

## ANESTHESIA FOR ELECTROCONVULSIVE THERAPY: A NOBLE APPROACH

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### INTRODUCTION

Electroconvulsive therapy was first used to provoke generalized epileptic seizures as treatment for schizophrenia by Italian neurologist, Lucio Bini and Ugo Cerletti on April 18, 1938 & was performed without anesthesia for almost 30 years<sup>[1]</sup>. Later came the period of "modified ECT"- including the use of general anesthesia & muscle relaxants, which led to its current acceptance as a result of reduced physical & physiologic trauma. The World Health Organization has now called for a worldwide ban on unmodified ECT.

**How does ECT work?** ECT consists of programmed electrical stimulation of the central nervous system to initiate seizure activity. According to one theory, seizure activity itself causes an alteration of the chemical messengers in the brain known as neurotransmitters and another theory proposes that ECT treatment adjusts the stress hormone regulations in the brain, which may affect energy, sleep, appetite and mood. The electrical stimulus results in generalized tonic activity for approximately 10 seconds followed by generalized clonic activity for a variable period lasting up to 120 seconds. The seizure should ideally last for more than 15 seconds and less than 120 seconds. Modified ECT is typically administered as a series of treatments two to three times a week for 6 to 12 treatments, in its acute phase. Maintenance therapy can be performed at progressively increasing intervals from once a week to once a month to prevent relapses<sup>[2]</sup>.

**Indications:** The National Institute of Clinical Excellence (NICE) UK Guidelines 2009<sup>[3]</sup> recommend that the ECT be considered for the patients who are suffering from -

1. Acute, life threatening depression (high suicide risk or very poor fluid intake)

### ABSTRACT

Electroconvulsive therapy (ECT) has always proved to be an effective mode of therapy in the field of Psychiatry. Modified ECT is applied in the form of electrical stimulus to the central nervous system; it is associated with acute physiologic response leading to autonomic nervous system stimulation with an initial parasympathetic stimulation followed by a more prominent sympathetic response as well as post-ictal effects like confusion and delirium. The factors governing the efficacy of Modified ECT are the strength of electrical current applied and the duration of the seizure activity. Modified ECT requires the use of general anesthesia and many of the anesthetic drugs also have an effect on the duration of seizure and could adversely affect the efficacy of the Modified ECT treatments. Therefore, there has to be a delicate balance between achieving an adequate anesthetic state and optimal duration of seizure activity.

2. Drug resistant depression (failure to respond to two medications given at adequate dose for adequate period of time) or where treatment is limited by unacceptable side effects. It may also be appropriate to consider initiation of ECT early if a patient has shown good response previously or it is known that they only respond to ECT
3. Acute catatonia (where first line treatment with intramuscular benzodiazepines has failed to produce improvement)
4. Mania, where treatment has failed to alleviate the condition or is limited by side effects.

There are no absolute medical contra-indications to ECT in current dates whereas relative contra-indications include space occupying lesions of the brain, high intracranial pressure, intracerebral bleeding, recent cerebral infarction, recent myocardial infarction (<3 months), retinal detachment, pheochromocytoma, untreated cerebral aneurysm, unstable major fractures or cervical, uncontrolled cardiac failure or severe valvular disease, deep venous thrombosis, pulmonary conditions like COPD, asthma or pneumonia, adolescents and children and anesthetic risk rated as ASA level 4 or 5 or a significant medical illness risk outweighs potential benefit.

**Anesthetic management:** The essential elements of anesthesia for Modified ECT include rapid loss of consciousness, effective attenuation of the hyperdynamic response to the electrical stimulus, avoidance of gross movements, minimal interference with seizure activity and prompt recovery of spontaneous ventilation and consciousness.

**Pre ECT evaluation:** Pre ECT evaluation is a collaborative approach between the psychiatrist, anesthetist and medical consultants and should include [4, 5,6,7,8,9,10]

- A thorough psychiatric history and examination including history of response to other treatments
- A medical history and examination with special attention to cardiovascular, pulmonary, neurological and musculo skeletal systems
- A history of dental problems and examination for loose or missing teeth.
- A history of personal and family experiences with anesthesia.
- A cognitive assessment (at minimum, evaluation of orientation and memory)
- A minimum battery of laboratory test includes complete blood count, serum chemistry, renal function, and an electrocardiogram and urine analysis.
- Additional test identified during preliminary evaluation are as follows:
- Chest radiograph (especially with cardiovascular and pulmonary disease or history of smoking)
- Electroencephalogram guided by history and examination.
- Neurological /neuropsychological tests guided by history and examination
- Spinal radiograph (especially with known or suspected spinal disease)
- Consultation with medical specialties such as cardiology, neurology, neurosurgery or endocrinology as requested by special medical conditions.

**Informed consent:** Patients have the right to be fully informed about the proposed Modified ECT treatments, unless they lack capacity which is determined by the attending psychiatrist. Patients have the right to consent to Modified ECT treatment or to refuse treatment. If a patient, determined to have capacity refuses Modified ECT treatment, Modified ECT treatment would not be sought through court authorization, court authorization would be sought only in cases where the patient is determined to lack capacity [7]. Prior to Modified ECT treatment, informed consent for Modified ECT must be obtained from the patients(18 years & older)or if the patient is under 18 years, from the parents or the legal guardian except when it has been determined that the patient lacks capacity to consent [7]

**Anesthetic implications of psychotropic drugs:** The management of the patients on psychoactive medications in the perioperative period is based on the individual clinician's experience. Challenges for the anesthetists arise from the nature of the psychiatric condition itself, interaction of psychoactive and anesthetic drugs.

**Tri cyclic anti depressants:** May cause sedation and reduce the seizure threshold so anticholinergics should be avoided in such cases. One of the most significant interaction for the anesthetists is to be aware of the potentiating effect of indirectly acting sympathomimetics (ephedrine and metaraminol) by TCA's .These should be avoided if possible and directly acting sympathomimetics used cautiously to prevent hypertensive crisis

**Selective serotonin reuptake inhibitors:** Considering more serious withdrawal symptoms and risks associated with remaining on an SSRI being low, it is better to continue these drugs throughout the peri- operative period.

**Mono-amine oxidase inhibitors:** The metabolism of indirectly acting sympathomimetics is inhibited by MAOIS resulting in the potentiation of their action. Traditionally, irreversible MAOIS have been stopped two weeks before operation; however omitting the dose of moclobemide (a reversible MAOI) on the day of surgery is acceptable in elective cases. Patients can be switched from an irreversible MAOI to moclobemide to avoid a prolonged period of discontinuation [11].

**Mood stabilizers:** Lithium should be stopped at least 24hr before the anesthesia .Valproate is associated with platelet- dysfunction. Carbamazepine being an Inducer of hepatic-cytochrome P450 can reduce the effects of other drugs metabolized by that system.

**Antipsychotics:** Antipsychotics, when discontinued are associated with a high relapse rate since they block dopamine receptors in limbic systems and their side effects are due to blockade of dopamine receptors histamine, alpha 1 adrenergic & cholinergic receptors.

**Anxiolytics:** Signs of withdrawal from benzodiazepines should be monitored particularly in patients who remain fasted for long periods

**Regional and local anesthetics:** May lead to hypertensive crisis due to adrenaline in patients receiving TCA's and MAOI'S.

**General Anesthesia for Modified ECT:** A standard general anesthesia for Modified ECT should be the one which meets the optimum clinical response which is predicted by the degree to which the electrical stimulus exceeds the seizure threshold[12]. So earlier the stimulus exceeds the seizure threshold, quicker will be the generation of seizure activity leading to seizure duration of sufficient length, which is the final determining factor. So the efficacy of ECT in alleviating acute depression is dependent on the duration of the induced seizure [13,14]. EEG seizure activity lasting from 25 to 50 sec. is alleged to produce the optimal antidepressant response.

Because many of the anesthetic drugs used for ECT have anticonvulsant properties, they would be expected to decrease the duration of ECT induced seizure activity in a dose dependent manner. Use of larger than necessary dosages of general anesthetics will shorten the duration of ECT induced seizure activity and could adversely affect the efficacy of the ECT treatments therefore there is a delicate balance between achieving an adequate anesthetic state and an optimal duration of seizure activity. The type of anesthetic used has a significant impact on efficacy of the treatment [15]. The goal of ECT is to produce an EEG seizure that lasts long enough to elicit an optimal anti depressant effect [16]. The ideal anesthetic agent should have a rapid onset of action and short recovery time. The pharmacokinetic properties of the anesthetic agent determine the duration of therapy. The main concerning factor with these agents is their anti-convulsant properties; therefore the effects on seizure duration, strength of the stimulus charge and recovery time after each treatment are important. All of

these factors must be taken into consideration while managing the patient's long term cognitive complications.

**Induction agents:** According to American Psychiatric Association, Methohexital remains the most widely used general anesthetic for ECT and is considered the "gold standard". Although there are data to suggest that outcome are no different between methohexital and propofol despite the decreased seizure duration with propofol. With respect to recovery of cognitive function after ECT, propofol and etomidate offered no advantage over methohexital <sup>[14]</sup>. Therefore, unless there is a specific contraindication to barbiturates (e.g. - acute intermittent porphyria) methohexital should be the anesthetic of choice. It is effective and has established safety record and low cost. When thiopentone was compared with methohexital, it showed a frequency of increased sinus bradycardia, premature ventricular contractions <sup>[17]</sup>. Etomidate reduces seizure threshold and is associated with longer seizure duration and may be helpful in patient with short seizure times (<20 seconds) despite a maximal electrical stimulus <sup>[14,18,19]</sup>

Ketamine is an anesthetic agent with analgesic properties that are less desirable due to its ability to increase intracranial pressure. Benzodiazepines should be ruled out as an option because of their noticeable anticonvulsant activity. Although sevoflurane can be used to produce an adequate anesthetic state for ECT, being a volatile agent it is more time consuming and possesses no advantage over other IV anesthetics except for women requiring ECT in the late stages of pregnancy when it may reduce post ECT uterine contractions.

**Muscle relaxants:** Although it is not essential to have complete muscle paralysis, muscle relaxants are the indispensable drugs for modified ECT, if not used will result in vigorous physical restrained during the seizure and severe myalgia after the procedure.

As ECT is a short duration procedure, succinyl choline 0.5mg/kg is the agent of choice due to its rapid onset and short duration. In patients with a history of post ECT agitation related to increased levels of plasma lactate, increasing the dose of succinylcholine up to 1.5mg/kg may decrease the emergence delirium <sup>[20]</sup>.

Even small doses of this rapid and short acting muscle relaxants can produce side effects(e.g.- myalgias, hyperthermia and hyperkalemia ) in at risk patients with susceptibility to malignant hyperthermia , neuroleptic malignant syndrome (NMS), catatonic schizophrenia and organophosphate poisoning <sup>[21,22,23]</sup>. Therefore an ultra short acting non depolarizing muscle relaxant would be valuable addition to the anesthesiologists armamentarium. Mivacurium is the drug most often administered as an alternative to succinylcholine during ECT<sup>[21,24,25,26,27]</sup>. Mivacurium (0.08mg/kg) when compared to succinylcholine (0.5mg/kg), succinylcholine was found to be more effective in preventing muscular contractions during ECT <sup>[25]</sup>. In a patient with a history of NMS, only a full intubating dose of mivacurium (0.2mg/kg I.V) was effective for ECT <sup>[24]</sup>. But a full intubating dose of mivacurium can be associated with a clinically significant histamine release and occasional hypotension

and requires the use of anti-cholinesterase drugs to reverse residual paralysis after ECT. Rapacuronium is a newer amino steroid muscle relaxant with a rapid onset and short duration of action. It is associated with bronchospasm. Other non-depolarizing muscle relaxants like atracurium (0.3-0.5 mg/kg) or rocuronium (0.6 mg/kg) can be safely used, though sufficient time must be allowed for the onset of the drug and airway, management must be anticipated while waiting for the effects to wear off.

**Drugs used to control cardiovascular response:** - Anti-cholinergic drugs are used to block parasympathetic responses, whereas acute sympathetic responses are attenuated with B-blockers, calcium channel blockers, alpha2-agonists and direct acting vasodilators. Rapid short acting opioid analgesics also possess sympatholytic affect and have recently been investigated as adjuvants during ECT.

**Anti-cholinergics;** Glycopyrrolate does not cross blood-brain barrier and is preferred over atropine as it reduces oral secretions and bradycardia without producing post-ECT side effects.

**B-blockers:** Esmolol (short acting B1-receptor blocker) 1.0 mg/kg more effectively attenuates the blood pressure response than labetalol (0.3 mg/kg). However Labetolol is controversial about reducing seizure duration<sup>[28,29,30]</sup>. To minimize this labetalol can be administered immediately before or after the electrical stimulation is applied.

**Calcium-channel blockers:** Nicardipine (1.25-2.5 mg/kg i/v) in combination with Labetolol (10 mg/kg) more effectively reduces ECT induced hemodynamic response. Nicardipine in a bolus dose of more than 5 mg i/v was accompanied by a reflex increase in heart rate. Small dose of nicardipine did not alter the ECT –induced seizure duration <sup>[31]</sup>. Nifedipine has to be given sublingually 20 min before ECT.

**Alpha-2 Agonists/Antagonists:** Clonidine ( alpha-2 agonist/antagonist) when given orally in a dose of .05-0.3 mg , 60-90 min before induction of anesthesia produced a dose related decrease in mean arterial pressure but not in heart rate immediately before the electrical stimulus was applied , but no significant effect after the stimulus. Dexmedetomidine (an alpha -2 agonist) despite having no effect on seizure duration does not appear to control the acute hemodynamic response.

**Direct vasodilators:** Nitroglycerine (NTG) in a dose of 3µg/kg i/v effectively reduces hyperdynamic response without having any effect on seizure duration. It should be considered for ECT patients who are at a high risk of developing myocardial ischemia. It partially inhibits the increase in cerebral blood flow velocity associated with ECT.

**Ganglion blockers:** Trimethaphan in bolus doses of 5, 10 &15 mg also controls the hyperdynamic response during ECT without altering the duration of seizure.

**Local anesthetics:** Lidocaine (1.0 mg/kg) is not effective and it produces dose-related decrease in the duration of both motor and EEG activity.

**Opioid analgesics:** Alfentanil , a short acting opioid analgesic , in a dose of 25 µg/kg i/v has been found to increase the seizure duration by 45% when combined

with methohexital (0.5 mg/kg) in comparison to standard dose of methohexital 0.75 mg/kg alone<sup>[32]</sup>. Fentanyl does not attenuate the hyperdynamic response post-ECT. Remifentanyl also prolongs the seizure duration<sup>[33]</sup>. Therefore increased seizure duration associated with the short acting opioid analgesic alfentanil and remifentanyl appears to be related to a reduction in the intravenous anesthetic dosage requirements. In ECT patients with borderline seizure times, adjunctive use of a potent rapid and short acting opioid analgesic could be very beneficial.

**Standard general anesthetic technique:** Although patients are required to fast overnight for solid food, clear liquids are allowed for taking oral medications up to 1 hour before the procedure. Patients with cardiovascular disease should be encouraged to take all chronic antihypertensive medications before ECT. To prevent post-ECT myalgias patients can be pre-medicated with enteric-coated aspirin (650 mg orally) or acetaminophen (650mg orally). In younger patients at risk for severe ECT induced myalgias, headaches or both Ketorolac 30 mg i/v can also be administered before the induction of anesthesia. Finally, to minimize the pain on injection of methohexital and propofol, lidocaine 0.5-1.0 ml can be injected in i/v catheter immediately before administering the induction drug. Patient is oxygenated for 3 minutes. Adequate neuromuscular blockade is achieved; satisfactory ventilation with oxygen is ensured using a face mask with a standard circle or a simple bag-mask-valve system. A bite block is placed routinely before electrical stimulus is delivered. Since, ECT procedure lasts only a few minutes, tracheal intubation is not recommended except in very specific situations (e.g. Late pregnancy, emergency treatments with full stomach precautions, hiatal hernia and oesophageal reflux). Rapid sequence induction and endotracheal intubation with cricoids pressure is a reasonable approach in such cases. Adequate ventilation is ensured because hypoxia and hypercarbia decrease seizure duration<sup>[34,35]</sup>. Manual ventilation is commenced during the clonic phase to avoid oxygen-desaturation and should be maintained until adequate spontaneous ventilation resumes. Peripheral seizure is monitored by electromyogram and the central seizure is monitored by electroencephalogram. Central seizure duration may outlast peripheral clonic manifestations. A blood pressure cuff inflated on a limb to isolate it prior to neuromuscular block administration can assist in monitoring of peripheral seizure. During the recovery period, the most common side effects are confusion, agitation, amnesia and headache. Intranasal administration of 5-hydroxy tryptamine-1 agonist, sumatriptan may be used to treat headache. Nausea, vomiting and dizziness are infrequent complications after ECT. Standard non-invasive hemodynamic variables and oxygen saturation should be monitored for 15-30 minutes<sup>[36]</sup>, however adjusting the dose of succinylcholine and adding a small dose of methohexital (10mg i/v) at the end of the seizure may reduce the incidence of post-ECT agitation.

**Special cases:**

**Patients with cerebral aneurysm:** Because ECT provokes abrupt changes in both systemic and cerebral hemodynamics, the cerebrovascular changes increase wall stress in aneurysm leading to enlargement or rupture, arterial cannulation is required to control blood pressure<sup>[37]</sup>. Administration of sodium-nitroprusside 30 µg/min i/v in combination with atenolol 50 mg orally effectively controlled the cardiovascular changes associated with ECT<sup>[38]</sup>.

**Intracranial mass Patients with subdural hemorrhage and lesion:** In such patients intracranial pressure should be reduced by pre-treatment with steroids and diuretics and by hyperventilation before applying the electrical stimulus<sup>[39]</sup>. Use of dose-titration method of ECT with unilateral electrode placement away from the site of the lesion minimizes the risk of adverse neurologic outcomes and post-procedure neuroimaging scans are recommended.

**Patients with pre-existing cardiovascular disease:** Pre-treatment with beta-blockers is strongly recommended in patients with coronary artery disease<sup>[40]</sup>. In patients with atrial fibrillation, considering high risk of embolization anticoagulation therapy should be started before ECT. In cases with pre-existing bradycardia (or sick sinus syndrome) pre-treatment with atropine is strongly recommended, especially in patients with myasthenia gravis who are receiving pyridostigmine.

**Patients with NMS:** It shares some clinical similarities to malignant hyperthermia. Well known triggering drugs (e.g. Succinylcholine and sevoflurane should be avoided). Non-depolarizing muscle-relaxants (e.g. mivacurium) have been successfully used in place of succinylcholine.

**Patients with inadequate seizure activity:** Etomidate is the drug of choice in patients experiencing inadequate seizure activity when a maximal electrical stimulus is applied<sup>[14]</sup>. Aminophylline<sup>[41]</sup> has been reported to lengthen the seizure duration. Theophylline 100-200 mg infused approximately 30 min before the ECT treatment prolonged the seizure duration. Caffeine is also reported for the same.

**Pregnant patients:** ECT is considered safe and effective for the mother and fetus in the treatment of major depressive disorder during pregnancy<sup>[42]</sup>. Patients in late pregnancy should lie on their left side during ECT to ensure adequate blood flow to the fetus. Hyperventilation is to be avoided<sup>[43]</sup>. In addition to securing patient's trachea with endotracheal tube, after a rapid sequence induction with cricoids pressure, consideration should be given to prophylactic use of tocolytic therapy in cases of premature labor or uterine contractions. In later stages of pregnancy, use of sevoflurane as an alternative to methohexital may reduce the risk of uterine contractions.

**ECT in elderly:** As seizure threshold may rise with increasing age and effective seizures may be hard to induce<sup>[44]</sup>. Geriatric, patients may be at a higher risk for persistent confusion and greater memory deficits during and after ECT<sup>[44]</sup>.

**CONCLUSION**

As, despite many advancements in pharmacologic management of psychiatric illnesses, electroconvulsive therapy still remains the effective mode of treatment in many drug resistant cases. An effective treatment results, only when an adequate seizure of a minimum of 30 seconds duration results. This can be achieved only with a thorough knowledge and understanding of anesthetic drugs, their interactions with many other concurrent psychotropic drugs and various other special conditions often encountered in such patients. So, the role of a balanced general anesthesia in electroconvulsive therapy can never be ignored.

**Conflict of interest:** None

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