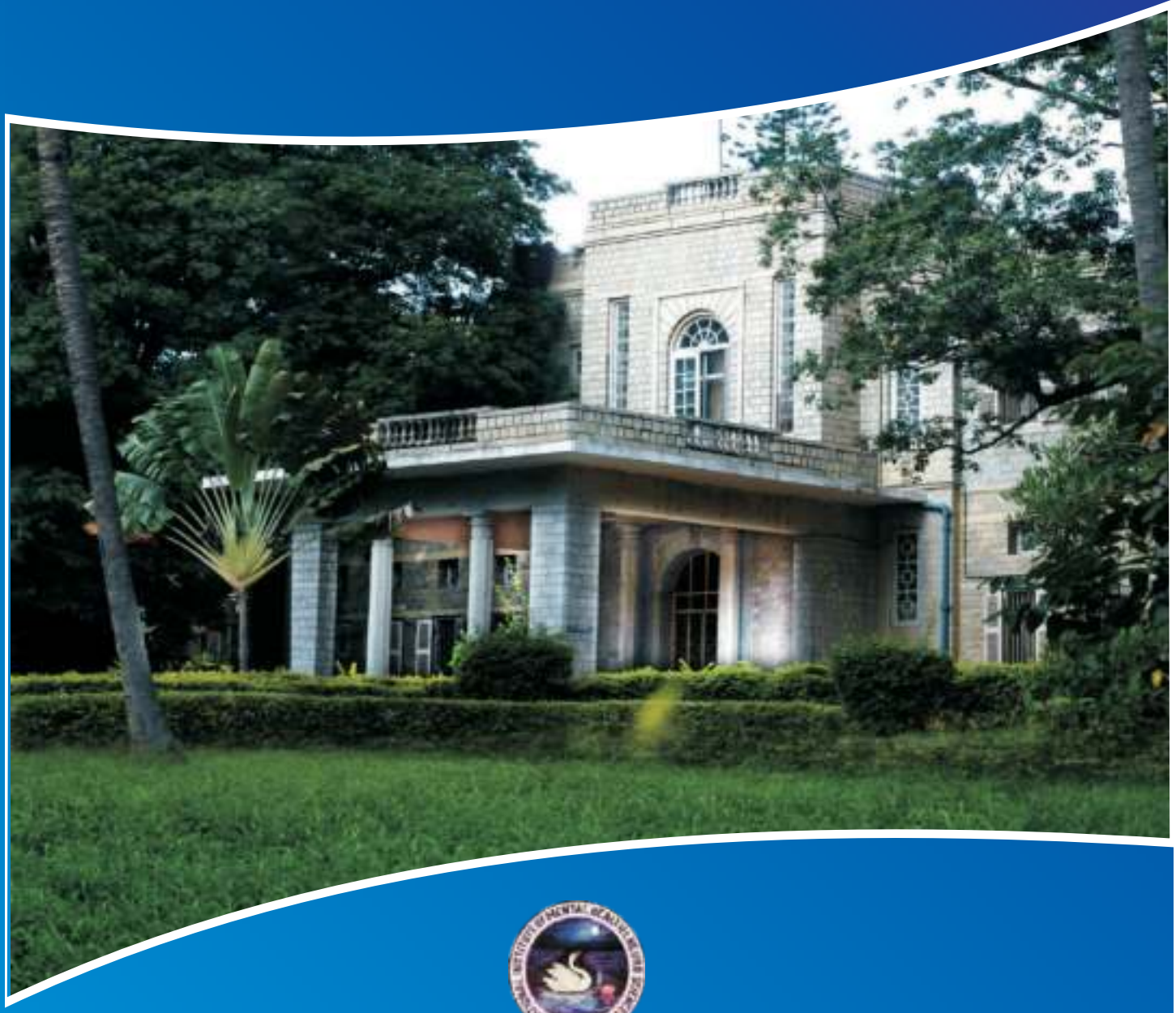
ECT ADMINISTRATION MANUAL

2ND EDITION



ECT-TEAM, DEPARTMENT OF PSYCHIATRY
NATIONAL INSTITUTE OF MENTAL HEALTH & NEURO SCIENCES (NIMHANS)
BANGALORE- 560029

NIMHANS ECT SUITE

ECT ADMINISTRATION MANUAL

ECT-TEAM, DEPARTMENT OF PSYCHIATRY
NATIONAL INSTITUTE OF MENTAL HEALTH & NEURO SCIENCES (NIMHANS)
BANGALORE- 560029

ECT ADMINISTRATION MANUAL (2nd Edition):

Bangalore N Gangadhar, A Shyam Sundar, Jagadisha Thirthalli, Shivarama Varambally, Kesavan Muralidharan, C Naveen Kumar, Preeti Sinha, Biju Viswanath

ISBN No.	**:**	**81-86442-00-X**
NIMHANS Publication No.	**:**	**93**
Number of Copies	**:**	**1,000**
Year of Publication	**:**	**2013 (Commemorating 75 Years of ECT)**
Number of Pages	**:**	**VIII + 62**
1st Edition	**:**	**1997**
2nd Edition	**:**	**2013**
Price	**:**	**Rs. 200/-**

Printed with support from Dr. Ramachandra N Moorthy Foundation for Mental Health and Neurological Sciences

Design & Printed at :
Navarathna Printers
35, 3rd Cross, Sudhamanagar
Bangalore - 560 027
Phone : 2222 3538 Fax : 4149 5048
e-mail : navarathnaprinter@gmail.com

FOREWORD

Electroconvulsive therapy (ECT) is an important modality of treatment in psychiatry. It occupies a unique position in psychiatric practice– while it is the first-choice of treatment and is life-saving in patients with high suicidal risk; it is also prescribed in situations where other treatment methods have failed. It is also one of the oldest physical methods of treatment, which has stood the test of time. Despite a chequered history, ECT has remained an essential part of any psychiatric setup across the globe even today.

It is likely that, in 1938, Italian Neuropsychiatrists Cerletti and Bini may not have envisioned the massive impact that ECT was to have on the treatment of psychiatric disorders over the next 75 years. Since then, ECT has seen a number of advances. Use of anaesthesia, refinements in the electrical stimulus and electrode placement, careful selection of patients and meticulous monitoring of safety issues have considerably reduced the stigma associated with it over the years. Significant strides in understanding the mechanism of action of ECT and its adverse cognitive effects have given a more scientific outlook to ECT practice.

Since 1947, when the first ECT machine was acquired, NIMHANS has been at the forefront of practice and research and in the field of ECT, not only in the country, but also in the world. Successive generations of academic psychiatrists from the Institute have contributed significantly to research in this area. NIMHANS was instrumental in the development of an indigenous pulse-wave ECT machine.

A culmination of these efforts resulted in the first edition of "ECT Administration Manual", which was published in 1997. It received rich accolades from academics, practitioners and students alike and has remained the ready reckoner for good ECT practice in the country. Its popularity and persistent demand by its readers, coupled with further advances in ECT research over the last decade, has spurred the ECT team from the Department of Psychiatry at NIMHANS to bring out its second edition. This edition covers the advancements

of practical significance in the field of ECT fairly comprehensively. Some of the highlights of this edition include newer electrode placements, monitoring and reducing cognitive adverse effects of ECT and updated information on all other aspects of ECT, written in simple language that makes it easy to comprehend for both students and experienced professionals.

I congratulate the NIMHANS team for bringing out this very useful manual and wish them all the success.

Dr. P. Satish Chandra
Director and Vice Chancellor
NIMHANS , Bangalore

TABLE OF CONTENTS

BLANK

INTRODUCTION

Electroconvulsive therapy (ECT) was introduced 75 years back by Italian neuropsychiatrists Ugo Cerletti and Lucio Bini. The technique of ECT has evolved considerably and has proven to be a safe and effective treatment when used judiciously. The introduction of modified ECT, brief pulse stimulation, alternative methods of electrode placement and use of EEG monitoring have decreased the adverse effects and increased the acceptability of treatment. Despite the progress, there is marked variation in the practice of ECT worldwide, with a general trend of underutilization, inadequate training and poor adherence to guidelines (Leiknes, Jarosh-von Schweder, & Høie, 2012). It is essential, therefore, that psychiatrists should have opportunities to critically review their ECT practice, update their skills and consciously maintain adequate standards.

PART – I :
ECT STIMULUS

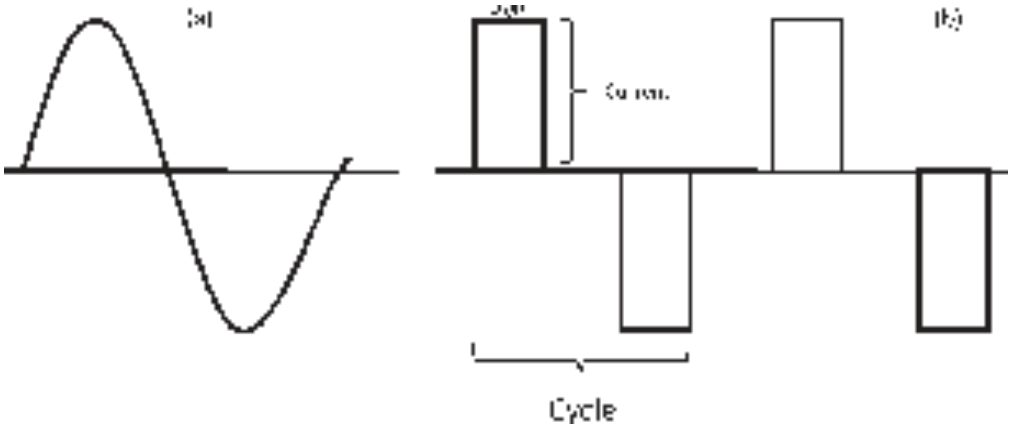
The objective of ECT is to electrically induce a generalized seizure, which is an essential but not sufficient ingredient for therapeutic effect. It is now established that additional factors like electric dosage relative to the seizure threshold; and anatomical positioning of electrode determine the efficacy of ECT (Merkl, Heuser, & Bajbouj, 2009). It is important to understand the role of electrical stimulus parameters, as they exert neurobiological effects that determine the efficacy and adverse effects (Peterchevet al, 2010). The twin goals are to maximize therapeutic effect and minimize adverse effects, especially cognitive morbidity. Unfortunately, the procedures which enhance the former often increase the latter too. Hence, an optimal balance between the two must be attempted by combining appropriate choices on the following parameters:

1. Waveform of the stimulus
2. Stimulus relative to threshold
3. Electrode placement
4. Frequency of treatments
5. Number of sessions

1. Waveform of the stimulus

Earlier ECT machines delivered current in the form of sine waves (Fig.1a). The slow rising and trailing edges of the sine wave do not produce efficient cerebral stimulation. This delivers substantial amounts of electrical stimulation below seizure threshold and continues stimulation even while the neuronal tissue is in post-depolarization refractory period. This may increase the cognitive adverse effects without enhancing the benefits. Further, the slow rising current increases the neuronal threshold. Hence, the sine wave has been modified to

eliminate the rising and trailing edges, retaining only the peaks in the form of 'rectangular' pulses (Fig. 1b). Stimulus is given in the form of brief pulses (0.5ms) with no stimulus in between the pulses. Intermittent stimulation using pulses is more efficient as it allows the tissue to recover from post-depolarization refractory period. Brief pulse ECT is able to induce seizures with substantially



less charge and energy compared to sine wave.

Figure 1: Shapes of waveforms of stimulus (a) sine wave (b) pulse wave. PW= pulse-width in milliseconds; current in Amperes; a pair of pulses in the opposite directions make a cycle.

Older sine wave ECT devices allowed the setting of stimulus voltage. However, in such devices, the delivered current varied depending on the impedance. Despite constant voltage, the predicted current strength of the stimulus is not ensured even within the same patient across sessions. It is hence necessary to obtain the same by special instrumentation and hold the strength of the current in the stimulus constant (e.g., 800 mA). The stimulus sources available today evaluate the inter-electrode impedance and dynamically adjust the voltage to ensure a constant current throughout the stimulus application.

Table-1 elucidates the differences between a typical voltage-constant sine wave and a current-constant brief pulse stimulus used in ECT. The latter is

more efficient, needing less than a third of stimulus dose as compared to the

	Current	Voltage	Frequency	Width	Duration	Impedance	Energy	Charge
PULSE	0.8 A	100 V	100 PPS	1 ms	1 sec	200 Ω	12 J	80 mC
SINE	0.8 A	420 V	50 Hz	-	0.5 sec	200 Ω	50 J	200mC

former if given to a patient with same impedance (200 Ohms, in this example).

Table-1: Comparison of waveforms

NOTE: At conventional threshold settings, SINE: PULSE = 3:1

Parameters which can be chosen by the operator are in bold face.

Voltage is the electromotive force between the two electrodes which drives the flow of electrons. The unit is Volt. Over 90% of this force drops as a result of the impedance offered by the scalp and skull. Across brain tissue less than 10% of applied voltage exists.

Current is the flow of electrons between the electrodes which more truthfully reflects the stimulus exposure to brain tissue. The unit is Ampere.

Impedance is the resistance (Ohm) offered for the flow of electrons by the matter between the two electrodes.

The Ohm's law governs the flow of electrons: **Current = Voltage/ Impedance**.

While using the earlier ECT devices of voltage-constant sine-wave stimuli, the clinicians expressed the stimulus dose in units of volts delivered for a known duration. This however, poorly reflects the actual stimulus exposure to the brain.Contemporary methods of stimulus dosimetry supercede older convention. The values of current/voltage are uniform across the brief pulses and are

maintained throughout pulse. Whereas in sine wave stimulus, the amplitude of current or voltage gradually rises and trails in each cycle; peak amplitude is reached only for a fraction of the cycle.

In summary, brief pulses of constant current offer better stimulus economy than the conventional constant voltage sine wave stimuli. Comparative studies have shown that brief pulse ECT is as effective as sine wave ECT (American Psychiatric Association, 2005). Brief pulse ECT produces lesser cognitive dysfunction than sine wave ECT as measured by - (a) postictal disorientation, (b) postictal EEG suppression, and (c) standard memory tests (short term; <3 months post- ECT). Hence, brief pulse stimulus is recommended by most guidelines (American Psychiatric Association, 2005; Royal College of Psychiatrists, 2005).

The following parameters can be manipulated in a brief pulse ECT:

Pulse width: Pulse width is a measure relevant only to pulse wave stimuli. Pulse widths in the range of 0.5 - 2 ms (known as brief pulse) are commonly used. The optimum duration of a pulse to produce neuronal depolarization is around 0.2 msec. Longer duration will unnecessarily provide the current during the refractory period which increases the cognitive side effects without improving the efficacy. Hence, briefer pulse durations are preferred. Recently ultra-brief ECT (pulse width <0.5 ms) has been proposed to decrease the cognitive adverse effects. However, its therapeutic superiority over brief pulse ECT has not been conclusively demonstrated (Loo et al, 2012).

Stimulus duration: Stimulus duration is the total period for which the stimulus train is applied. In the sine wave stimulus; it is usually less than 1 second and in pulse wave it is between 0.5 and 8 seconds. It should be remembered that in the brief pulse ECT, the actual duration of stimulus will be even lesser if the inter-train interval (where stimulus is not provided) is excluded. It is calculated by this equation (pulse width x pulses per second x total length of stimulus train) and the value obtained is usually less than 10% of the total duration of current.

Frequency: Frequency refers to number of cycles per second and the unit is Hertz (Hz). In the transformer-based sine wave stimuli, this is always the line frequency (50Hz or 60 Hz). In brief pulse ECT, the frequency can be varied by the operator. The term pulses per second (PPS) is preferable in this form of stimulus. It has been seen that lower frequency of current stimulation is associated with lower seizure threshold, but it does not have an effect on seizure duration, ictal cardiovascular responses and therapeutic outcome (Girish et al, 2003; Kotreshet al, 2004). However there is no consensus on the optimum frequency of stimulation (Peterchev et al., 2010). As per the current understanding, optimizing other parameters like pulse width and stimulus duration may be simpler for routine use.

Directionality & Polarity: The stimulus used in ECT is usually bidirectional, where the direction of the current alternates with each successive current, as shown in Figure 1. There has been a renewed interest in unidirectional current, as it has been shown to decrease the seizure threshold (Spellman, Peterchev & Lisanby, 2009). With unidirectional current, it is possible to provide specific polarity of currents (anodal v/s cathodal) to specific brain regions. Varying the polarity of current may lead to differential effects on the underlying neurons as seen in other brain stimulation techniques. For example, in transcranial direct current stimulation (tDCS), anodal current potentiates the activity of the underlying nervous tissue whereas cathodal current has the opposite effect (Nitsche et al., 2005). Modulation of polarity of the ECT stimulus is currently being explored, but it is still at a nascent stage.

Pulse amplitude: The amplitude of the delivered current is an important determinant of the volume of nervous tissue that is directly stimulated by the ECT. Modern ECT machines usually maintain the current constant at 800 or 900 mA to ensure that adequate charges are delivered. Some machines allow some degree of manipulation of the current delivered. Currents lower than 800 mA do not result in optimal electric fields. Likewise, currents above 800 mA do not increase the electric fields across brain tissue. However, it has been seen recently

that current amplitudes less than 800 mA can elicit adequate seizures (Peterchev et al., 2010). The clinical significance of manipulating the amplitude of current has not been demonstrated conclusively.

Charge: Charge is the total amount of electrons traversing the inter-electrode tissue including the brain. It is the product of current and actual duration of stimulus. Its unit is Coulomb or milli-Coulomb(mC). Stimulus estimation by charge (coulometry) is used in contemporary ECT practice. In brief pulse ECT, charge = current x pulse width xpulse frequency x length of train. For example ; for a current of 0.8 A, pulse width of 1 ms, pulse frequency of 100 and a train length of 1.5 ms, the charge provided will be 120 mC. The length of the train is usually varied to provide the desired charge by keeping the other parameters constant.

2. Stimulus intensity

The stimulus intensity (measured as charge in milli-Coulomb) is an important determinant of therapeutic efficacy and cognitive adverse effects of ECT. Treatment with stimulus intensity barely above seizure threshold may be less effective despite production of generalized seizure. This is more apparent for unilateral ECT. It is recommended that the stimulus intensity should be modestly to moderately suprathreshold (1.5 to 2.5 times threshold) for bilateral ECT. For unilateral ECT, the stimulus should be moderately to markedly suprathreshold (2.5 to 6 times above seizure threshold)(American Psychiatric Association, 2005; Enns et al, 2010). Unilateral ECT using lesser stimulus intensities is less effective than bilateral ECT.

Cognitive morbidity is not as much dependent on the absolute dose per second, as on the degree by which the dose exceeds threshold. Hence, the stimulus intensity should be titrated carefully to maintain a balance between efficacy and side-effects. It is recommended that the stimulus titration is carried out from the lowest setting given in the ECT devices. Usually, this corresponds to a dose of 30 mC of charge. Increments are made by 25-50% until the seizure occurrence is confirmed. There is wide variation in seizure threshold across patients; from 5 to 40 folds in different studies. Formula based titration of

dosages is possible in some machines. Important factors that determine seizure threshold include electrode placement, gender, age, size and shape of skull, anesthetic dosage, and concomitant medications (van Waarde, Verwey, & van der Mast, 2009; Girish et al., 1998). Regression equations have been derived to predict seizure threshold using age as an independent variable for both unilateral and bilateral ECT (Gangadhar et al., 1998; Girish et al., 2000). However, these equations either overshot or underestimated thresholds in a sizeable minority of the patients (Gangadharet al, 2000). Titration method is recommended for clinical use, while these formulae can be used for guidance.

Seizure threshold increases with increasing age and hence the titration should be slower in children. Once the seizure threshold is determined, subsequent ECTs should be provided at sufficiently suprathreshold intensity as described above. Seizure threshold increases during the course of ECT. If ECTs are to be continued beyond two weeks, it is recommended to re-estimate the threshold in the first ECT session of third week and readjust the dose accordingly. Subsequent stimulations should take into account the new seizure threshold to calculate stimulus intensity. For example, for a person who had an initial seizure threshold of 60 mC, threshold may increase to 120 mC on later stimulations. Subsequent stimulations should use the later value to calculate the suprathreshold dosage.

3. Electrode placement

In unilateral ECT (ULECT), stimulus is provided to only one hemisphere (usually the right) to minimize cognitive dysfunction. ULECT given at moderately suprathreshold doses (2.5 times) is inferior in efficacy compared to bilateral ECT (BLECT). Increasing the stimulus intensity to markedly suprathreshold dose (6 times the threshold) improves the efficacy of ULECT at the cost of higher cognitive side effects. The onset of action may also be slower with ULECT. Brief pulse ULECT, sine wave ULECT, brief pulse BLECT and sine wave BLECT cause increasing levels of cognitive dysfunction in that order.

When there are serious concerns about potential cognitive morbidity, either

because of patient characteristics (e.g. elderly, brain-damaged, children, etc.) or instrument limitations (e.g. sine wave stimulus), ULECT should be preferred. In ULECT, one electrode is placed on the fronto-temporal site i.e., one inch above the midpoint of an imaginary line linking the outer canthus and the external auditory meatus. The other electrode is applied one inch lateral to the vertex position of the 10-20 system, on the same side (usually right). Both electrodes are placed on the non-dominant side (usually the right side).

However, bilateral electrode application is recommended if – a) the diagnosis is mania or schizo-affective disorder, (b) the patient has not satisfactorily responded to ULECT (usually after 6 ECTs), or (c) when more urgent response is essential (suicidal, starving etc). Bitemporal placement is the standard method of stimulation in bilateral ECT, where electrodes are placed on the fronto-temporal sites (as described earlier), one on each side. Alternatively, bifrontal ECT(BFECT) can be used where electrodes are placed 5 cm vertically above the outer canthus of each eye along an imaginary vertical line perpendicular to a line connecting the pupils. Bifrontal ECT has been found to be equally effective to bitemporal ECT (BTECT), with possibly lesser cognitive side effects (Phutane et al, 2013). Electrodes with a concave surface have been specifically designed for bifrontal ECT.

4. Frequency of treatments

ECT is generally administered twice or thrice a week. More frequent ECTs add to cognitive morbidity. A recent meta-analysis showed that twice weekly ECT has similar efficacy to thrice weekly ECT and is associated with lesser number of sessions (Charlson et al., 2012). Twice weekly ECT seems to have the best balance between therapeutic outcome and adverse effects in the immediate treatment of major depressive disorder while using bilateral ECT (Gangadhar & Thirthalli, 2010). However, it is recommended that the patient be evaluated both for therapeutic and adverse effects (especially cognitive side-effects) following each treatment. This would help optimize the treatment frequency.

5. Number of sessions

There is no fixed number of sessions. The number should be decided based on

the degree and rate of clinical improvement and adverse effects. A typical course of ECT for depression consists of 6 to 12 sessions. Patients with schizophrenia require more treatment sessions. The number of treatments required to achieve clinical response varies widely between patients. Once the patient achieves remission of symptoms, the ECT course can be terminated. If a patient does not show noticeable improvement within 6 sessions, a change in technique may be considered. These include increasing stimulus intensity, switching from unilateral to bilateral ECT, etc. If the patient shows plateauing of clinical response despite adequate duration (6-12 sessions) and changes in technique, the course may be terminated. Unlike pharmacotherapy, the treatment can be terminated abruptly (American Psychiatric Association, 2005).

Continuation ECT can be considered in rare patients for whom pharmacotherapy prophylaxis is ineffective or intolerable and sometimes based on patient preference. Continuation ECT begins after the index course, lasts up to 6 months, and is designed to prevent relapse of the episode while maintenance ECT is provided to prevent further episodes for longer periods (Andrade & Kurinji, 2002; Petrides et al , 2011). For continuation and maintenance ECT, the frequency of ECT is slowly tapered off to once a month. The patient should be assessed frequently for adverse effects and the treatment plan should be updated. Pre-anesthetic evaluation should be conducted at least once in 6 months for maintenance ECT (American Psychiatric Association, 2005).

The above considerations are however applicable largely to depression. Similar research in non-depressive patients is lacking. In particular, there is a need to evolve standards of ECT parameters in mania and schizophrenia. There is some evidence to suggest that bifrontal ECT is superior in terms of efficacy and adverse effect profile in patients with mania and schizophrenia (Hiremani et al, 2008; Phutane et al, 2013).

PART – II :
ECT PROCEDURE

Administration of ECT without a muscle relaxant is known as unmodified ECT. In modified ECT, anesthesia, muscle relaxant, and the seizure-eliciting electrical stimulus are administered in the same order (Andrade et al., 2012). Use of a muscle relaxant prevents musculoskeletal injuries resulting from peripheral seizures. The anesthetic agent induces sleep and prevents anxiety associated with the cessation of breathing due to a muscle relaxant. Other medications are also provided as needed.

Preparation of patient:

Before each treatment, the patient should be assessed for adherence with the pretreatment orders. The patient should avoid solid food for at least 6 hours before treatment. Moderate amounts of clear fluids can be taken till 2 hours before treatment. Oral medications should be taken 2 hours before treatment with sips of water. The patient should be encouraged to pass urine to prevent bladder rupture during ECT. Dentures, jewellery, hair clips, contact lenses and hearing aids should be removed. Hair should be dry and clean. Damp hair and presence of cream may lead to short-circuiting of the current over scalp.

Modification Procedures:

Short acting barbiturates are commonly used as induction agents. Methohexitane (0.5-1 mg/kg body weight) is recommended as it has been shown to have minimal effect on seizure threshold, while ensuring quick induction and recovery (Mayo et al, 2010). Thiopentone (3-5 mg/kg) is also commonly used, but may be associated with shorter seizure duration and increased hemodynamic changes (Saito, 2005). Alternate anesthetic agents and their properties are given in Table 2. Succinylcholine (0.5-1 mg/kg) is the preferred muscle relaxant due to its rapid onset, short duration of action, and rapid recovery (Mirzakhani et al, 2012). Higher dose of succinylcholine (1 mg/kg) is more effective in modifying the peripheral convulsion (Murali et al., 1999). However, the time to recovery may be longer. It is recommended that 1 mg/kg of succinylcholine dose be used

in the first ECT session. For subsequent sessions, the dose may be altered, depending on the response for optimal motor seizure modification (Murali et al., 1999)(Mirzakhani et al., 2012).

Table 2 Alternate anesthetic agents

Anesthetic agent	Dosage	Properties
Propofol	0.75-1 mg/kg .	Decreases the hemodynamic changes associated with ECT, but decreases seizure duration and the injection may be more painful.
Etomidate	0.15-3 mg/kg	Increases seizure duration & less likely to lead to hypotension, but the sympathomimetic responses may be slightly higher. May be preferred in patients with congestive cardiac failure and in people with very high seizure threshold.
Ketamine	2-3 mg/kg	Accentuate the acute hemodynamic changes associated with ECT due to its sympathomimetic property. May cause hypertension and produce altered sensorium in the recovery period. It may be preferred in patients where it is difficult to produce any seizure activity despite maximal charge delivery.
Sevoflurane	Inhalational anesthetic	Slow onset; reduces the hemodynamic changes secondary to ECT.

Before trolleying the patient into the ECT suite, an IV cannula is inserted in the left forearm and is maintained till the patient recovers. Patient is allowed to breathe 100% oxygen through face mask for 3 minutes at 15 -20 breaths per minute.Thiopentone in 2.5% dilution mixed with atropine is injected from the same syringe. Longer periods of oxygenation may be needed in patients with myocardial infarction and brief seizures. This is followed by succinylcholine using a different syringe. Succinylcholine should be given only after complete loss of consciousness. Before and after succinylcholine, the IV cannula is flushed with saline.

After injecting the muscle relaxant, the patient is ventilated with 100% oxygen under positive pressure until he is totally paralyzed and the fasciculations have ended. Use of mere atmospheric air instead of 100% oxygen or fewer or no ventilations before stimulus can result in lower oxygen saturation as revealed by pulse oximetry. Pulse oximetry is recommended at least in medically compromised patients, e.g. in patients with Chronic Obstructive Pulmonary Disease (COPD), cardiovascular problems including ischemic heart disease. Ventilating the patient prior to stimulation reduces anoxia during seizures. A bite block is placed and the stimulus is administered. The block should be made of flexible material. Rigid blocks like the Guedel airway are avoided due to risk of tooth fracture. Positive pressure ventilations should be resumed after stimulation, continued throughout the seizure and stopped only when the patient has resumed spontaneous and regular respirations.

Some patients experience muscle aches post-ECT – usually as a result of intense fasciculations with succinylcholine. A small dose of atracurium about 5 mg prior to succinylcholine is recommended in such cases. Non-depolarising neuromuscular blockers like atracurium (0.3–0.5 mg/kg) and rocuronium (0.6–0.9 mg/kg)are also preferred inpatients with actual or potential hypokalemia (burns, prolonged immobilization), pseudocholinesterase deficiency, organo-phosphorous poisoning (leads to inhibition of cholinesterase), concomitant cholinesterase inhibitor use, history of malignant hyperthermia and neuromuscular disease. However the muscle relaxation produced by these

agents is long lasting and may not be complete. They have to be reversed with a cholinesterase inhibitor like neostigmine.

Atropine (0.6 mg) given along with intravenous anesthetic helps in preventing vagal effects of electrical stimulation on heart. Glycopyrrolate (0.2-0.4 mg/kg i.v.) is an alternative which has the advantage of limited CNS effects. When given intravenously, it should be given 2-3 minutes before seizure induction. Some advice its use 30 minutes before ECT to dry up secretions. It may not be required in patients who continue on certain psychotropic drugs, which by their anti-cholinergic effects would cause sufficient drying of secretions. It is a risk that a waiting patient may drink water if atropine causes excessive dryness of mouth. If ECG monitoring is available, use of atropine as a routine pre-anesthetic may be unnecessary.

Electrode Application

Scalp preparation before applying the electrodes is essential. Non-greasy scalp surface is recommended. The stimulus electrode skin contact is established by using electrode jelly. Saline-soaked pads should not be used as conductant on electrodes. The saline may flow down and short-circuit the two electrodes, particularly in unilateral ECT. Jelly is preferable as it remains in the electrode site besides having less corroding effect on the electrodes.

Both electrodes should be held firmly by the treating psychiatrist. The forceps type of electrodes is discouraged as – (a) this cannot be used for unilateral application and also (b) the head contour variations can lead to poor electrode-skin contact. The treating psychiatrist should operate the stimulus release switch, which is optimally provided on the electrode handle itself.

Seizure Monitoring

It is imperative that the response to stimulation – the seizure– is monitored and its occurrence confirmed. Unmodified ECT is a dramatic event and the standard tonic-clonic seizure is not missed. Modified ECT however demands some method of seizure monitoring. Available method sinclude: Cuff method and EEG monitoring. However; it has been seen that the length of the seizure

duration either measured through EEG monitoring or visualization of tonic clonic movements is not related to clinical efficacy. The goal of monitoring is to ensure that a generalized seizure occurs as described below and to monitor for complications like prolonged seizure.

Cuff method

Way back in 1960s, soon after introduction of anesthesia for ECT, Hamilton demonstrated that isolating the limb using a BP cuff and preventing the muscular relaxation of this limb allows satisfactory recording of convulsions. The last clonus on the cuffed forearm heralds seizure termination. Rarely though, clonic movements may be seen to last longer in other parts of the body. In such event, the last clonus anywhere else is considered to herald the end of seizure. Convulsive movements of the face alone may result from focal seizure activity and does not ensure adequate seizure. The cuff method should be avoided in people with peripheral vascular disease, deep vein thrombosis or sickle cell anemia.

Technique

1) Place the BP cuff (preferably between knee and ankle). If unilateral ECT is used cuff should be attached to the right side (ipsilateral) to confirm generalization of seizures.
2) Just prior to the injection of succinylcholine inflate the cuff to a pressure 50 - 80 mm Hg above systolic blood pressure.
3) Observe that fasciculation after succinylcholine injection, which appear in other parts of the body do not appear in the isolated limb.
4) Apply the stimulus and note the time (start the stopwatch).
5) At the start of tonic phase of seizure release the cuff.
6) Record the total duration of tonic and clonic phases till the last clonic movement

EEG monitoring: Although not mandatory, it has distinct advantages and is recommended where possible as it is the most direct measure of cerebral activity

available. The length of the convulsions measured by direct visualization is approximately 70% that of EEG seizures (Mayur et al, 1999). EEG monitoring during ECT helps to detect both adequacy of cerebral seizure and also to detect prolonged seizures(Girish, Gangadhar, & Janakiramaiah, 2002). EEG is liable to artefacts because of peripheral movements and other technical problems.

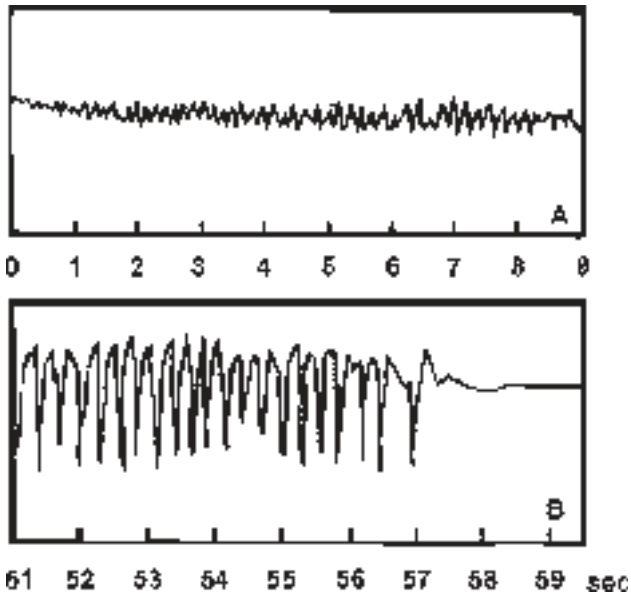
The fronto-mastoid montage is preferred. The recording electrode is placed on the frontal area. The reference electrode is placed on the ipsi lateral mastoid and the ground electrode on the centre of forehead. If a single channel is used the left side is preferred as it ensures that seizure generalization has occurred in case of unilateral ECT. A two channel EEG is preferred as it helps to assess generalized seizures and in differentiation from artefacts (Royal College of Psychiatrists, 2005).

The EEG amplifier built into the ECT machine has usually the following settings: gain- 1000-2000, low pass filter- 40 Hz, high pass filter – 0.5-2 Hz and a 50 Hz notch filter. However, if the ECT team uses a conventional independent EEG amplifier, care should be taken to use the amplifier settings described above, particularly the lowest gain. The typical ictal EEG pattern is described in Table 3.

Table 3. Ictal EEG activity

	Duration	Wave pattern
Phase 1	1-3 seconds	High frequency low amplitude
Phase 2	10-20 seconds	Hypersynchronous polyspikes, 10 Hz, recruiting rhythm
Phase 3	20-40 seconds	Spike and wave, rhythmic 2.5 –3Hz ending with post-ictal EEG suppression

The EEG disconnect feature in the ECT machine de-links the EEG electrodes from the patient electronically, while the stimulus is applied. The voltages of ECT stimulus can saturate the EEG amplifier and adversely affect the EEG recording quality. The seizure onset is indicated by occurrence of spikes and poly spikes which are above 8-10 Hz in frequency. These are gradually replaced by more rhythmic spike and slow-wave discharges. The frequency of this slow-wave phase initially is about 4 Hz and reaches 3-2½ Hz.



Typical seizure EEG during ECT: A) Following the stimulus, progressively higher amplitude poly spikes of about 10 Hz occur. (B) The poly spike phase is followed by a more rhythmic (about 3 Hz) spike and wave phase of higher amplitude. Seizure ends with a fit-switch (between 57 and 58 seconds) which is characterized by abrupt cessation of EEG discharges. Seizure EEG data was digitized and re- plotted. This is a computer output of the EEG.

The seizure terminates with the last spike and wave discharge into a flat line. Sometimes, the termination is not a dramatic fit switch. The high amplitude spike and wave activity is replaced by low voltage slow waves. Its amplitude is smaller than that seen in the EEG before the stimulus application.

Either paper recording or digital monitoring are accepted methods. Standards

for the rating of these have been developed. Automated estimation of seizure duration using signal processing algorithms is possible.With ULECT, EEG seizure may last longer on the stimulated hemisphere. The discharges may also be more regular with higher amplitude on this hemisphere. BLECT produces more symmetric seizure discharges. Methods have been developed to quantify the seizure amplitude and asymmetry using signal processing algorithms. These measures may become relevant in assessing the adequacy of seizures in future. These trends in literature may mandate use of two-channel EEG. The motor seizure duration is 30% shorter than EEG seizure.

When the stimulus intensity is subthreshold or just at threshold, abortive seizure or very brief seizure occurs. When it is barely suprathreshold, a long seizure with low amplitude and absent postictal suppression is seen. When it becomes substantially suprathreshold, the seizure duration decreases but high amplitude EEG with postictal suppression is observed (American Psychiatric Association, 2005).

It is generally suggested that seizure duration below 15 seconds is inadequate. However, recent evidence suggests that seizure duration is not a marker of seizure adequacy (Mayur, 2006). Various EEG measures have been suggested to determine seizure adequacy, but none have been validated. EEG expression may be affected by various factors like medication status, treatment number, age etc. Nonetheless, the presence of high amplitude synchronous seizure activity and post-ictal suppression are associated with better clinical outcome (Thirthalli et al, 2003; Abhishekh et al., 2013). The occurrence of a generalized seizure observed either by EEG or cuff method signifies adequate treatment. If in doubt, the 15 second cutoff can be used.

Other Physiological Monitoring: Vital signs such as blood pressure and pulse should berecorded before and after ECT. ECG monitoring is considered mandatory during ECT procedure. Particularly in centers where atropine is not used routinely, this helps in recognizing vagal bradycardia. ECG monitoring has distinct merits especially inelderly patients who are more likely to have pre-existing or occult cardiac pathology. Single channel ECG monitoring of the

cardiac rhythm is usually sufficient. Other physiological monitoring includes pulse oximetry. Ventilation and oxygenation can be insufficient in the absence of pulse oximetric monitoring. Patients may remain on low pO2 levels during seizures. Pulse oximetry is hence recommended during modified ECT.

Complications during ECT: Missed seizure, inadequate and prolonged seizures are known phenomena during ECT. These are dependent on the seizure threshold. The threshold is lower in a) young subjects, b) manic patients, and c) during the first ECT session. Theophylline lowers the seizure threshold. Conversely, benzodiazepines, carbamazepine and thiopentone elevate the threshold. Effect of other psychotropic drugs on threshold is uncertain. The causes of sub-convulsion and prolonged seizure can be understood in this background.

Missed seizures: When electrical stimulation is not followed by any motor or EEG seizure activity, it is termed a missed seizure. It may be due to various factors like high impedance, inadequate stimulation, concomitant medications and other technical factors. If basic requirements like electrode contact and other technical aspects of the machine have ensured that the stimulation was done properly, then a restimulation should be attempted. It is advisable to wait for 20 seconds before restimulation to allow for delayed onset of seizures and also to ensure that restimulation does not occur in the refractory period. Re-stimulation ina situation of inadequate seizure should occur only after 45 seconds as it may cause longer refractory period. The patient should receive vigorous ventilation before re-stimulation. It should be ensured that the patient is still under anesthesia during restimulation. Restimulation should be attempted at a higher dosage (usually 25 -50 mC above the previous dosage). The number of re-stimulations allowed in each ECT session is a practice limited only by the duration of muscle relaxation. Each center may have to evolve a protocol. Rarely can the anesthetic effect last longer than 1-2 minutes, within which only 4 or 5 re-stimulations are possible. No research is available to support or refute the role of re-stimulation in the genesis of CNS adverse effects. It is well known that the seizure threshold increases with successive ECT sessions.

When there is a repeated difficulty in eliciting a seizure response, the cause should be elucidated. For example, if anticonvulsant is being prescribed, its dosage schedule may be altered accordingly. It is better to avoid antiepileptics the night before ECT. The dosage may be shifted to the morning after the ECT session. Alternate methods of managing missed seizure include:

1) Change of anesthetic agent to etomidate or ketamine

2) Vigorous hyperventilation

3) Use of EEG monitoring if not used already

4) Change to unilateral ECT as the seizure threshold may be less with unilateral ECT

5) Reducing the frequency of sessions (from 3/wk to 2/wk) can facilitate adequate seizures

6) Lower change rate by shortening pulse width or increasing interpulse intervals by decreasing frequency of stimulus.

7) Short acting opioids like remifentanil (1 μg/kg) can be combined with lower than usual doses of anesthetic agents like methohexitol or proprofol (0.5 mg/kg).

8) Injection flumazenil (0.5-1 mg i.v.) can be tried in patients on high dose benzodiazepines

9) Pretreatment with caffeine, theophylline or aminophylline (xanthines) prolongs the duration of ECT seizures but has not been clearly shown to increase efficacy. Further they increase the risk of status epilepticus. Oral long acting theophylline (200-400mg), the night before ECT or injection etophylline (70~100 mg) intravenously before anesthesia has been used for this purpose. This should be done only under EEG monitoring.

Prolonged seizure: In most situations, the seizure terminates within 100 seconds. The American Psychiatric Association task force defines a prolonged seizure if seizure duration is beyond 180 seconds (American Psychiatric

Association, 2005). Prolonged seizures result in increased cognitive deficits and hence should be prevented. Prolonged seizures can be terminated by intravenous diazepam 10 mg. or thiopentone 100-200mg. Risk of prolonged seizure is high in the first ECT session and in younger patients. EEG monitoring aids in prompt detection of prolonged seizure (Table-3). The stimulus dose has to be reduced in the subsequent ECT session unless there is evidence that such dose on earlier sessions was sub-convulsive.

Status epilepticusis another rare complication of ECT. Such patients may often be on drugs like lithium or theophylline. This complication is rarely witnessed in early sessions of ECT. Non convulsive status epilepticus may be missed in the absence of EEG monitoring.

Treatment emergent delirium is an uncommon sideeffect. Patients recovering from post ictal state may develop an acute organic psychosis. Disorientation, agitation and hallucinations mark the clinical picture. Delirium can be controlled by use of parenteral diazepam or haloperidol intravenously.

Prolonged apnea (not resuming spontaneous respirations within 10minutes) is an uncommon but serious complication during ECT warranting intubation. Genetic deficiency of pseudo-cholinesterase can be screened but not cost effective on a routine basis. When it is suspected on the basis of demographic characteristics or when there is a history of recent organophosphorous poisoning, non-depolarizing muscle relaxants like atracurium (0.1-0.3 mg/kg) should be used to reverse the muscle relaxation.

Use of concomitant drugs during the course of ECT

Drugs taken physical illness:

- Diuretics, anticonvulsants and hypoglycemic (including insulin) should be withheld in the morning and can be given after ECT session.
- Theophylline may be avoided and changed if possible.
- Other drugs (antihypertensives in particular) can be taken up to two hours before ECT with sips of water

Psychotropics

1) **Antidepressants:** There is no conclusive evidence to suggest that antidepressants increase the efficacy of ECT. However, they may act as prophylactic agents. Hence it is advisable that they may be continued during ECT. SSRIs may decrease seizure threshold. Higher dose of venlafaxine may cause hypertension.

2) **Antipsychotics:** The combination of antipsychotics and ECT mayhave a synergistic effect in schizophrenia. Antipsychotics may also be useful to prevent relapse after treatment. Despite being seizurogenic, clozapine can be safely combined with ECT.

3) **Mood stabilizers:** Earlier recommendations discouraged concomitant lithium use due to risk of delirium and prolonged seizures. However, recent evidence suggests that lithium can be safely combined with ECT. Post-ECT recovery may be delayed in people with high lithium levels. Hence lithium may be continued during ECT keeping a watch on blood levels, seizure threshold and post-ECT delirium. Blood levels of lithium may be monitored and it is safe to maintain lithium at the lower end of therapeutic range (Thirthalli, Harish, & Gangadhar, 2011). Anticonvulsant mood stabilizers may be continued, but the morning dosecan be skipped until after ECT. Patients receiving anticonvulsants during ECT have a higher seizure threshold, higher incidence of failure to obtain seizures, shorter duration of motor seizures and a higher number of ECT sessions. However the overall clinical outcome is not affected by ECT(Virupaksha et al, 2010).

4) **Benzodiazepines:** Benzodiazepines can be avoided if possible.Short acting agents are preferred when necessary. Morning dose should be skipped until after ECT.

Monitoring cognitive functions:

Cognitive impairment is commonly seen in people receiving ECT. The degree

of cognitive impairment is more for bilateral, thrice weekly, high dose ECT than that seen in unilateral, twice weekly, low dose ECT. A recent meta-analysis showed impairment of orientation, verbal episodic memory, autobiographical memory, visual episodic memory, attention, psychomotor speed and executive functions after ECT (Semkovska & McLoughlin, 2010). Most of these deficits disappear within 15 days after ECT. However, retrograde amnesia of autobiographical information can be persistent in some patients. This is especially apparent for events that occur closer to the treatment course. ECT may also improve some of the cognitive deficits that are secondary to depression such as processing speed, verbal working memory, and executive function.

Close monitoring of cognitive deficits is recommended in all patients receiving ECT. A baseline assessment should be conducted before commencing the treatment and then repeated weekly throughout the course of ECT.The assessment should be conducted at least 24 hours after ECT to prevent post-ictal effects (American Psychiatric Association, 2005). If gross deficits are present, a change of ECT technique or adjustment of medications should be considered. The evaluation should include at least orientation and memory. Global assessments like Mini Mental State Examination (MMSE) may not be sensitive enough to pick the memory deficits. A brief battery called Battery for ECT Related Cognitive Deficits (B4ECT-ReCoDe), has been designed specifically for this purpose and validated (Viswanath et al., 2013). It assesses verbal, visual, working and autobiographic memory, sustained attention, psychomotor speed and subjective memory impairment. The battery can be administered within 20-30 minutes.

Outpatient ECT

ECT can be safely provided on outpatient basis with a few precautions. Outpatient ECTis generally used for continuation and maintenance ECT. For the initial sessions, inpatient ECT is preferred unless it can be ensured that the patient will be compliant with the preparation for ECT, including medication dosage adjustments, avoiding oral intake, bowel and bladder preparation. A responsible caregiver should be assigned to help the patients in this regard.

Further, postictal confusion and acute cognitive disturbance may impair driving skills. Hence it is mandatory to assign a caregiver to accompany the patient. Inpatient ECT is preferred in patients who are at high risk of post ECT delirium (those with preexisting neurological illness and electrolyte disturbances) and those with comorbid medical illness which increase the risk of ECT related complications.

Use of unmodified ECT:

Despite the progress in modification of the ECT process, unmodified ECT is still widely used, especially in developing countries. Around 50% of patients in India receive unmodified ECT (Chanpattana et al, 2005). Various factors like lack or unaffordability of anesthesiological support, urgent need for ECT, and contraindication for anesthesia and/or muscle relaxants have been cited for this practice(Andrade et al., 2012). In this background, the Indian Association of Private Psychiatry, the Indian Association of Biological Psychiatry and the Indian Psychiatric Society issued a joint position statement regarding the use of unmodified ECT (Andrade et al., 2012). It was pointed out that unmodified ECT increases the risk of complications like musculoskeletal complications, pre-ECT anxiety, and post-ECT confusion. Thoracic spinal compression fractures are the commonest fractures and may be asymptomatic. However, the authors report risk for these complications may not as large as historically portrayed. The authors recommended that unmodified ECT should not be used as a routine form of treatment and should be used only under exceptional circumstances.

If an unmodified ECT is inevitable, risk factors for musculoskeletal injuries like muscularity and osteoporosis should be evaluated prior to treatment. A dental examination should be conducted to look for loose teeth and dental pathology. X-ray of the thoraco-lumbar spine (anterio-posterior and lateral) should be done before the first ECT (for comparison) and after completion of the course of treatment. X-rays may be repeated if the patient develops any symptoms suggestive of spinal injury. Some authors recommend the use of diazepam 10 mg slow i.v., 1-3 minutes before the commencement of treatment.

This may act as an anxiolytic and muscle relaxant. Alternatively 10 mg of oral diazepam can be given 1-2 hours before ECT. Bilateral electrode placement has been preferred due to its faster onset of action and lesser number of sessions needed. Restraints should be used to minimize convulsive movements during the seizure. Provision of 100% oxygen through a face mask is preferable, starting a minutebefore the passage of the electrical stimulus and extending to the onset of regular breathing after the end of the seizure. If hemodynamic changes are noticed, they should be managed accordingly(Andrade et al., 2012).

However it should be noted that the use of unmodified ECT is prohibited under The Mental Health Care Act Bill, 2013 that could soon become an Act.

LIST OF DRUGS IN ECT STATION (APA, 2005)

- Thiopentone
- Succinylcholine
- Atropine/Glycopyrrolate
- Anticonvulsants - Diazepam/Phenytoin
- I V Fluids
- Physostigmine
- Beta Blockers Labetalol, Esmolol
- Alpha Blockers- Prazosin, Clonidine
- Vasodilators – Nitroglycerine, hydralazine
- Antiarrhythmic drugs
- Antianaphylactics -epinepurine, steroids
- Ketamine
- Atracurium
- Bronchodilators, beta - agonists for nebulisation , aminophylline, steroids
- Antiemetics
- Analgesics

LIST OF EQUIPMENTS IN ECT STATION
(American Psychiatric Association, 2005)

- Bite blocks
- Intravenous sets
- Intravenous fluids
- Masks for oxygen delivery
- Oro- and nasopharyngeal airways, endotracheal tubes
- Suction catheters
- Syringes and syringe needles in assorted sizes
- Electrode gel or paste
- Monitoring electrode pads and leads
- Stimulus and monitoring cables for ECT device
- Recording paper for monitoring use
- Alcohol pads
- Material to prepare stimulus and monitoring electrode sites
- Gauze pads in assorted sizes
- Tape in assorted sizes
- Disposable gloves
- Containers for disposal of sharps and clinical waste

References

1. Abhishekh, H. A., Thirthalli, J., Manjegowda, A., Phutane, V. H., Muralidharan, K., & Gangadhar, B. N. (2013). Ictal EEG fractal dimension in ECT predicts outcome at 2 weeks in schizophrenia. *Psychiatry Research*, *209*(2), 155–159.

2. American Psychiatric Association, & Committee on Electroconvulsive Therapy. (2005). The Practice of Electroconvulsive Therapy Recommendations for Treatment, Training, and Privileging. American Psychiatric Association.

3. Andrade, C., & Kurinji, S. (2002). Continuation and maintenance ECT: a review of recent research. *The journal of ECT*, *18*(3), 149–158.

4. Andrade, C., Shah, N., Tharyan, P., Reddy, M. S., Thirunavukarasu, M., Kallivayalil, R. A., Mohandas, E. (2012). Position statement and guidelines on unmodified electroconvulsive therapy. *Indian Journal of Psychiatry*, *54*(2), 119–133.

5. Chanpattana, W., Kunigiri, G., Kramer, B. A., & Gangadhar, B. N. (2005). Survey of the practice of electroconvulsive therapy in teaching hospitals in India. *The journal of ECT*, *21*(2), 100–104.

6. Charlson, F., Siskind, D., Doi, S. A. R., McCallum, E., Broome, A., & Lie, D. C. (2012). ECT efficacy and treatment course: a systematic review and meta-analysis of twice vs thrice weekly schedules. *Journal of affective disorders*, *138*(1-2), 1–8.

7. Enns, M. W., Reiss, J. P., & Chan, P. (2010). Electroconvulsive Therapy. *Canadian journal of psychiatry. Revue canadienne de psychiatrie*, *55*(6), Insert 1–12.

8. Gangadhar, B N, Girish, K., Janakiramiah, N., Subbakrishna, D. K., Parameshwara, G., & Prasad, K. M. (1998). Formula method for stimulus setting in bilateral electroconvulsive therapy: relevance

of age. *The journal of ECT*, *14*(4), 259–265.

9. Gangadhar, B.N., Girish, K., &Janakiramaiah, N. (2000). Is a grandmal seizure necessary and sufficient for the efficacy of ECT? *Indian Journal of Psychiatry*, *42*(4), 443–444.

10. Gangadhar, B. N., & Thirthalli, J. (2010). Frequency of electroconvulsive therapy sessions in a course. *The journal of ECT*, *26*(3), 181–185.

11. Girish, K., Gangadhar, B. N., & Janakiramaiah, N. (2002). Merits of eeg monitoring during ect: a prospective study on 485 patients. *Indian Journal of Psychiatry*, *44*(1), 24–28.

12. Girish, K., Gangadhar, B. N., Janakiramaiah, N., &Lalla, R. K. (2003). Seizure threshold in ECT: effect of stimulus pulse frequency. *The journal of ECT*, *19*(3), 133–135.

13. Girish, K., Mayur, P. M., Saravanan, E. S., Janakiramaiah, N., Gangadhar, B. N., Subbakrishna, D. K., &Rao, G. S. (2000). Seizure threshold estimation by formula method: a prospective study in unilateral ECT. *The journal of ECT*, *16*(3), 258–262.

14. Girish, K., Prasad, K. M. R., Gangadhar, B. N., Janakiramiah, N., Subbakrishna, D. K., &Parameshwara, G. (1998). Clinical predictors of seizure threshold in bilateral ECT. *Indian Journal of Psychiatry*, *40*(4), 327–330.

15. Hiremani, R. M., Thirthalli, J., Tharayil, B. S., & Gangadhar, B. N. (2008). Double-blind randomized controlled study comparing short-term efficacy of bifrontal and bitemporal electroconvulsive therapy in acute mania. *Bipolar disorders*, *10*(6), 701–707.

16. Thirthalli J, Gangadhar, B., Janakiramiah, N., Girish, K., &Ramakrishnan, A. (2003). Post-seizure EEG fractal dimension and spectral power predict antidepressant response to unilateral ECT. *Indian Journal of Psychiatry*, *45*(1), 16–20.

17. Kotresh, S., Girish, K., Janakiramaiah, N., Rao, G. U., & Gangadhar, B. N. (2004). Effect of ECT stimulus parameters on seizure physiology and outcome. *The journal of ECT*, *20*(1), 10–12.

18. Leiknes, K. A., Jarosh-von Schweder, L., &Høie, B. (2012). Contemporary use and practice of electroconvulsive therapy worldwide. *Brain and Behavior*, *2*(3), 283–344.

19. Loo, C. K., Katalinic, N., Martin, D., & Schweitzer, I. (2012). A review of ultrabrief pulse width electroconvulsive therapy. *Therapeutic Advances in Chronic Disease*, *3*(2), 69–85.

20. Mayo, C., Kaye, A. D., Conrad, E., Baluch, A., & Frost, E. (2010). Update on anesthesia considerations for electroconvulsive therapy. *Middle East journal of anesthesiology*, *20*(4), 493–498.

21. Mayur, P. (2006). Ictal electroencephalographic characteristics during electroconvulsive therapy: a review of determination and clinical relevance. *The journal of ECT*, *22*(3), 213–217.

22. Mayur, P. M., Gangadhar, B. N., Janakiramaiah, N., & Subbakrishna, D. K. (1999). Motor seizure monitoring during electroconvulsive therapy. *The British journal of psychiatry: the journal of mental science*, *174*, 270–272.

23. Merkl, A., Heuser, I., &Bajbouj, M. (2009). Antidepressant electroconvulsive therapy: Mechanism of action, recent advances and limitations. *Experimental Neurology*, *219*(1), 20–26.

24. Mirzakhani, H., Welch, C. A., Eikermann, M., & Nozari, A. (2012). Neuromuscular blocking agents for electroconvulsive therapy: a systematic review: Neuromuscular blocking agents for electroconvulsive therapy. *ActaAnaesthesiologicaScandinavica*, *56*(1), 3–16.

25. Murali, N., Saravanan, E. S., Ramesh, V. J., Gangadhar, B. N., Jananakiramiah, N., Kumar, S. S., Subbakrishna, D. K. (1999). An

intrasubject comparison of two doses of succinylcholine in modified electroconvulsive therapy. *Anesthesia and analgesia*, *89*(5), 1301–1304.

26. Nitsche, M. A., Seeber, A., Frommann, K., Klein, C. C., Rochford, C., Nitsche, M. S., Tergau, F. (2005). Modulating parameters of excitability during and after transcranial direct current stimulation of the human motor cortex. *The Journal of Physiology*, *568*(1), 291–303.

27. Peterchev, A. V., Rosa, M. A., Deng, Z.-D., Prudic, J., &Lisanby, S. H. (2010). Electroconvulsive therapy stimulus parameters: rethinking dosage. *The journal of ECT*, *26*(3), 159–174.

28. Petrides, G., Tobias, K. G., Kellner, C. H., &Rudorfer, M. V. (2011). Continuation and Maintenance Electroconvulsive Therapy for Mood Disorders: Review of the Literature. *Neuropsychobiology*, *64*(3), 129–140.

29. Phutane, V. H., Thirthalli, J., Muralidharan, K., Naveen Kumar, C., Keshav Kumar, J., & Gangadhar, B. N. (2013). Double-blind randomized controlled study showing symptomatic and cognitive superiority of bifrontal over bitemporal electrode placement during electroconvulsive therapy for schizophrenia. *Brain stimulation*, *6*(2), 210–217.

30. Royal College of Psychiatrists, Special Committee on ECT, Scott, A. I. F., & Royal College of Psychiatrists. (2005). *The ECT handbook: the third report of the Royal College of Psychiatrists' Special Committee of ECT*. London; [Champaign, Ill.]: Royal College of Psychiatrists.

31. Saito, S. (2005). Anesthesia management for electroconvulsive therapy: hemodynamic and respiratory management. *Journal of anesthesia*, *19*(2), 142–149.

32. Semkovska, M., &McLoughlin, D. M. (2010). Objective Cognitive Performance Associated with Electroconvulsive Therapy for Depression: A Systematic Review and Meta-Analysis. *Biological Psychiatry*, *68*(6), 568–577.

33. Spellman, T., Peterchev, A. V., &Lisanby, S. H. (2009). Focal Electrically Administered Seizure Therapy: A Novel form of ECT Illustrates the Roles of Current Directionality, Polarity, and Electrode Configuration in Seizure Induction. *Neuropsychopharmacology*, *34*(8), 2002–2010.

34. Thirthalli, J., Harish, T., & Gangadhar, B. N. (2011). A prospective comparative study of interaction between lithium and modified electroconvulsive therapy. The World Journal of Biological Psychiatry,*12*(2), 149–155.

35. Van Waarde, J. A., Verwey, B., & van der Mast, R. C. (2009). Meta-analysis of initial seizure thresholds in electroconvulsive therapy. *European archives of psychiatry and clinical neuroscience*, *259*(8), 467–474.

36. Virupaksha, H. S., Shashidhara, B., Thirthalli, J., Kumar, C. N., & Gangadhar, B. N. (2010). Comparison of electroconvulsive therapy (ECT) with or without anti-epileptic drugs in bipolar disorder. *Journal of affective disorders*, *127*(1-3), 66–70.

37. Viswanath, B., Harihara, S. N., Nahar, A., Phutane, V. H., Taksal, A., Thirthalli, J., & Gangadhar, B. N. (2013). Battery for ECT Related Cognitive Deficits (B4ECT-ReCoDe): Development and validation. *Asian Journal of Psychiatry*, *6*(3), 243–248.

38. Phutane, V.H., Thirthalli. J., Muralidharan, K., Naveen Kumar, C., Keshav Kumar, J & Gangadhar, B.N. (2013). Double-blind randomized controlled study showing symptomatic and cognitive superiority of bifrontal over bitemporal electrode placement during electroconvulsive therapy for schizophrenia. *Brain Stimulation*. 6(2), 210–7.

PART – III :
ECT IN SPECIAL POPULATIONS

ECT is an effective treatment when used for its primary indications. It is effective in the treatment of depressive disorder, catatonia, mania and schizophrenia. It is especially used when there is a need for rapid response (e.g.suicidalilty, poor oral intake, aggression) or when there is poor response and intolerance to medications. It is no longer considered a treatment of last resort. However, the administration of ECT results in transient physiological changes, as discussed below. The use of general anesthetics and muscle relaxants are also associated with a small risk of complications. These considerations are especially important in medically compromised and elderly subjects. Fortunately, technical advances in recent years have rendered the old time contraindications into relative contraindications. If ECT is indicated and particularly when alternative treatments are riskier and/or less efficient, efforts must be made to overcome these contraindications. Here we discuss the issues related to provision of ECT in special populations like those with comorbid physical illness, children, pregnant women and the elderly.

A. ECT IN PATIENTS WITH COMORBID PHYSICAL ILLNESS

I. Cardiovascular disorders

Effect of ECT on the cardiovascular system

Prior to the administration of ECT there may be some tachycardia secondary to dehydration, anxiety and psychotropic medications. Thiopentone may lead to hypotension, which is not seen when ketamine or etomidate is used. Immediately after the administration of electric stimulus, independent of seizure response, a direct vagal effect on the heart occurs. This initial parasympathetic surge causes bradycardia for a few seconds. This may lead to transient asystole in unatropinised subjects. Pre-medication with atropine usually abolishes this effect. Bradycardia lasts for less than a minute and is usually not associated with much cardiac risk. This is slowly replaced by sinus rhythm of faster rate caused

by sympathetic outflow during seizure. The latter is maintained by the release of catecholamines from the adrenal gland. Peak heart rates up to 160/min may be reached during the seizure. The increased heart rate is accentuated by hyperventilation induced hypocarbia and atropine. The heart rate returns to normal within five minutes of termination of seizure.

Along with changes in heart rate, there is also a corresponding change in blood pressure (BP). There is a transient fall in BP due to vagal effect after the stimulus, which is soon compensated by rapid increments. The increase of BP is facilitated by increased cardiac return as well as peripheral vasoconstriction. Marked elevations up to 200mm of Hg in systolic BP may be recorded for 1-5 minutes after the seizure has ended. BP normalizes within 10-15 minutes after seizure termination. Unmodified ECT leads to higher elevation of diastolic BP. The cardiac output increases, but rarely over two folds. However, the rate-pressure-product (RPP = BP X Heart Rate) increases by 50-400 %. This reflects the increased cardiac work load and oxygen demand on the myocardium. RPP elevations are larger in men than in women. The sympathetic stimulation gradually decreases if the patient is ventilated properly and hypoxia is prevented.

Given this effect on the cardiac function during the seizure, benign changes in rhythm can be observed in ECG monitoring. Transient postictal arrhythmias can occur, some of which may require intervention. Vagal arrhythmias including those of atrial, junctional or nodal origin as well as sinus bradycardia, sinus arrest, atrial premature contractions, paroxysmal atrial tachycardia, atrial flutter, atrial fibrillation, first, second, or third-degree atrioventricular block, and premature ventricular contractions can occur during the sinus bradycardia phase. Tachyarrhythmias which occur during the sympatho-adrenal rush are more dangerous. These include ventiruclar bigeminy, trigeminy, ventricular tachycardia and ventricular fibrillation. ECT increases QTc interval and QT dispersion, which increases the risk of ventricular arrhythmias. Benign atrial arrhythmias and unifocal ventricular premature contractions (VPCs) do not usually require medical intervention, in contrast to ventricular arrhythmias. Other transient ECG changes, both ischemic and non-ischemic, are recorded

even in normal subjects but are more often in cardiac patients. They include peaked T' waves, inverted T' waves and S-T segment depression. Thiopentone produces more ECG abnormalities than methohexital. ECG abnormalities are rarely present beyond the acute postictal phase.

Non-convulsive stimulus produces only the vagal effect leading to bradycardia and can potentially prolong the asystole. The chance of non-convulsive stimulation is more in the initial titration phase. Despite the general belief that non-convulsive stimuli are not associated with any CNS morbidity; the cardiac risk cannot be ignored. The sympatho-adrenal outflow associated with the convulsive response counteracts the vagal cardiac inhibition. The non-convulsive stimulus hence, fails to overcome the vagal inhibition. Beta-blockers increase the risk of asystole in such patients. Atropine pre-medication is used by most practitioners to prevent bradycardia and asystole. However, it increases ictal tachycardia and contributes to larger RPP. Glycoprrolate is sometimes used in place of atropine and which has a lesser chance of inducing tachycardia. In patients with normodynamic cardiac function, atropine pre-medication as a routine may not be indicated.

Effects of psychotropic drugs

Tricyclic antidepressantsare known to produce sinus tachycardia, intra-ventricular conduction abnormalities, and arrhythmias. However they do not increase CVS risk during ECT. No rationale is offered for their discontinuation during ECT on grounds of CVS risks. Likewise, lithium also poses no extra risk to CVS morbidity in the absence of electrolyte disturbances. Benzodiazepines, by virtue of increasing seizure threshold, contribute to sub-convulsions.

Pre-ECT medical evaluation of high-risk CVS patient

Majority of ECT complications result from cardiovascular risk. The risk is more apparent in people with pre-existing cardiovascular diseases. The decision to provide ECT in patient with pre-existing cardiac illness should be taken on a case-by-case basis after a risk-benefit analysis. The dictum is to titrate pre-ECT evaluations to each patient with the knowledge of pre-existing CVS disease. A

detailed physical examination should include careful evaluation of cardiac function. Stabilizing blood pressure in hypertensive needs no emphasis. Stress test is preferred over coronary angiogram in high risk patients, as the former evaluates the functional integrity of the heart. Echocardiography can be used to assess myocardial function, kinetic segments and size of the individual chambers. Consultation by a cardiologist should be obtained in all patients who have had known ischemic attacks, valvular disease (operated or otherwise) and in patients with congestive cardiac failure. Routine metabolic investigations and ECG should be supplemented with investigations such as serum digitalis levels, prothrombin time, and arterial blood gas levels.

Medical evaluation is best repeated at intervals during the course of ECTs rather than merely declaring the patient's fitness at the start of the ECT course. Detecting early evidence of compromised cardiac function effectively prevents morbidity related to ECT. ECT is considered as a "low risk procedure" for cardiac conditions (Christopher, 2003). With appropriate pretreatment and monitoring, the procedure can be safely conducted in most patients.

Individual cardiac disorders

Systemic hypertension:

People with hypertension may have large elevations of ictal and postictal blood pressure. The patient should take their routine antihypertensives at least two hours before ECT. Diuretics should be avoided as they may lead to incontinence or bladder rupture. An occasional patient may need intravenous antihypertensives. Current guidelines advocate the use of either labetalol (a mixed alpha- and beta-blocker) in the dose of 0.1 to 0.2 mg/kg or esmolol (a short-acting beta blocker) in the dose of 1 to 1.3 mg/kg body weight. Either of the above drugs given during the procedure blunts the sympathetic response and maintains heart rate and blood pressure under control. Beta blockers increase the risk of asystole and hence anticholinergics (preferably glycopyrrolate) should be used concomitantly. Routine use of beta-blockers for prophylaxis may be avoided as it may increase the risk of asystole. Beta-blockers may blunt the

necessary increased hemodynamic response to meet the excessive oxygen demand during seizures. The use of calcium channel blockers like nicardipine may cause tachycardia secondary to vasodilation. Rarely nitroglycerine infusion (0.01%) may be necessary to maintain blood pressure around 140-150 mm Hg in patients with myocardial ischemia. Successful use of ECT has been documented even in patients with pheochromocytoma.

Arrhythmias:

Arrhythmias detected before ECT should be corrected. Close liaison with a cardiologist is essential. Digitalis and calcium channel blockers may be used in atrial fibrillation as prescribed. Blood levels of the drug should be within therapeutic range to prevent the risk of sinoatrial arrest. ECT may convert fibrillation into sinus rhythm and may dislodge thrombi. Hence adequate anticoagulation should be ensured. While arrhythmias occurring during ECT are often benign and self-limiting (benign atrial arrhythmia or unifocal premature ventricular contractions [PVCs], drugs should be available to control the more complicated ones. Patients with high-grade atrioventricular block, symptomatic ventricular arrhythmias, and supraventricular arrhythmias with uncontrolled ventricular rate are especially considered to be of high risk.

Atropine is the drug of choice for vagal arryhthmias including sinus bradycardia and arrest, atrial and junctional arrhythmias, and ventricular premature contractions during sinus bradycardia. Lidocaine and beta-blockers are recommended to control tachyarrhytmias. These usually occur late in the seizures or in the immediate post-ictal period, the most common being ventricular premature contractions (VPC). Occasional and unifocal VPCs are benign, however multifocal and frequent VPCs could lead to ventricular tachycardia or ventricular fibrillation.

A rapid bolus of lidocaine at a dose of 1 to 2 mg/kg body weight controls VPCs. However, the drug also potentially elevates the seizure threshold and shortens the seizure duration. Esmolol and labetalol are effective anti-arrhythmic agents, which do not affect the seizure duration or threshold. For

control of supra-ventricular and nodal arrhythmias, verapamil (5 mg) intravenously is helpful. However, caution should be exercised in digitalized patients and in those on beta-blockers; hypodynamic state may be a complication.

ECT is safe even in those with implanted cardiac pacemakers. Electrical stimulus applied during ECT does not usually interfere with the pacemaker unless poor grounding exists in the electro-medical equipment used. In fact, ECT with the pacemaker results in fewer cases of arrhythmias as the heart rate is controlled by pacemaker. However, hypertensive episode may still occur. Pre-ECT X-ray should confirm intact insulation around pacemaker wires. The patient should be sufficiently paralyzed with the muscle relaxants. The demand pacemakers, which are more commonly used, can be inhibited by the discharges from muscle potentials during succinylcholine depolarization and during the clonic phase of the seizure if the paralysis is inadequate. Some practitioners routinely convert the pacemaker from the demand (rate responsive) mode to fixed (rate non-responsive) mode to prevent unnecessary triggering during ECT. However a consultation with the cardiologist should be helpful in this regard. A ring magnet should be available to convert the demand mode to fixed rate mode if severebradycardia should occur. Patients with Automatic Implantable Cardiac Defibrillators (AICD) can undergo ECT. They may be turned off before ECT and reactivated later. However their pacemaker function should be left operative.

Congestive Cardiac Failure (CCF):

CCF should be adequately controlled before ECT. In patients with CCF, the ictal hypertensive response may at times precipitate failure and pulmonary edema. Therefore the ictal hypertensive response should be well controlled. Beta-blockers are helpful in this regard. Etomidate may be preferred as an anesthetisic agent as it decreases the hemodynamic response. If barbiturates are used for anesthesia, the dose should be carefully titrated as they are potential myocardial depressants.

Recent myocardial infarction (<3 months) is only a relative contraindication for ECT. CVS complications of myocardial infractions should be well controlled before ECT. Well compensated old myocardial infarction (> 6 months) is no contraindication for ECT. Vigorous pre-anesthetic oxygenation may be helpful in meeting the increased oxygen demand to the heart. Nevertheless, all precautions including stress test should be taken before ECT. A complete cardiac assessment is necessary as discussed earlier.

Vascular malformation in systemicas well as cerebral circulatory systems, poses risks during ECT. Aneurysms pose a risk as they may rupture and bleed during thehypertensive responses to ECT. However successful use of ECT in patients with aneurysm has been documented. The hypertensive response should be blunted, preferably with a beta blocker.

II. CNS conditions

Immediately after the stimulus, there is a decrease in cerebral blood flow for a few seconds which is followed by a drastic increase for few minutes. ECT induced seizures increases the regional cerebral blood flow, oxygen demand and glucose utilization in the brain. The degree of cerebral hyperemia is positively correlated with the extent of systemic hypertension. The increase in cerebral blood flow may be necessary to meet the increased oxygen demand during the seizure. There are also elevations, in cerebral venous PO2 and PCO2. Increased cerebrovascular permeability to tracer molecules has also been demonstrated. In this background, it is expected that seizures during ECT contribute to elevations in intracranial pressure (lCP). These physiological changes should be considered while administering ECT to patients with CNS disorders.

Intracranial space occupying lesions (lCSOL): ECT has been used successfully in patients with small and chronic ICSOLs. The decision to undergo ECT in patients with ICSOL should be made only in consultation with neurosurgeons. Concerns arise when there is increased ICP, which may be accentuated during ECT. ECT may have to be withheld when there is evidence for raised ICP. Dexamethasone may be used in patients with ICSOL to prevent further elevations of ICP during ECT. Cerebral hemodynamic changes may be decreased by using beta-blockers.

Post-craniotomy: Electrodes should be placed away from the craniotomy site preferably equidistant from the skull defect to avoid abnormally high charge densities and status epilepticus. Safe use of ECT in patients who have undergone a neurosurgical procedure with craniotomy has been reported.

Cerebro-Vascular Accidents (CVA): CVAs can lead to severe affective states warranting ECT. Patients with CVA may have difficulty tolerating antidepressants. No additional risk is anticipated if ECT is used 3 months after a stroke. ECT has been used even in one month post-stroke patients uneventfully. Careful monitoring of blood pressure is required for patients with hemorrhagic strokes. When patients are considered to be at risk for a hemorrhage, the blood pressure should be controlled adequately. However, hypotensive responses should be prevented in patients with ischemic stroke. Antihypertensives vary in their ability to alter cerebral blood flow (Saito, 2005). For example, nitroglycerine induces significant decrease in cerebral blood flow when compared with alprenolol or nicardipine.

Epilepsy: Epileptic patients may develop severe psychiatric disorders necessitating use of ECT. Patients on anticonvulsants may need a higher stimulus to produce seizures. Anticonvulsants can be continued in patients on ECT but the morning dose has to be postponed until after ECT. Blood levels of anticonvulsants may be monitored and maintained at the lower end of the therapeutic range. ECT increases seizure threshold and hence can be safely used in patients with epilepsy.

Dementia: Dementia is not a contraindication for ECT. Major depression occurring in demented patients can be successfully and safely treated with ECT. However their cognitive functions may deteriorate transiently.

Others: In patients with ventriculoperitoneal shunts, the patency of the shunt should be confirmed before referring them for ECT. Patients with myasthenia gravis may show increased sensitivity to muscle relaxants and hence their dosage should be reduced. Patients with traumatic brain injury are at increased risk for cognitive deficits.

III. Other comorbid physical illness:

Patients with depressive disorder can have primary or subclinical hypothyroidism. In the former case, the CVS effects of the endocrinopathy should be properly evaluated and if abnormal, it should be brought under control. In primary hyperthyroidism, use of beta-blockers helps to overcome the risks from thyroid storm during the seizure.

Patients with COPD should receive systemic anticholinergics to decrease secretions. Theophylline should be avoided as it prolongs seizure duration. Adequate preoxygenation should be done. Oxygen monitoring may be necessary. Beta-blockers have to be used carefully as they may lead to bronchoconstriction.

Electrolyte imbalances have to be corrected before ECT. Succinylcholine may aggravate hyperkalemia and increase the risk of cardiac complications. ECT can be safely given in patients with diabetes mellitus. The morning dose of oral hypoglycemic or insulin should be postponed until after ECT. Patients with gastro-esophageal reflux disease are at increased risk of aspiration. Pretreatment with antihistamines and metoclopramide may be necessary. Patients with joint and bone disease may need higher dose of muscle relaxants. Care is needed regarding dental conditions such as loose teeth, dentures etc. In patients undergoing anticoagulant treatment, International Normalized Ratio (INR) is suggested between 1.5 and 2.5.

B. ECT IN PREGNANCY

- ECT has been found to be safe in all trimesters of pregnancy. However the treatment should be given only after consultation with an obstetrician.
- The anesthetic agents and muscle relaxants used in ECT are too short acting to cause teratogenic effects. However, barbiturates may rarely slow down fetal heart rate. A slightly lower dose may be used.
- Adequate preoxygenation is necessary to maximize oxygen delivery

to the fetus. However hyperventilation may decrease fetal blood flow and reduce dissociation of oxygen from hemoglobin.

- Because of increased risk of gastric reflux and aspiration in pregnant women, 30 ml of 0.3 M sodium citrate, 15 to 20 minutes before ECT can be used. Ranitidine and metoclopramide are the other choices. Anticholinergics may worsen the reflux by relaxing the esophageal sphincter.
- The seizure does not cause uterine contractions and is not detrimental to the fetus. However, physical injury and hypoxia for the mother affect the child adversely. Oxytocin release caused by the seizure may increase uterine contractions.
- After 20 weeks of pregnancy, the right hip should be elevated to prevent uterus from compressing the aorta, vena cava and diaphragm.
- After the first trimester, fetal heart monitoring should be done before and after each ECT session.
- ECT should be done in pregnant women only in settings which have facilities to manage obstetric and neonatal emergencies. Tocolytic therapy in the form of ritodrine or magnesium can be used in patients developing persistent uterine contractions during or shortly after ECT.

C. ECT IN CHILDREN AND ADOLESCENTS:

There has been very less systematic data on the use of ECT in children and adolescents. The available evidence supports the use of ECT in acute control of mood and psychotic disorders. ECT is especially indicated in these conditions if there is a need for rapid improvement (because of suicidality, poor oral intake, uncontrollable aggression with risk of harm to self or others) or with poor response to other treatments. There are a few reports of improvement in self-injurious behavior and catatonia in children with autism too. Despite the efficacy, ECT is often underutilized in this population.

The administration of ECT in this population is similar to that of adults, but a few points require specific mention.

- Informed consent for ECT should be obtained from the legal guardian, with ascent from the child.
- Guidelines recommend an independent second opinion from another psychiatrist before proceeding with ECT in children.
- In-patient ECT is generally recommended.
- The seizure threshold is low in children, hence the stimulus dose should be titrated gradually.
- There is a risk of prolonged seizures; hence EEG monitoring would be helpful.
- Cognitive deficits are similar to that seen in adults. It is recommended that cognitive assessment should include age appropriate memory testing with focus on short-term memory and new knowledge acquisition. Cognitive assessment is recommended during the treatment and after 3-6 months to monitor for long term deficits.

D. ECT IN THE ELDERLY

Old age per se is not a contraindication for ECT. In fact, it has been seen that elderly people are more likely to receive ECT in many countries. Factors like poor tolerance and response to psychotropics as well as higher chance of complications like psychosis, suicidality and inanition may explain this trend. ECT in elderly is provided for the same indications as that in other populations. The administration is also largely similar. Nevertheless, caution is needed on a few issues:

- Elderly people are at higher risk of having a physical comorbidity and to be on multiple medications. Administration of ECT in such conditions has been discussed above.

- Seizure threshold usually increases with age. Sometimes the recommended intensity in elderly people may be higher than the maximum output of the ECT device. Options in such cases include (1) changes in medications like anticonvulsants, benzodiazepines, etc., (2) change of anesthetic agents used (e.g., Etomidate in place of barbiturates), (3) use of unilateral ECT which has a lower threshold.
- Elderly people are at risk of having cognitive deficits. Hence, modifications in techniques like use of unilateral or bifrontal electrode placement, twice weekly ECT etc., maybe tried to minimize cognitive deficits.

References

1. Abrams, R. (2002). Electroconvulsive therapy (4th ed.). Oxford; New York: Oxford University Press.
2. American Psychiatric Association, & Committee on Electroconvulsive Therapy. (2005). The Practice of Electroconvulsive Therapy Recommendations for Treatment, Training, and Privileging. American Psychiatric Association.
3. Anderson, E. L., &Reti, I. M. (2009). ECT in pregnancy: a review of the literature from 1941 to 2007. *Psychosomatic medicine*, *71*(2), 235–242.
4. Christopher, E. J. (2003). Electroconvulsive therapy in the medically ill. *Current psychiatry reports*, *5*(3), 225–230.
5. Ghaziuddin, N., Kutcher, S. P., Knapp, P., Bernet, W., Arnold, V., Beitchman, J., … AACAP. (2004). Practice parameter for use of electroconvulsive therapy with adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*(12), 1521–1539.
6. Girish, K., Gangadhar, B. N., &Janakiramaiah, N. (2002). ECT in neurological conditions. Indian Journal of Psychiatry, 44(3), 228–239.
7. Pandya, M., Pozuelo, L., & Malone, D. (2007). Electroconvulsive therapy: what the internist needs to know. *Cleveland Clinic journal of medicine*, *74*(9), 679–685.
8. Royal College of Psychiatrists, Special Committee on ECT, Scott, A. I. F., & Royal College of Psychiatrists. (2005). *The ECT handbook: the third report of the Royal College of Psychiatrists' Special Committee of ECT*. London; [Champaign, Ill.]: Royal College of Psychiatrists/ ; Distributed in North America by Balogh International.

9. Saatcioglu, O., &Tomruk, N. B. (2011). The use of electroconvulsive therapy in pregnancy: a review. *The Israel journal of psychiatry and related sciences*, *48*(1), 6–11.

10. Saito, S. (2005). Anesthesia management for electroconvulsive therapy: hemodynamic and respiratory management. *Journal of anesthesia*, *19*(2), 142–149.

11. Shoirah, H., &Hamoda, H. M. (2011). Electroconvulsive therapy in children and adolescents. *Expert review of neurotherapeutics*, *11*(1), 127–137.

12. Tezuka, N., Egawa, H., Fukagawa, D., Yamaguchi, S., Hamaguchi, S., Kitajima, T., & Minami, J. (2010). Assessment of QT Interval and QT Dispersion During Electroconvulsive Therapy Using Computerized Measurements: *The Journal of ECT*, *26*(1), 41–46.

13. Van Herck, E., Sienaert, P., &Hagon, A. (2009). [Electroconvulsive therapy for patients with intracranial aneurysms: a case study and literature review]. *Tijdschriftvoorpsychiatrie*, *51*(1), 43–51.

PRACTICE AT NIMHANS

At NIMHANS, modified ECTs are used routinely. Psychiatry senior resident/ consultant, anesthesiologist, trainee residents, psychiatric nurses and other para-medical staff form the ECT team. The treatment centre comprises a waiting hall, preparation room, ECT Suite and recovery room.

The ECT suite is equipped with constant current ECT machines, Boyle's machines, EEG monitor, ECG monitor and pulse oximeter. Thiopentone (3mg/ kg), atropine (0.65mg) and succinylcholine (0.75mg /kg) are used for anesthesia. Bilateral ECT is used unless otherwise specified by the referring psychiatric team. Both bitemporal and bifrontal ECT are used as specified by the referring psychiatric team. Threshold is estimated using titration method during the first ECT session. For subsequent sessions, 1.5 times the threshold in bilateral ECT is the recommended practice; for unilateral ECT 4-6 times threshold stimulus is used. The frequency of ECT sessions is thrice weekly unless otherwise specified. The clinical unit can however indicate alternative stimulus standards or session frequency to be followed in selected cases.

The ECT machine has been designed in collaboration with the NationalInstitution for Quality and Reliability (NIQR). It offers constant current pulses of 800 mA strength and 1.5 ms width. The pulses are of alternating polarity and recur at 125 pulses per second. Ten levels of stimulus can be chosen by varying the total stimulus train duration from 0.2 to 3.6 seconds. The stimuli start with a dose of 30mC followed by 60mC and subsequently increments in steps of 60 mC. The maximum stimulus available is 540mC.

The same machine can also be computerized. In this condition the stimuli can be chosen in either 'Auto' or 'Manual' mode. The stimulus settings in the former are similar to those described earlier, except that the computer sets the stimuli in doses of 19 steps between 30mC and 540mC, the first two increments are 15mC and the rest 30mC. In the latter mode, all parameters of the stimuli can be chosen as desired by the treating doctor. The general preference, however, is to use the 'Auto' mode. The machine is connected to an independent EEG machine

using an 'EEG-disconnect' feature. EEG is monitored on the computer display after being digitized. The EEG data can also be stored if required for analysis in the computer.

Informed consent is obtained from the patient or patient's guardian using the consent form provided. The names of the patients are entered in the ECT register at least 24 hours before the next ECT session. The patients needing special medical attention are discussed with the anesthetist by the prescribing unit well in advance.

The ECT proforma is completed by the resident in charge and verified by the senior resident. Necessary preparations for ECT are also advised for the nurse to take action. Cognitive functions are assessed before the initiation of ECT, where feasible and at regular intervals thereafter using the B4ECT-RECODE.

Psychotropic drugs are continued at the discretion of the parent unit. Changes in the drug status during the ECT course are avoided unless absolutely necessary. Changes in the medical status and introduction of required drugs are preferably discussed with or brought to the notice of the anesthetist, e.g. sadding a bronchodilator (etophylline), antihypertensive (atenolol), etc.

Typical tonic phase followed by tonic phase of generalized seizure should beobserved. The seizure durations arerecorded using the cuff method. Other vital parameters before and after ECT are recorded. The treatment team would leave the ECT suite only after the last patient has left the recovery ward. The prescribing resident would review the patient and ECT notes in the afternoon. If ECT sessions have to be terminated, treating resident should mention the same in the ECT form.

APPENDIX-I

ECT CONSENT FORM

Information:

After examination and considering all aspects of your condition we opine that you would require ECT as a form of treatment. The treatment is given under anesthesia so that you do not experience any discomfort. You will be required to be on empty stomach in the morning of ECT. The anesthesia is given as an intravenous injection, after which we will administer a small and safe dose of electricity using a well controlled electrical circuitry. This stimulates the brain cells and produces a brief seizure which will last less than a minute. You will recover consciousness within 30 minutes. Till such time, you will be under the supervision of a medical team. The treatment is given on alternate days or possibly twice a week, as decided by the treating doctor till sufficient improvement occurs – usually fewer than 12 sessions.

The treatment causes some temporary side effects such as headache and or a little confusion, for sometime on the day of ECT. You may notice some lapses in memory for events occurring on the days of ECT. Rest of the memory including all you have learnt earlier will not be affected. ECT with anesthesia has a few serious side effects which are very rare. (If the patient is under 16 years no harm to his / her growth or development is known to occur as a result of ECT). Over all, the advantages of ECT are expected to outweigh the risks.

ECT will be administered to you only if you consent for the treatment. You have a right to refuse now as well as withdraw the same during the course of the treatment. In either case, the best available alternative treatments will be offered without prejudice. You are also free to reconsider the matter and request ECT if need be. Please feel free to contact us.

DECLARATION OF CONSENT FOR ECT

I have been advised that my present condition requires Electro Convulsive Therapy (ECT), and sufficiently informed about the procedure with its risks and benefits. I am aware that I have the right to refuse this treatment now or at any time during the course of treatment without compromising my right to obtain all other services,

I _____

(Name of the patient)

hereby solemnly declare my consent for the administration of ECT as required to myself.

I own full responsibility for the consent and exonerate NIMHANS from any consequences.

_____

(Signature of Relative) (Doctor's signature)

_____

(Name of the Doctor) (Doctor's signature)

In the event the patient cannot comprehend the information and consent

I, _____ having understood the nature of the disease and treatment (ECT) offered to _____

who is my _____ give my consent for the treatment.

I own fully responsibility for consent and exonerate NIMHANS from any consequences.

Date:

_____

(Signature of Relative)

APPENDIX - II

PRE-ECT EVALUTION PROFOMA

Name_____P /No. _____Age _____(Yrs)

Father/Spouse's name:_____**Date**_____

Gender – Male / Female **Unit**_____ **Ward**_____

ICD 10 Diagnosis:_____

Past History of ECT – Yes / No If yes, when_____

Response to ECT-Good / Poor/Don't Know

ECT is prescribed because: (Circle relevant; can be more than one reason)

1. Adequate dosage and duration of drug therapy failed
2. Cannot afford to wait for drug effects
3. Drug compliance / administration is a problem
4. Drug intolerance – actual / anticipated
5. ECT was effective earlier
6. ECT is chosen as first line of treatment
7. ECT is needed to augment drug therapy,
   If other reasons exist specify_____
8. Reasons for discontinuation of ECT

Unit's preference about ECT: (Circle relevant)

1. Electrode Placement:

a. Unilateral
b. Bitemporal
c. Bifrontal

2. Stimulus Dose:

a. Threshold
b. 1.5 suprathreshold
c. 2.5 suprathreshold
d. Others: specify

Notes to the ECT team:

PREANESTHETIC EVALUATION SHEET

Medical History: (Tick appropriate box) Diabetes ☐ Hypertension ☐ IHD ☐ Asthma/COPD ☐ Epilepsy ☐ Others: ☐ Specify_____ NIL ☐

Medications (all psychiatric and medical drugs):

No.	Present Drugs	Dose	Schedule	Remarks / skipped night before ECT
1				
2				
3				
4				
5				
6				
7 8				

Body Weight ____ (Kg) **Handedness**: Right / Left

AIRWAY: Mouth opening**:** Adequate ☐ Restricted ☐ **Loose tooth: Yes**☐ **No**☐

RS:

NVBS ☐ Crepitations ☐ Ronchi ☐ Others: Specify

CVS:

HR: BP: S1….. S2….. Murmur ☐ Others:

CNS: Record relevant findings

FUNDI (tick if normal): R ☐ L ☐ Specify if abnormal:

INVESTIGATIONS:

Hb%: RBS: Na: K: Urea: Creatinine:

ECG: Other relevant investigations:

Suspected pseudocholinesterase deficiency? Yes / No.

Anesthetist's Comments:

Anesthetist's Signature:

APPENDIX-III

ECT RECORD

Patient's Name: _____P/ No_____

Electrode Placement: Bitemporal (BT); Unilateral – LUL / RUL; Bifrontal (BF)

Details	1	2	3	Date:#	4	5	6	Date:#	7	8	9	Date:#	10	11	12	Date:#
Date																
Thiopentone																
Suxamethonium																
Atropine				Subjective Total =				Subjective Total =				Subjective Total =				Subjective Total =
Electrode Placement																
Charge (mC)																
Motor Sz duration (seconds)																
EEG Sz duration (seconds)																
JR / SR Sign.				Objective Total =				Objective Total =				Objective Total =				Objective Total =
Remarks*																
Anesthetist's Sign																

* **Indicate any observation of prolonged seizures/apnea, bradycardia/tachycardia, hypotension/hypertension, post-ECT confusion/ delirium or any other complications and how it was handled. Please write in the file if you need more space to document.**

\# **Date of cognitive assesment and scores.**

WARD-NURSES' CHECK LIST FOR THE PREPARATION OF PATIENTS FOR ECT

PATIENT'S NAME: **AGE:** **SEX:** **WARD:** **WEIGHT:**

1. Explanation of procedure to patient and relatives ☐ 2. Informed consent & proforma ,

3. Check for special precautions: heart disease, epilepsy, brain tumor, pregnancy☐ **Specify if any medical problem:**

ECT	1	2	3	4	5	6	7	8	9	10	11	12
Date												
Tick when you have done the following checks												
Overnight fasting												
Oil free scalp												
Removal of dentures and contact lenses, valuable & metal objects from patient												
Pt has empties bowel and bladder before leaving for ECT												
Changing of patient's dress												
HR & BP												
Premedication, if any, given												
Signature of the nurse												

POST-ECT RECOVERY CHECKLIST FOR NURSES

ECT →	1	2	3 4 5	6	7	8 9 10	11 12
Pulse BP							
Respiratory Rate							
Anesthetist's clearance for shifting							
Signature of the nurse							