcholine may be appropriate. Hyperventilation with oxygen is started a minute before the electrical stimulus to hyperoxygenate and diminish carbon dioxide levels, both of which promote seizure activity. With slow circulation time or impaired lungs hyperventilate longer. Extensive burns, muscle crush injury, or motor neuron disease predispose to hyperkalemia in reaction to succinylcholine, so alternatives to succinylcholine should be considered with these.

Atropine (1 mg IM or 0.4 –0.6 mg IV) or its congener glycopyrrolate (0.2-0.4 mg IV) are often but not always used to diminish the brief bradycardia that routinely accompanies the ECT stimulus, and to decrease salivation. This dose of atropine is small enough for its cognitive effects to usually be negligible. Some patients produce enough saliva to complicate respiration during ECT, and even to provoke laryngospasm. An atropinic agent should be strongly considered for patients predisposed to bradycardia, e.g., taking a beta-blocker. The brief cardiac standstill that ordinarily follows the ECT stimulus is typically longer if a seizure does not occur, so an atropinic agent appears more desirable with stimulus titration or if the patient is not likely to readily respond to the ECT stimulus as from a high seizure threshold.

Electrode placement

There are four electrode placements in the modern ECT literature. In traditional bitemporal (BT) ECT an electrode is placed on the flat section of each anterior temple, just behind the forehead. This placement is generally considered to have maximal efficacy, but the most cognitive side effects (Swartz, 2009b).

In right unilateral (UL) ECT one electrode is just to the right of the vertex, the highest part of the skull. The other electrode is on the flat of the right anterior temple. The asymmetry of this placement is a rationale for lower side effects. At low stimulus doses UL ECT shows lower side effects and less efficacy than BT ECT. Lower efficacy means more ECT sessions are needed for response or less overall improvement is achieved. The response rate to low dose UL ECT was reported as half that for BT ECT (Sackeim et al, 2000). With UL ECT, raising the stimulus dose increases both efficacy and side effects, and at high doses the side effects are similar to those of BT ECT (McCall et al., 2000; McCall et al., 2002).

In bifrontal (BF) ECT each electrode is 25 mm anterior to the BT ECT site (Swartz, 2009b). With ECT electrodes of 50 mm diameter, the rear half is on the flat of the anterior temple and the forward half is on the forehead. BF placement is symmetrical, as is BT placement.

In left anterior right temporal (LART) ECT the left side electrode is entirely on the forehead, about 50 mm anterior to the location for BT ECT. The right side electrode is on the flat of the right anterior temple, just as with BT and UL placements. As with UL ECT the asymmetry of LART is a rationale for lower side effects, while its bilateral nature suggests efficacy. The distance between the two electrodes is the same for LART and BF placements (along the surface of the head). In several small studies LART provided high efficacy with low side effects (Swartz, 1994).

In circumstances of high risk or intense scrutiny (e.g., active suicidality, self injury, malignant catatonia, forensic hospitalization, assaultiveness), when cognitive side effects do not weigh heavily, BT ECT is probably most defensible. When there is no such risk or emergency, but minimizing cognitive side effects is crucial, UL ECT should be considered.

Ultra-brief Vs. Brief Pulse Stimuli. Pulse Width, Frequency, and Current

The pulse width of the stimulus strongly influences efficacy and side effects. Stimulus frequency has a much smaller effect. The pulse widths of brief pulse stimuli range from 0.5 msec to 2 msec. Ultrabrief pulses are shorter, typically 0.25 or 0.30 msec. Accumulated evidence shows that ultrabrief stimuli are less effective than brief pulse stimuli but have lower cognitive side effects. The side effects of 0.5 msec brief pulse ECT can be negligible, so likewise for side-effect differences between brief pulse and ultrabrief stimuli.

Unilateral brief pulse (UL-B) ECT improved quality of life more than Bitemporal ultrabrief (BT-UB) and Bifrontal ultrabrief (BF-UB) ECT (Galvez et al., 2016). Most patients receiving unilateral ultrabrief (UL-UB) were changed to brief pulse because of nonresponse. In this study response rates were 40% to UL-UB (12 ECTs/course), 81% to UL-B (10 ECTs/course), 74% to BF-B (7 ECTs/course) and 78% to BT-B (7.5 ECTs/course) (Ramalingam et al., 2016). In elderly depressed patients UL-B brought remission in 3.6 wks vs 4.6 wks with UL-UB, and corresponding

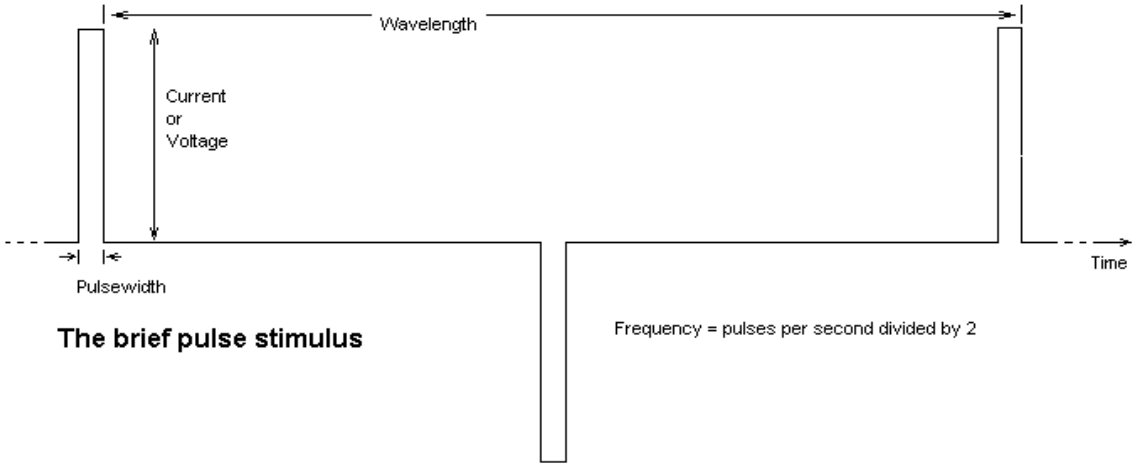
remission rates were 68% and 49% (Spaans et al., 2015). In this study nortriptyline or venlafaxine alone brought remission in only 21% after 5 weeks. So, UL-UB is more effective and faster than medications.

RUL-UB was less efficacious than RUL-B but showed less side effects (Tor et al., 2015). Perhaps UB stimuli should be reserved for selected patients such as those at high risk for confusion (e.g., dementia, Parkinson's) without life-threatening urgency for response.

Among brief pulse stimuli 0.5 msec may be most efficient because doses needed are lower than with wider pulse widths (Swartz & Manly, 2000). Side effects with 0.5 msec stimuli are minimal (Warnell et al., 2011), although some exceptions should be expected. A retrospective study found similar efficacy between 0.25 msec and 0.5 msec stimuli at 900 mA current (Niemantsverdriet et al., 2011); this result does not necessarily apply to 800 mA current. It raises the possibility that UB stimuli are as effective as brief pulse at 900 mA current but not 800 mA.

Sine wave stimuli are less efficient than brief pulse stimuli, with greater cognitive side effects and higher doses needed (Daniel & Crovitz, 1983; Squire & Zouzounis, 1986; Sackeim et al., 2007). This is probably because the 8.3 msec sine wave phase width is much longer than brief pulse widths (Swartz, 2009c). Marked side effects from sine wave ECT are common, so it is much less desirable than brief pulse ECT and widely considered obsolete.

Stimulus frequency represents waves per second. The alternating current of the Thymatron® instrument delivers two pulses per wave, so that 50 Hz means 100 pulses per sec. Stimuli of 30 Hz show mildly greater efficiency than 60 Hz stimuli (Swartz & Larson, 1989). Accordingly, stimuli near 30 Hz are more desirable than higher frequencies, but selecting pulse width deserves greater priority than frequency.



The brief pulse stimulus

The stimulus current affects the dose more strongly than its charge. The effect of current is so strong that it is preferable to adjust the stimulus dose by changing the charge and leaving the

current constant. The Thymatron® instrument uses 900 mA current. Lower currents are used in experiments on laboratory animals because their brains are much smaller than those of humans.

The charge rate is the amount of charge delivered per second of stimulus, counting gaps between pulses. Lower frequency and narrower pulsewidth give lower charge rate.

Initial Stimulus Dose

The ECT electrical stimulus dose is not an amount of charge or energy because any amount can be delivered slowly enough to be impalpable. The stimulus energy represents the amount of heat it contains, most of which is liberated in the scalp (Swartz, 1989). Charge is current multiplied by duration of current flow, and it represents the number of electrons in the stimulus. Although charge has traditionally been regarded as the dose, the minimum charge needed to induce seizure varies strongly with the stimulus current (Swartz et al., 2012; Swartz, 2009c).

Rather, the stimulus dose is represented by the amount of seizure foci generated. Physical modeling identifies stimulus dose as charge multiplied by current cubed (Swartz, 2014). With the Thymatron® ECT device the current is always 900 mA, so that stating the charge always specifies the dose. For ECT devices with adjustable current specifying the dose requires calculating charge multiplied by current cubed.

There are two common ways to select the initial electrical stimulus dose, age based and seizure threshold based. With both the initial stimulus dose depends on whether the electrode placement is right unilateral or bilateral. Both methods identify a dose that is typically high enough for good efficacy but not so high as to cause undue cognitive side effects. The minimum dose to induce seizure is called the seizure threshold, but is not a distinct characteristic such as body temperature because measuring it alters it (Swartz, 2014). The age based method is founded on the rise of seizure threshold with age, but does not involve measuring this threshold.

Age based dosing required 25% fewer ECTs for the same clinical improvement as titration based ($p<0.02$) dosing, in a prospective study of 79 patients (Aten et al., 2015). Post-ECT MADRS scores averaged 9.5 with age-based and 11.6 with titration. MMSE scores averaged 28 for both. ECTs were given with a Thymatron® System IV at 0.25-0.5 msec pulse widths.

For the three bilateral placements (BF, BT, LART) the initial stimulus dose is set in the same way. With the age based method the Thymatron® %Energy dial is set to half the patient's age. This sets a charge of 2.5 mC per year of age at 900 mA current. With a different ECT device at 800 mA current, a charge of 3.5 mC per year is appropriate. With threshold based dosing the initial dose is typically set 50 to 150% above seizure threshold. For the vast majority of patients initial doses set by the “half-age”method are within this range of the seizure threshold method (Swartz & Michael, 2013). The half-age method worked reliably in Japanese patients who were not taking high-dose benzodiazepines (Yasuda et al., 2015).

Choosing an initial dose for UL ECT is analogous to choosing an antipsychotic drug dose. If everyone receives the same high dose, patients who would respond to lower doses will experience unnecessary side effects. About half of patients who would respond to bilateral ECT will respond as well to low dose UL ECT, according to reported response rates (Sackeim et al., 2000).

Although studies have not been done to identify patients who will respond to low dose unilateral ECT, these patients probably show clear motor tonus, high peak heart rate during the seizure (typically over 140 bpm), and intense EEG activity at low doses. Rationally, UL ECT may be started with a high dose with the dose adjusted at later sessions according to these signs of seizure strength, as described in the "Benchmark Method" below.

With the age based method the initial dose in Thymatron® %Energy units for right unilateral ECT is set to the patient's age. This gives a charge of 5 mC per year of age at 900 mA current. With a different ECT device at 800 mA current, a charge of 7 mC per year is appropriate. For right unilateral ECT by the threshold method the dose is 3 to 7 times seizure threshold.

In the threshold based method the seizure threshold is measured in the first ECT session. A low stimulus dose is given. If it does not induce seizure, progressively larger doses are given at intervals of 20 to 30 sec until seizure occurs. Traditionally multiple sequential stimuli are regarded as independent of each other and the seizure threshold is the final stimulus dose. However, stimuli separated by less than 2 min are cumulative (Swartz, 2014). Accordingly, when more titration stimuli are given the result is less accurate.

Setting the steps for titration stimuli according to age appears to require fewer stimuli than previous titration methods (Swartz & Michael, 2013). In females under 65 years of age the first titration stimulus (in %Energy units) is one quarter age with later titration stimuli as needed of 3/8, ½, 5/8, ¾, 1 and 1.2 times of age. In older females the first stimulus is 3/8 age; later titration stimuli are ½, 5/8, ¾, 1 and 1.2 times age. In males the initial stimulus is 3/8 age, with later titration stimuli of ¾, 1, and 1.25 times age if needed. To convert from %Energy units to mC multiply by 5 mC per %Energy unit at 900 mA, or 7 mC per %Energy unit at 800 mA. For example, the initial titration dose for a 40 year old female is ¼ age, which is 10% Energy and corresponds to 50 mC at 900 mA. For a 40 year old male the initial titration dose is 3/8 age, which is 15% energy and corresponds to 75 mC at 900 mA. Older titration-based methods did not adjust the titration by age (McCall, 2009).

PROCEDURES FOR ECT TREATMENT

Routine ECT orders include no intake by mouth (NPO) for at least six hours, expected anesthesia medications and doses, and other medications needed, e.g., for headache, nausea, post-ECT hypertension or tachycardia. Hospitals vary in expectations for physician orders for ECT nursing procedures, such as vital signs, intravenous line, post-ECT monitoring, and discharge from the treatment area.

For an acute course of ECT two or three sessions of ECT are usually given each week. If there is a strong risk for cognitive dysfunction (e.g., elderly patient) and no overriding urgency, two sessions per week may bring a lower maximum accumulation of cognitive side effects (Lerer et al., 1995). In special circumstances one session per week can be reasonable. Conversely, patients who have shown negligible ECT side effects may be able to tolerate ECT more frequently or with greater flexibility in scheduling. In cases of psychotic self mutilation or lethal catatonia with unstable vital signs greater frequency may be advisable until the danger has passed. Two ECTs in one session ("double ECT") is not advisable without exceptional urgency because of greater side effects (Roemer et al., 1990).

Reassuring and Examining The Patient

Patients generally appreciate the physician's encouragement and reassurance on each ECT treatment day. This can be given while making routine pretreatment inquiries about changes in symptoms such as mood, suicidality, hopelessness, or delusions. This is an opportunity to ask about memory difficulties, and to examine for spontaneity, responsivity, psychomotor activity, and agitation. Vital signs are reviewed, and if there are pulmonary or cardiac concerns heart and lungs are examined. Before each ECT absence of oral intake for at least 6 hours should be verified with the patient.

Adjusting Stimulus Dose

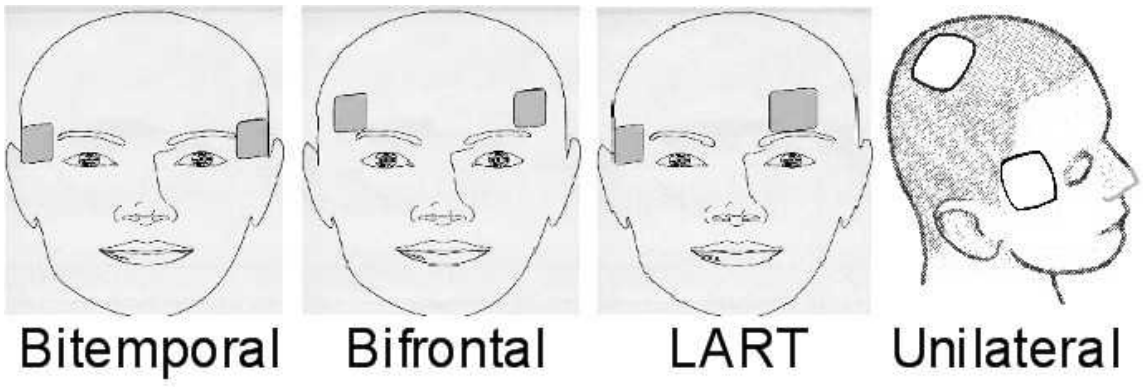
The stimulus dose is commonly adjusted along the course of ECT to counter a pattern of shorter seizure duration, lower peak heart rate, and weaker EEG intensity. This pattern is more common and substantial in elderly patients. Increasing the stimulus dose is one of several methods to consider when the seizure weakens (typically 5-15% increase) or does not occur in response to the stimulus (typically 20-40% increase). See the section below that lists methods to potentiate ECT seizure.

For some patients the initial stimulus dose may be possibly higher than needed. This is suggested by occurrence of long motor tonus, sustained high peak heart rate, and long intense EEG signs of seizure. A typical dose decrease is by 10-15%.

The Benchmark Method guides stimulus dose adjustment along the course of ECT (Swartz, 2002). A stimulus dose is given at the first ECT to produce vigorous seizure, e.g., half to two thirds times age for bilateral or full age for unilateral ECT. This establishes initial benchmarks for physiologic measurements such as peak heart rate during the ECT seizure, tonus duration, and EEG postictal suppression amount. If the stimulus dose is then decreased and the physiological measurements remain about the same the lower dose is regarded as sufficient. If a physiologic measurement after the first ECT exceeds the benchmark it becomes the new benchmark. If the physiologic measurement decreases substantially (e.g., peak HR 15 bpm below benchmark) the stimulus dose is increased. Several EEG measurements printed by the Thymatron® instrument are reported to reflect greater seizure intensity, including higher postictal suppression (Azuma et al., 2007), greater maximum sustained interhemispheric coherence, and longer times taken to reach peak power and maximum sustained coherence (Dinwiddie et al., 2012; Kemp et al., 2015).

Physical preparation of the patient

Jewelry, makeup, dental prostheses, contact lenses and hearing aids are removed and removal is verified. The patient should void. The patient is assisted to lay on a hospital cart with brakes applied. An IV line is started and kept open with a slow drip of an infusion fluid such as Ringer's lactate.



Electrode sites are washed with soap and water, and dried. Acetone is not used because repeated exposure is toxic to personnel. Stick on disposable monitoring electrodes (Somatics #EEDS) remain in place, are quickly applied, involve only minimal cleanup, and avoid contamination among patients. One electrode is applied to a shoulder to connect to the green (ground) lead wire. For recording a single channel of EEG, two electrodes are placed asymmetrically so that low frequency EEG waves are can be seen. For unilateral and LART ECT one recording electrode is placed on the left temple and the other on the lateral right side of the forehead. For bifrontal or bitemporal ECT one recording electrode is placed on the left lateral forehead and the other on the right mastoid process. For recording two EEG channels two electrodes are used for each channel, one over the mastoid process and the other near the middle of the forehead. Sometimes mastoid EEG electrodes record the QRS complex of the ECG, but at a smaller amplitude than the EEG. The Thymatron® instrument automatically examines the EEG and prints measurements of EEG seizure duration and several other EEG characteristics in the end of treatment report. This report appears when the “print stop” button is pressed, after the EEG seizure appears to end.

For ECG monitoring electrodes are placed above and below the heart. Connecting and enabling ECG monitoring by the Thymatron® instrument also produces a printed statement of the heart rate every 4 sec during the ECT seizure and prints the peak seizure heart rate in the report at the end of the paper strip.

For EMG monitoring the two long leadwires are connected to electrodes on the dorsal surface of the right foot and at the bottom of the calf muscle just above the ankle. A blood pressure cuff is wrapped around the right calf, just above the EMG electrode. The right leg is routinely used for the cuff, rather than the left, to observe crossover of seizure activity from the right hemisphere to the left with asymmetric placements such as unilateral and LART. The Thymatron® instrument prints the true electromyographic (EMG) signal and automatically states the motor seizure duration in the end of treatment report.

Thymapad stick on disposable stimulus electrodes remain in place, thereby freeing the clinician's hands. Electrode application is quick, cleanup is none to little, and there is no contamination between patients. With these electrodes, a small drop of PreTac lotion is first applied to each electrode site and rubbed to dryness. The electrode pouch is opened. Holding the tab on a side of the electrode, it is peeled from its backing and then applied to the treatment site on the patient. This is repeated for the second electrode except for unilateral ECT. For the vertex stimulus

electrode in unilateral ECT a cupped steel electrode is used, mounted in a insulated electrode handle. Stimulus cables and monitoring electrode lead wires are connected. Shortly before pressing the Thymatron® treat button conductive gel is applied to the concave electrode surface of the steel electrode. Applying gel sooner predisposes to accidental gel spill.

A headstrap with steel plate electrodes, and conductive jelly can be used instead. Handles can be substituted for the headstrap. Handles with a remote treat button are available for use with the Thymatron® device. Use of these electrode methods involves cleaning away conductive jelly between patients. With LART and the other bilateral electrode placements disposable stimulus electrodes can be used exclusively, minimizing cleanup. With right unilateral ECT the vertex electrode involves cleaning unless the site is free of hair and a Thymapad stick on stimulus electrode can be applied. If you prefer, the remote treat button on a handle can be used with Thymapad stick-on electrodes, simply for the remote treat button function.

After the stimulus electrodes are placed, and the stimulus cable and monitoring leadwires are connected, static impedance is tested. Impedance testing measures the quality of electrical connection to the patient. Excessively high impedance (e.g., over 3000 ohms) suggests incomplete electrical connection or inadequate contact between electrode and skin. Skin contact can be improved by examining electrodes for incomplete contact and by pressing on the back of the electrodes with a dry nonconductive object, such as a plastic handle.

If the printed EEG line is unusually thick or noisy, this suggests a partially broken connection in the leadwire. This is the thin wire that connects the recording electrode to the thick monitoring cable. Leadwires typically last from 3-12 months and are inexpensive.

Anesthesia Procedure

An atropinic medication if any is then infused (if not previously given intramuscularly), immediately followed by a narcosis agent such as methohexital. When the patient becomes unable to respond, the narcosis is usually sufficient. Inhibition of the eyeblink reflex occurs at a deeper level of anesthesia than needed to achieve amnesia for the ECT procedure. A mouth protector is then inserted to help prevent injury to teeth, tongue and mouth wall. The disposable Ventil-A mouth protector provides an air channel through the lips to permit ventilation, while protecting teeth and tongue. The leg blood pressure cuff is then inflated above systolic as the muscle paralytic agent (e.g., succinylcholine) is given.

The patient’s limbs are bared for view and monitored for muscle fasciculations as a sign of succinylcholine effect. Fasciculations usually begin around the eyes and progress down the body. A few patients show no fasciculations. To monitor for paralysis a weighty reflex hammer is desirable, e.g., large head Queen Square hammer (http://medexamtools.com/r1-mega.htm). The common taylor tomahawk hammer can not gather enough momentum. An electrical peripheral nerve stimulator set to a "train of four" pattern can be used in place of a reflex hammer. The goal is one or two twitches to the four pulses; more twitches suggests insufficient relaxation. When reflexes disappear and muscle tone at the knee joint lightens the patient is usually ready for the stimulus.

Active hyperventilation with oxygen by mask is given after the muscle relaxant (succinylcholine) because hyperoxia and hypocarbia facilitate the seizure. Ventilation is continued

during the seizure, with brief interruptions as needed for observations such identifying that the EEG seizure ended. After the seizure ventilatory support without hyperventilation continues until the patient breathes on his own, usually 3 to 4 minutes after the ECT stimulus.

Stimulus delivery

Before delivering the stimulus the ECT dose setting is verified to be as intended. With the Thymatron® device the single %Energy dial sets the stimulus dose; with other devices several knobs each adjust the stimulus dose and each must be checked. The pulsewidth, frequency, and total duration of the ECT stimulus are automatically set by the Thymatron® instrument to conform to the dose set with this %Energy dial. The pulsewidth and frequency settings for each %Energy stimulus dose on a Thymatron® instrument are referred to as a program, and several different programs are built in. The default program uses 0.5 msec pulsewidth at each dose with the frequency automatically chosen so that the stimulus is about 8 sec long, except at the few lowest doses. The Thymatron® device identifies this program as “LOW .5” or as “PRESET.” Pressing in the Percent Energy dial displays the name of the program that is currently selected.

Ultrabrief stimuli can be quickly selected on the Thymatron® device by pressing in the Percent Energy dial and rotating this dial while continuing to press in. Rotate this dial until “LOW .25” or “LOW .3” is displayed. The number shown corresponds to the pulsewidth in msec. Clinicians can configure the USER program to provide the pulsewidth and frequency they choose at each %Energy stimulus dose setting. This program is stored in the Thymatron® device after the power is switched off. In some countries (not USA) a double dose program is included that delivers stimulus doses up to 200 %Energy (1008 mC at 900 mA). This double dose program uses 0.5 msec pulsewidth up to 100 %Energy, and 1 msec pulsewidth above that.

Nursing staff are then positioned to prevent the patient from falling during the treatment. Then the position of the mouth protector is checked. To guard the jaw and mouth the patient’s jaw is firmly closed and elevated when applying the ECT stimulus. The thickness of the Somatics Ventil-A disposable mouth protector helps to keep the jaws separated and prevent jaw injury. During the electrical stimulus each staff person should avoid contact with the patient at more than one point. An announcement such as “treating now” is made by the treating doctor. Then the “Treat” button is pressed and held. This delivers the chosen stimulus. Releasing the Treat button before the stimulus concludes immediately stops stimulus delivery, and the dose will then be less than was chosen.

When the Thymatron® Treat button is pressed there is a one second delay before the stimulus. During this period a cautionary tone sounds. Releasing the Treat button during this period aborts the stimulus. During stimulus delivery a buzzing tone sounds and the Thymatron® Treat button is lit. At the end of this stimulus the buzzing tone stops, the light in the Treat button turns off, and the paper printout begins. After the stimulus ends the Treat button is released. If abnormal functioning of a stimulus electrode or the ECT device occurs during the stimulus the Treat button should immediately be released to discontinue the stimulus.

You can familiarize yourself with these signals and general operation of the Thymatron® instrument by operating it with no cables connected. Learning will be more realistic and

straightforward by using the Somatics Ectobrain-II device to simulate the ECT physiology shown by patients. The Ectobrain-II is connected as if it were a patient, the Treat button is pressed, and the Thymatron® device prints EEG, ECG, and EMG recordings as if a patient were undergoing treatment.

Monitoring The Patient

When the electrical stimulus starts the patient's muscles enter a state of tetany. This reflects direct electrical stimulation of the motor cortex and does not indicate seizure. The muscles usually show a short sudden movement as the tetany transitions into tonus at the end of the stimulus. Sometimes the motor seizure begins a few seconds after the stimulus ends. Seizure onset can begin 15-60 seconds after the stimulus, but such late onset is unusual. When a motor seizure does not start within a few seconds after the stimulus, increasing heart rate and EEG amplitude are checked for to see if a seizure is brewing. If neither heart rate nor EEG amplitude is increasing consider delivering another stimulus about 30 sec after the first. It is not necessary for this second stimulus to be higher than the first because the effects of the two stimuli should add together. If the second stimulus fails consider raising the stimulus dose substantially, such as by 50%. If the third stimulus fails, consider raising the stimulus again, perhaps by 50% or 100%.

In seizure monitoring the occurrence of motor activity indicates basic efficacy, and EEG is also examined to generally assure that the seizure ends. Motor activity is usually monitored in the cuffed right leg. The term “cuffed” refers to placement and inflation of a blood pressure cuff around the right calf to pressure above systolic. The cuff is inflated when the muscle paralytic agent (e.g., succinylcholine) is given. A sign of good motor seizure is tonus lasting at least 3 sec and a total duration of tonus and clonus lasting at least 18 sec. If motor activity is less than this consider restimulation at a higher electrical dose.

EEG seizure is typically about 50% longer than motor seizure. Listening to the Audible EEG of the Thymatron® instrument can replace looking at the EEG printout during the treatment. The Audible EEG seizure endpoint is heard as a steady tonal pitch that lasts for at least one second. This corresponds to the flattening of the printed EEG at the end of the seizure. Sometimes the ECG is visible in the EEG tracing, especially if an EEG electrode was placed on the mastoid. Young patients are more likely to have long seizures. If the EEG seizure duration reaches about 100 sec duration consider terminating the seizure with intravenous propofol (e.g., 0.5 - 0.7 mg/Kg) or midazolam (2-4 mg). Seizures longer than about a minute have more side effects but probably little if any additional efficacy. An identifiable seizure endpoint on the printed or Audible EEG is useful but not definitive evidence that the seizure stopped. This is because seizure can continue in brain regions far away from the EEG electrodes (Swartz, 1996). Still, it is standard practice to judge that the seizure ended by observing an EEG endpoint and examining the patient’s behavior.

Shortly after the seizure starts the heart rate usually accelerates, reaching its maximum during that seizure in 15 to 30 sec. A peak heart rate under 130 bpm usually indicates a weak treatment, unless a cardiac condition or medication interference is present. The peak heart rate during a good quality ECT seizure is similar to but typically higher than in a cardiac treadmill test (Swartz & Shen, 2007). A few seconds after the EEG seizure appears to end, press the print “start/stop” button. The end of treatment report is then printed by the Thymatron® instrument. Printing then automatically stops.

The peak EEG amplitude in a vigorous seizure usually occurs 6 to 12 sec after the ECT stimulus. An EEG sign of intense seizure activity is high amplitude rounded waves (3 to 5 per second) with smaller faster waves riding on it. EEG flattening at the end of the seizure is also a sign of intense seizure activity and therapeutic efficacy (Azuma et al., 2007). This flattening represents suppression of electrical activity, and so is called postictal suppression. Flatter means greater suppression. The Thymatron® instrument end of treatment report includes a measurement of postictal suppression; this measurement corresponds to seizure intensity (Porter et al., 2008).

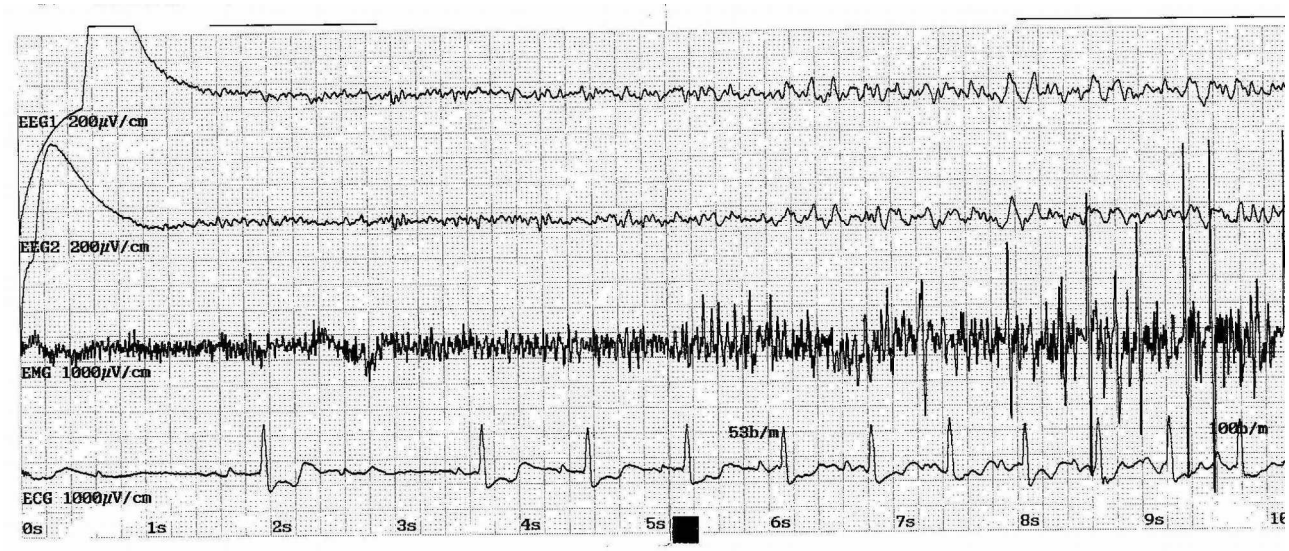


Figure above: Typical early ECT seizure. EEG: top 2 lines. EMG: line 3. ECG: bottom shows slowing after ECT stimulus to rate of 30 bpm with quick return to about 100 bpm.

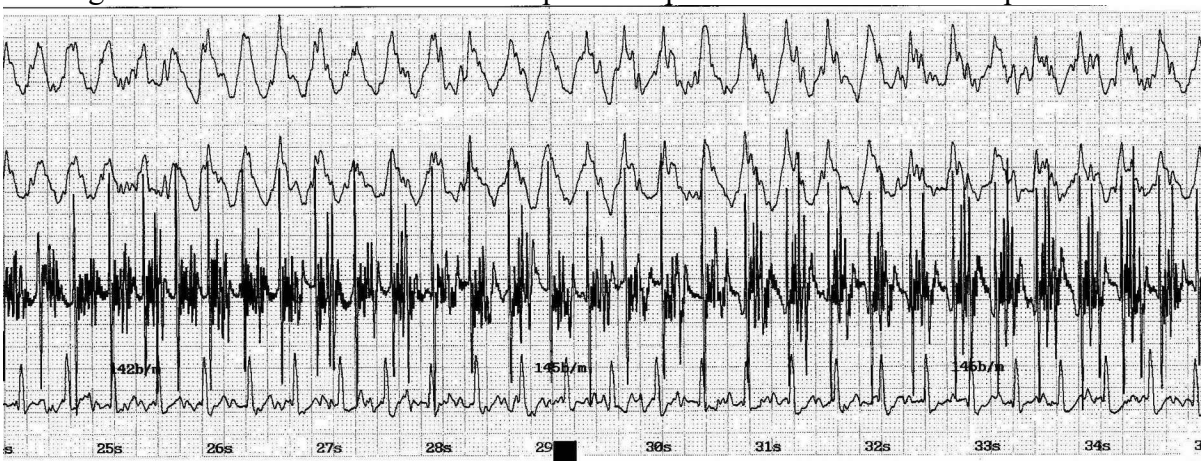


Figure above: Typical middle seizure. EEG shows high amplitude 3 Hz waves with high frequency waves on top. The heart rate reaches about 145 bpm.

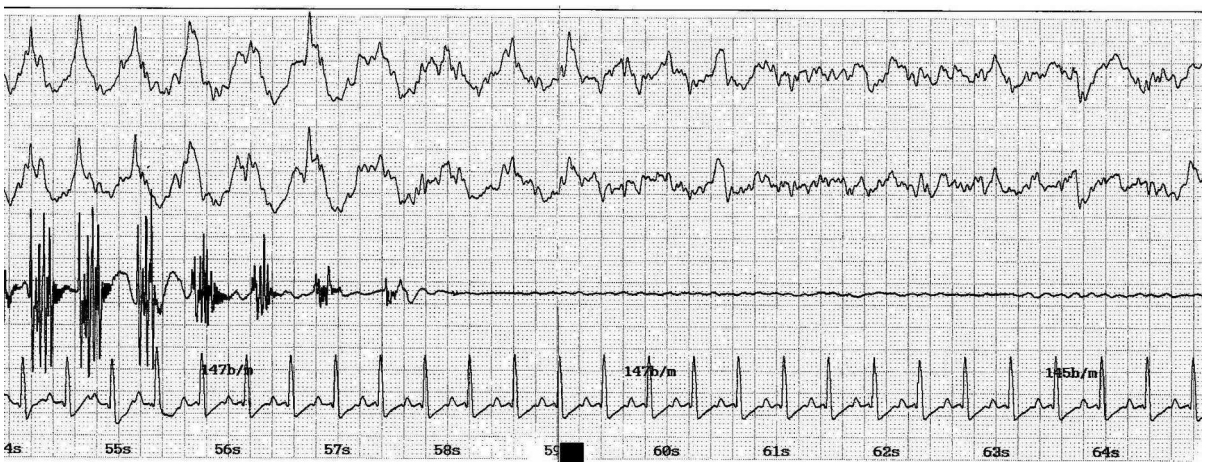


Figure above: Typical end of motor seizure seen on EMG. EEG seizure continues.

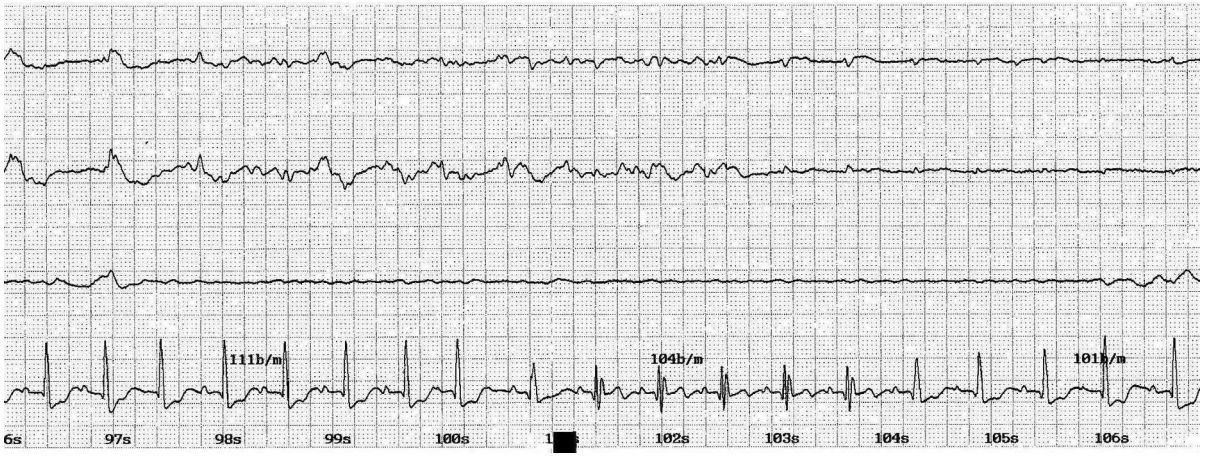


Figure above: Typical EEG seizure endpoint with strong postictal suppression (flattening). ECG is visible on the flattened EEG. HR decreases to about 100 bpm. These four figures are from the same ECT treatment.

If the patient appears delirious or unusually disoriented after ECT it is possible that occult seizure activity is occurring, and administration of propofol or midazolam should be considered to better assure termination of the seizure. The "propofol interruption method" described below aims to prevent continuing occult seizure activity routinely.

Awakening from the ECT treatment typically occurs gradually, with return to full orientation in 10 to 15 minutes. Quicker awakening after ECT with full alertness and orientation suggests that the generalized seizure was not achieved, and that efficacy is less than expected.

Effectiveness of the session. Restimulation.

If the treatment session is assessed as probably ineffective, it is routine to consider delivering another stimulus in the same session, under continuing anesthesia. Effectiveness as clinical improvement it is not assessed until hours or days after the ECT session, so physiological measurements (EEG, heart rate, motor seizure) are used to assess probable

effectiveness during the session. If one or more observations indicates a vigorous seizure, the treatment is usually considered effective. These observations include any of:

1) tonic-clonic motor activity at least 18 sec, with at least 3 sec of tonus;
2) seizure peak heart rate at least 130 bpm with increase above baseline at least 30 bpm;
3) strong EEG postictal suppression;
4) a high intensity EEG waveform starting 6-12 seconds after the stimulus consisting of both high frequency and low frequency waves, as illustrated on the previous page.

Elderly patients and patients receiving propofol anesthesia can experience effective ECT sessions without showing the signs listed above, despite increases in stimulus dose. So, absence of the signs above suggests but does not definitely indicate absence of effectiveness. Absence of all EEG, motor and heart rate evidence of seizure suggests a probably ineffective treatment, and restimulation 1 to 2 minutes after the previous stimulus is a consideration. The younger the patient the lower is this author's impression of treatment effectiveness when any of the four signs listed above is missing.

Printed report and progress note

The end of treatment report printed by the Thymatron® instrument lists date, time, stimulus charge, current, frequency, pulsewidth, duration, and impedances. It also lists physiological measurements including baseline and peak heart rates, measurements of seizure duration from EEG and EMG recordings, and several EEG measurements.

After the ECT treatment a progress note is written in the medical record. Some of this is included in the Thymatron® end of treatment report, e.g., stimulus characteristics, EEG seizure duration, peak heart rate. The end of treatment report printed by the Thymatron® device is sometimes saved; the rest of the paper printout is typically discarded after inspection.

Managing common complications

When an exceptionally long seizure occurs (e.g., longer than 60 sec motor or 120 sec EEG) consider seizure termination with an intravenous infusion of methohexital 40-50 mg, propofol 0.5 – 0.7 mg/Kg, or midazolam 1-4 mg. Risks for long seizure (or status epilepticus) include: young, male, early in ECT course, meds that can provoke seizure (theophylline, stimulants, bupropion, lithium, antipsychotic e.g., risperidone), epilepsy history, concussions, previous long seizure, alcohol or drug withdrawal, history of psychotic reaction to stimulants, and stimulus titration.

Post-ECT hypertension or tachycardia is commonly mitigated with labetalol (5-10 mg) or esmolol (about 1 mg/Kg) intravenously immediately *after* the ECT seizure ends. Both diminish hypertension and tachycardia but there are differences. Esmolol works in 1-2 minutes, and more strongly on blood pressure than heart rate. Labetalol acts in 2-5 minutes, and more strongly on heart rate than blood pressure. CNS-active beta-blockers have anticonvulsant effects and both these drugs weaken ECT seizure when given before the stimulus (Van den Broek et al., 1999). Still, some fragile patients may require medication before the ECT seizure ends or even before it starts.

Post-ECT headache and musculoskeletal pain is commonly managed by oral NSAID or acetaminophen, or with ketorolac 15-30 mg. Heat and massage also help. Further appearance is prevented by giving NSAID or acetaminophen 2 hrs prior to ECT with a sip of water, or ketorolac IV shortly before ECT.

Nausea or vomiting post-ECT is commonly treated with ondansetron 4 mg IV. Further occurrence is prevented by giving this medication 10-30 minutes before ECT.

Troublesome pain from infusion of the narcosis agent (e.g., methohexital) is reported by a few percent of patients. This is usually prevented by infusing 5-10 mg of lidocaine immediately prior to the narcosis drug. A larger lidocaine dose before ECT weakens the seizure and treatment efficacy.

ECT Session Recovery

After the ECT seizure, heart rate, blood pressure, respirations, level of consciousness, orientation, and agitation level are monitored until all return to stable safe levels. Supervision is given to each patient while in the ECT area, before and after treatment. To prevent aspiration, the patient is routinely turned on his side after ECT until recovery is complete. Inpatients are escorted back to the ward, and visits are postponed until at least four hours after ECT.

Postictal Agitation: Diagnosis, Treatment and Prevention

The syndrome of postictal agitation typically lasts for several hours. When severe it includes yelling, flailing, and demands to get up from the cot. Onset of postictal agitation often occurs before awakening from anesthesia, with moaning and purposeless limb movements; this has led to incorrect identification of this syndrome as delirium. Rather, postictal agitation appears to be an expression of lactate and hypercarbia induced panic (Swartz, 2009d). Most patients who show postictal agitation have muscular or lean body builds. Even mild postictal agitation typically lingers subjectively if not visibly, leaves patients uncomfortable for the rest of the day, and tempts them to refuse further ECT. So, it is essential to identify and treat postictal agitation quickly, prevent recurrences, and clearly reassure patients that it should not happen again.

Immediate treatment of postictal agitation in the ECT area is usually with parenteral midazolam (1 –2 mg). When the patient becomes able to swallow, this is followed by a dose of oral alprazolam or oxazepam. With midazolam infusion the possibility of sudden apnea is carefully monitored for. When postictal agitation is mild and discovered when the patient has regained the ability to drink water, an oral benzodiazepine with reassurance usually suffices on that day. Once postictal agitation develops, it typically recurs at later ECTs unless prevented. Prevention is by raising succinylcholine to 1.1 mg/Kg when tolerated, and infusing methohexital 40 mg or propofol immediately after 30 sec of motor seizure or at the motor seizure endpoint, as you prefer. Prevention is appreciated by both the nursing staff and the patient.

Postictal Delirium

Postictal delirium refers to marked and lingering disorientation with or without visual hallucinations and other cognitive dysfunction after an ECT. Onset of agitation within 15 minutes

of the ECT treatment is probably the syndrome of postictal agitation not delirium if the patient regains orientation and remains anxious or agitated. Postictal delirium is presumably sometimes caused by ongoing brain seizure activity. Delirium has been said more common in patients with Parkinson's disease (Borisovskaya et al., 2016). When delirium occurs after ECT consider reconnecting the EEG monitor.

Regardless of what the EEG shows, consider giving propofol or midazolam intravenously to better assure seizure termination. Intravenous lorazepam or diazepam can be used but their effects persist for several days. If troublesome delirium continues for an hour or longer consider a standard diagnostic EEG. If delirium persists to the next day consider stopping or shortening the ECT course. If further ECTs are considered see the "propofol interruption method" below. A few case reports mention using acetylcholinesterase inhibitor or memantine to prevent recurrence.

A few patients show intense confusion and disorientation from ECT. Perhaps this derives from postictal delirium and can be limited by the propofol interruption method. When none of the methods described here limit it and the patient's confused behavior is risky, consider decreasing the frequency of ECT sessions or discontinuing ECT.

Tardive Seizure

A tardive seizure is a seizure that occurs after the ECT session in a patient who recently received ECT. It is extremely rare but dangerous, and fatalities are known. One presentation of tardive seizure is the apparent onset of seizure shortly after the ECT treatment session while the patient is still in the ECT recovery area. The patient may show clonus or worsening delirium. This timing suggests that the ECT seizure never stopped, but continued in part of the brain and then became more generalized. When tardive seizure appears intravenous anticonvulsant treatment should be immediate and vigorous, e.g., full anesthesia with propofol. The patient should be transferred to the hospital intensive care unit because of high risks for sudden cardiac or pulmonary arrest. This risk may last for a day or even several days.

A different presentation of tardive seizure is onset of seizure at an ECT session after anesthesia was started but before the electrical stimulus is applied. This tardive seizure is precipitated by the anesthetic narcosis agent, e.g., methohexital, etomidate, ketamine. This is handled in the same way an ECT seizure is, with insertion of a mouth protector, vigorous ventilation, and monitoring of EEG, ECG, and motor seizure. If the seizure is longer than 60 sec motor or 100 sec EEG it should be terminated, such as with propofol or midazolam.

Discharge From the ECT Area

Discharge from the ECT area usually occurs when the patient returns to full alertness and orientation , shows repeated vital signs in normal range, walks in a stable manner, and shows no new behavioral disturbance such as postictal agitation or delirium. This is typically 20 to 30 minutes after the ECT session. Intravenous lines should be removed before the patient leaves the ECT area.

Methods to Potentiate ECT Seizure

Listed below are steps to consider when the ECT seizure becomes weak or short, or if no

seizure develops. The first five steps are routine for all patients. Steps six and higher are in order of priority.

1. Discontinue anticonvulsant medication, in the absence of epilepsy.
2. Discontinue sedative hypnotic agents. Gradual taper over 2-3 weeks is needed if the patient is physically dependent or had used daily for a month or longer.
3. Minimize other concomitant medications. Some medications have anticonvulsant effects that are not widely known, e.g., allopurinol, MAO inhibitors, dextromethorphan, beta-blockers.
4. Identify and treat hypothyroidism, congestive heart failure, pulmonary insufficiency and other medical problems that apparently diminish oxygenation and metabolic rate.
5. Prevent hypothermia. Hypothermia is common in the elderly.
6. Avoid propofol. Minimize doses of barbiturate narcosis agents, e.g., methohexital.
7. Increase the stimulus current if possible
8. Increase the stimulus charge if possible.
9. Consider narcosis with etomidate (0.15 – 0.3 mg/Kg) or remifentanil (0.4 - 0.8 mcg/Kg) instead of barbiturate. Seizure thresholds are lower and seizures are longer with these (Sullivan et al., 2004; Avramov et al., 1995).
10. Consider narcosis with ketamine (0.5 – 1 mg/Kg) or a mixture of ketamine with another narcosis agent. Ketamine is risky with epileptic patients because it can provoke seizure.

Methods of Minimizing Cognitive Side Effects From ECT. These can be combined.

1. Minimize or taper out benzodiazepines, anticonvulsants and anticholinergics.
2. Use LART, UL or BF placement when possible. Alternate among these placements.
3. Propofol interruption method (see below).
4. Decrease electrical stimulus dose if feasible.
5. Decrease number of ECT sessions per week.
6. In selected patients use ultrabrief stimuli, e.g., dementia, Parkinson's, confusion history, absence of urgency for response

Cognitive side effects include disorientation, orbital-frontal syndrome (sometimes misidentified as ECT induced hypomania or mania), forgetting, and delirium. When disorientation occurs it is typically not noticeable until after three or more ECTs (Calev et al., 1991). Orbital-frontal syndrome involves disinhibition, intrusiveness, overfriendliness, impatience or indiscreet behavior. Disorientation and orbital-frontal symptoms accumulate gradually, and they typically fade gradually and completely over weeks to months. If side effects are more clinically substantial than benefits the ECT method may be undesirably intense or frequent, or there may be a complicating medication (e.g, a stimulant) or coarse brain disease (e.g., Parkinson's).

ECT-induced forgetting refers to personal information such as phone numbers and names (“autobiographical memory.) Forgotten memories usually reappear gradually but some patients report that some details are permanently forgotten and need to relearned. Patients who complain of forgetting after ECT typically experienced more cognitive deficiencies before ECT (Sobin et al., 1995). It is rare that anterograde memory deficits persist as long as 12 weeks (Smith et al., 2010).

Cognitive side effects vary widely among patients. Both acute and persistent cognitive deficits can be caused by psychiatric illness and psychotropic medications, particularly

benzodiazepines and drugs with anticholinergic effects. If cognitive symptoms develop during ECT, they are likely to worsen with further ECTs, although changing the ECT method can sometimes help. If no cognitive symptoms have occurred from one or more previous ECT courses, it is reasonable to expect little to none from additional ECT courses given with similar method. Scheduling can be more flexible and frequent for patients who have shown negligible side effects. Patients with cognitive side effects from ECTs, and those who need to avoid even temporary mild cognitive side effects, are candidates for the methods to reduce side effects noted below.

Discontinuing or tapering psychotropic medications before ECT can diminish cognitive side-effects. Benzodiazepines, anticholinergics (e.g., tricyclic antidepressants), and lithium can exacerbate cognitive side effects. Anticonvulsants weaken ECT seizure and sometimes its efficacy, e.g., benzodiazepines can prevent good quality seizure and diminish response, even to bitemporal ECT (Minelli et al., 2016). Antipsychotic drugs predispose to aspiration. They can also hide psychopathology that ECT aims to remove, e.g., depressive or manic symptoms, and thereby confound evaluation.

Some ECT methods usually produce less cognitive side effects. Bifrontal ECT and the asymmetrical placements of right unilateral and LART ECT generally have fewer side effects unless excessive stimulus doses are used. Varying the electrode placement among two of these or all three may have still lower cumulative side effects. The pulsewidth of 0.5 msec showed low side effects, even with bitemporal placement (Warnell & Swartz, 2011). Ultrabrief pulsewidths of 0.25 or 0.30 msec tend to have low side effects, perhaps even lower than with 0.5 msec, but may require more treatments for remission. Ultrabrief stimuli are discussed on pages 6 & 7.

Studies have failed to find evidence that memantine and acetylcholinesterase inhibitors (e.g., rivastigmine, donepezil) improve memory along a course of ECT. Changes made by these agents were negligible despite authors' claims (Alizadeh et al., 2015; Abbasinazari et all., 2015).

The "propofol interruption method" can be used together with any of the methods above. Propofol 0.5 mg/Kg is infused intravenously starting 15-20 sec after the end of the ECT stimulus. This prevented EEG seizure duration from reaching 100 sec (Warnell & Swartz, 2010). Surprisingly, the time to recovery from an ECT treatment was not longer with propofol interruption, and the trend was for quicker recovery. There is no obvious rationale to avoid using propofol interruption routinely, especially in patients at risk for cognitive side effects.

For bilateral placements higher stimulus doses tend to increase cognitive side effects without increasing efficacy, as along as a generalized seizure is obtained. For unilateral ECT higher doses have generally higher efficacy, but if a patient responds to a low dose it may suffice.

Giving fewer ECT treatment sessions per week should decrease peak cognitive side effects in patients who have experienced substantial side effects (Lerer et al., 1995). The usual range is 2 to 3 treatments per week, but reported treatment frequency has varied from one treatment per week to one treatment per day for 5 days per week. ECT given 5 times per week gave responses and side-effect similar to 2-3 times per week for a similar number of treatments, with UL-ultrabrief stimuli; however, troublesome confusion sometimes occurs (Rasmussen et al., 2016).

AT THE CONCLUSION OF THE ECT COURSE

When to Stop the Acute ECT Course

The psychopathologic signs of illness that originally led to ECT treatment are monitored along the course. For depression this may be psychomotor retardation, low spontaneity and responsiveness, withdrawal from interacting with other people, appearing sick or exhausted, abulia, anergia, and obsessions about sickness. The ECT course is usually continued until signs of illness disappear, or until further improvement does not occur over the last three ECTs.

The opinions of family and close visiting friends about the patient's return to usual personality and skills can be helpful, but temporary side effects from ECT can interfere with this. Asking the patient for how depressed he feels does not distinguish between depression and dysphoric anxiety or personal situations, but if the patient feels happy and well this can confirm remission.

The ordinary minimum number of ECTs in a course is six. If a patient achieves complete remission by the third ECT and the illness included no violence or suicidality it is reasonable to consider concluding after five ECTs. If the patient was severe, chronic, or psychotic consider treating to maximum improvement in what you see, then adding two more ECTs. Courses are generally longer with unilateral ECT and with ultrabrief ECT.

The longest courses may be with very elderly patients, with bipolar patients who switch phase from depression to full mania during the course, and with catatonic patients who have comorbid coarse brain disease (e.g., traumatic brain injury, developmental disorder). A typical course is 6 to 10 treatments. If your average is higher than this, consider seeking an explanation.

Evaluation After ECT Course

After ECT it is helpful to examine for cognitive dysfunction, anxiety disorder, and residual pathology from the condition ECT was given to treat, e.g., major depression. Comorbid dysphoric anxiety disorders are common in patients with major depression, even those with melancholia or bipolar disorder. When present an anxiety disorder needs a treatment plan that is separate from ECT. Dysphoric anxiety has many of the same subjective symptoms as major depression, including low mood, loss of pleasure, insomnia, worry, hyperphagia, feelings of sickness, fatigue, remorse, and suicidality. Suicidality in anxiety disorders is several fold more frequent than in the normal population (Ramsawh et al., 2014; Sareen et al., 2005), so its presence does not distinguish between major depression and anxiety disorders. Dysphoric anxiety disorder is a common reason for what appears to be incomplete response to ECT or early relapse. Identifying and treating comorbid dysphoric anxiety disorder is essential to achieving remission from complaints of depression.

If the patient complains of depression after ECT, whether immediately or within a month, but you do not see the psychopathologic signs of illness that were present before ECT, dysphoric anxiety disorder is a likely cause. Detection of anxiety disorders after ECT involves examination after both 1-3 weeks and 4-6 weeks post-ECT, not just shortly after the ECT course. This is because anxiety disorders typically respond somewhat to ECT for a few days and up to a month. Signs of anxiety disorder include being upset at small things, ventilatory behavior, wanting to enjoy life but feeling frustrated, and dissatisfaction with relationships and situations rather than just feeling ill.

Basic cognitive evaluation includes recall of personal biographical information, orientation, recall of details of current news events, self-care such as hygiene and dress, and judgment about returning home and resuming work. After an ECT course the patient should remain under supervision by others for two to four weeks. Major life decisions should be avoided during this period (e.g., marital, large investment, house purchase or sale), and the patient should not drive or operate machinery.

Preventing Recurrence

After the ECT course a plan is started to prevent recurrence of the illness. Medication can prevent episodes of melancholia, psychotic depression, or mania that it is not able to treat. For major depression with classical melancholia or catatonia consider bupropion, nortriptyline, or high doses of a SNRI, e.g., 150-300 mg/day of venlafaxine, 120 mg/day or more of duloxetine. Lithium may be added for further protection or if the patient is bipolar. Patients with a mood disorder that has resisted prevention with medication can benefit from ambulatory maintenance ECT, typically given once every 3 to 4 weeks. At its start ambulatory maintenance ECT is sometimes given weekly frequently and then gradually slowed.

Depression and Brain Changes

Major depression itself is associated with microanatomical abnormalities in the brain. Hippocampal shrinkage increases with depression duration and has been observed after even one episode in patients receiving treatment (Sheline, 2000). The hippocampus participates in learning and memory, and shrinkage occurs early in the course of Alzheimer's Disease.

QUALITY ASSURANCE MONITORED ASPECTS

For quality assurance purposes you may wish to identify aspects of the ECT procedure for monitoring. Monitoring some trends listed below may be useful. They are listed only as suggestions and illustrations.

- Procedure efficiency. Percentage of days in which the average time needed by the ECT doctor exceeded 15 minutes per patient. Record total time from when you began working in the ECT area until you completed time, divide by number of ECT patients seen.

- General efficacy. Percentage of patients whose acute ECT course exceeded 12 treatments. An initial goal might be less than 10%.

- Complication incidence. Percentage of patients whose ECT courses are stopped before clinical plateau is reached, along with reason, e.g., confusion, consent withdrawn, medical complication, financial limitation, hospital environment.

- Seizure induction success. Percentage of patients whose ECT courses are stopped because seizures of adequate quality were not obtained.

- Consent. Percentage of patients who withdrew consent for ECT after giving it, along with when

it was withdrawn, e.g., before first ECT, before both patient and doctor identified remission, before just doctor identified remission, after remission, after course.

- Medical complication incidences: headache, nausea, hypertension or tachycardia lasting over 2 minutes after ECT, postictal agitation, postictal delirium, delayed awakening, other.

- Anxiety disorder incidence post-ECT: Percentage of patients examined for anxiety disorder at end of ECT and 3-8 weeks later. Percentage of patients with new diagnosis of anxiety disorder found at end of ECT and 3-8 weeks later.

- Prophylaxis: Percentage of ECT patients with depression placed on SNRI, tricyclic, bupropion, lithium, or MAO-inhibitor after ECT for prophylaxis.

- Thymatron® Instrument Integrity. On selected days (e.g., the first treatment day of the month or the week) test the integrity of the stimulus output with the Somatics Ectobrain-II instrument. The Ectobrain-II instrument indicates if the stimulus is correct. The Thymatron® instrument has built-in patented independent circuitry to monitor its own output, and every stimulus is measured and monitored accordingly, but this monitoring is entirely internal and automatic. Moreover, monitoring with the Ectobrain-II instrument can be done whenever any staff member communicates a doubt or question about the stimulus delivered by the Thymatron® device. Such a question naturally arises when seizure induction does not occur readily with one or more patients. Having an Ectobrain-II instrument on hand can prevent unnecessary unavailability of the ECT instrument by sending it for inspection when it is actually working properly.

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This publication is distributed by Somatics LLC. http://www.thymatron.com 800-642-6761

Conrad Swartz, Ph.D., M.D. has published clinically useful findings about ECT and psychotropic medications. He is a past president and elected Fellow of the International Society for ECT and Neurostimulation, and Emeritus Professor of Psychiatry, Southern Illinois University School of Medicine. His publications identified the enterohepatic circulation as the main repository of common medications in the body, hypogonadism as a risk for myocardial infarction and bone fracture in aging men, methods to prevent ECT postictal agitation, and ECT stimulus dose as charge times current cubed. Dr. Swartz received Bachelors of Engineering from Cooper Union, M.S. from Caltech, and Ph.D. In Engineering and then M.D. from the University of Minnesota, Minneapolis.

Appendix: INSTRUCTIONS TO PATIENTS

as published 2019 by Somatics LLC, Venice, FL in the Thymatron® System IV User's Manual.
According to the FDA, instructions to patients are provided by ECT device manufacturers to physicians to give.

Safety Information

Precautions: For patients with brain tumor, brain aneurysm, myocardial infarction, coronary insufficiency, heart failure, or aortic aneurysm medical specialists in Neurology or Cardiology should be consulted to determine additional precautions needed, if any.

Warning: When used as intended, this device provides short-term relief of symptoms. The long-term safety and effectiveness of ECT treatment has not been demonstrated.

Warning: ECT device use may be associated with disorientation, confusion, and memory problems. ECT treatment may be associated with disorientation, confusion and memory loss, including short-term (anterograde) and long-term (autobiographical) memory loss following treatment. Based on the majority of clinical evidence, these side effects tend to go away within a few days to a few months after the last treatment with ECT. Although the incidence of permanent cognitive memory loss was not supported by the clinical literature, some patients have reported a permanent loss of memories of personal life events (*i.e.,* autobiographical memory).

Patients treated with ECT may experience manic symptoms (including euphoria and/or irritability, impulsivity, racing thoughts, distractibility, grandiosity, increased activity, talkativeness, and decreased need for sleep) or a worsening of the psychiatric symptoms they are being treated for. Depressed patients have committed suicide even after ECT. Partial decrease in depression may increase risk of suicide.

The physical risks of ECT may include the following (in order of frequency of occurrence): Pain/somatic discomfort (including headache, muscle soreness, and nausea); Skin burns; Physical trauma (including fractures, contusions, injury from falls, dental and oral injury); Prolonged or delayed onset seizures; Pulmonary complications (hypoxemia, hypoventilation, aspiration, upper-airway obstruction); Cardiovascular complications (cardiac arrhythmias, heart attack, high or low blood pressure, and stroke); and death.

Clinical testing

Since FDA cleared the Somatics Thymatron® ECT device for marketing in 1984, more than 4,300 Thymatron devices have been used worldwide. Numerous clinical studies have evaluated use of the device in treating severe depression. Rates of improvement for depressive illness have ranged from negligible to complete recovery, with most studies reporting substantial improvement. No reports of significant clinical worsening have occurred.

No major adverse events or deaths have been reported in the medical literature naming the Thymatron® ECT device, nor have significant adverse effects been found in the numerous brain imaging and brain biochemical studies performed to assess the impact of these treatments.

The primary adverse effects of ECT with a Thymatron® device have been limited to relatively short-term, reversible disturbances of verbal and nonverbal memory and general cognition, and most studies actually show improvement in these functions several weeks or months after treatment. A single study found impairment in spatial recognition memory continuing one month after ECT.

Typical course of treatment:

The Thymatron® System IV works while the patient is unconscious from anesthesia, by delivering a brief, controlled dose of electric current to the scalp in order to induce a brain seizure. This seizure changes chemicals in the brain called neurotransmitters, which are understood to be effective in treating depression and catatonia. During the seizure, the device measures the brain, muscle, and heart response to the electrical stimulus. The seizure usually ends by itself after a minute or two; if not, it may be halted by the clinician via administration of a known medication. A typical course of treatment is two to three sessions a week for two to three weeks. Some patients require a longer treatment course.

Potential benefits:

Potential benefits of ECT include relief of the symptoms of catatonia, major depressive disorder, or bipolar disorder for which the treatment was given.

Alternative treatments:

Alternatives to ECT treatment, depending on the primary diagnosis and the patient's condition, may include antidepressant drugs, antianxiety drugs, lithium, antipsychotic drugs, behavioral therapy, and/or psychotherapy.

Self-Assessment Questions:

1. When the electrical stimulus is delivered to the patient you may feel it if (choose one):
A. You touch the hospital cart the patient is on
B. You touch the patient's head with your hand
C. You hold the patient's shoulder and leg
D. You press the "Treat Button" while touching the patient
E. None of the above

2. ECT patients may see other patients receiving ECT because they will forget it later: (True or False):

3. What is the main purpose of suppressing consciousness with anesthesia at ECT? (choose one):
A. So the patient will not feel the electricity
B. So the patient will not remember anything about the treatment
C. Protecting the patient
D. So the patient will not remember the muscle paralysis

4. An ordinary and desirable peak heart rate during ECT under anesthesia with methohexital, thiopental, or etomidate is in which range:
A. Above 130 bpm
B. 120-130 bpm
C. 110-120 bpm
D. under 110 bpm

5. An ordinary and desirable heart rate 10 minutes after the ECT session is in which range:
A. Above 135 bpm
B. 120-130 bpm
C. 110-120 bpm
D. under 110 bpm

6. When the electrical stimulus is applied, what may be in the patient's mouth (choose one best):
A. Dentures
B. A compressible mouth protector
C. Nothing
D. A plastic Guedel airway
E. An endotracheal tube

7. After ECT treatment but before awakening the patient yells and flails. This is probably: (choose one):
A. An ordinary reaction to ECT
B. Postictal agitation
C. Postictal delirium
D. A nightmare during anesthesia
E. Tardive seizure

8. After the ECT seizure each patient is routinely turned on his side (True or False):

9. After the ECT seizure all the following are routinely monitored for return to baseline before the patient leaves the ECT area except (choose one):
A. Heart rate and rhythm
B. Blood pressure
C. Respirations
D. Level of consciousness and orientation
E. Temperature

10. When given for sleep these medications may interfere with ECT efficacy except for:
A. Promethazine (Phenergan)
B. Zolpidem (Ambien)
C. Eszopiclone (Lunesta)
D. Temazepam (Restoril)
E. Flurazepam (Dalmane)

ANSWERS LISTED BELOW

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Answers:
1. C. Touching the patient with 2 hands makes a circuit through your body.
2. False
3. D
4. A
5. D
6. B. Teeth must be protected
7. B
8. True
9. E
10. A