TITLE OF THE ARTICLE: "ANAESTHETIC CONSIDERATION IN MODIFIED ELECTROCONVULSIVE THERAPY: A NARRATIVE REVIEW ARTICLE"

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INTRODUCTION

Electro convulsive therapy (ECT) is a safe and effective treatment for various psychiatric disorders like refractory major depression, mania and catatonic schizophrenia. ECT consists of programmed electrical stimulation of the central nervous system to initiate a generalized epileptic seizure, though its exact mechanism of action still remains unknown. LucioBini and UgoCerlettiwere the first to use ECT for the treatment of schizophrenia in the year 1938. Initially electrical current was used in controlled fashion in conscious patients to induce seizures activity in brain (Direct ECT), later in 1960 the term 'Modified ECT' was introduced which includes the use of anaesthesia and muscle relaxants.This article primarily focuses on physiological responses, preoperative evaluation and perioperative anaesthesia management during ECT administration.

Administration of ECT

During ECT, an electric current is passed briefly through the brain, via two electrodes applied to the scalp either unilaterally or bilaterally (bitemporalor bifrontal).Unilateral ECT is usually performed on the nondominant side of the brain, requires high doses and is associated with less cognitive impairment.

Bilateral ECT ismore effective than unilateral ECT andis more commonly used. Ideally the duration of seizure should range between 15 to 120 seconds. A seizure longer than 120secis classified as "prolonged" and needs to be terminated by pharmacological agents.During a course of ECT, most of the patients require 6 to12 settings, which are usually given on twice a week basis. The maintenance therapy used to prevent relapses can be performed at progressively increasing intervals from once a week to once a month, but its exact role is controversial.

Indications According to National Institute of Clinical Excellence (NICE) UK Guidelines [(1)] ECT should be considered forpatients suffering from:

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

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.

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.

ECT is not generally considered for the treatment of schizophrenia, unless there is an affective component. It is generally not considered for those less than 16 years of age.

Contraindications

Although there are no absolute contraindications to ECT, the relative contraindications include increased intracerebral pressure, space-occupying cerebral lesions, cerebral aneurysms, recent intracerebralhemorrhage, pheochromocytoma, recent myocardial infarction, unstable major fracture or cervical spine injury, decompensated cardiac failure, severe valvular disease and deep venous thrombosis.

Physiological responses to ECT

ECT affects functioning of various vital organs including cardiovascular, central nervous and endocrine systems and changes occurring are summarised below.

1. Cardiovascular system

The cardiovascular responses of ECT are secondary to autonomic nervous system stimulation. Initially there is a brief parasympathetic stimulation lasting for 10-15 seconds resulting in transient sinus bradycardia, hypotension, or rarely asystole. This is immediately followed by a more substained sympathetic response due to the release of catecholamines, resulting in tachycardia, hypertension, and arrhythmias commonly ectopic beats, bigeminy and supraventricular tachycardia, which are usually transient and self-limited.Most hemodynamic changes persist into the recovery period and resolve within 20-30 minute. During ECT, there is increased myocardial oxygen demand along with decrease myocardial oxygen supply due to seizure-induced increased tissue oxygen consumption, which may increase the risk of myocardial ischemia and infarction especially in patients with pre-existing cardiac dysfunction.

2. Central Nervous System

ECT leads to increase in cerebral blood flow, cerebral oxygen consumption, and intracranial pressure. Rarely it can produce transient neurologic ischemic deficits, intracerebral haemorrhages, and cortical blindness. [(2)]Among the cognitive effects headache, confusion, and transient memory loss are common in the immediate post-treatment period. The memory loss can be retrograde, anterograde, or both. The degree and type of memory loss are related to the electrode placement, type of stimulus, and age of the patient.[(3)]Serious cognitive dysfunctions are rare and are not associated with any direct neuronal damage.[(4)]The baseline seizure threshold increases in epileptic patients and during the course of ECT treatment. Most cognitive deficits usually resolve within 6 months after the initiation of treatment.

3. Other Systemic Effects

In addition to acute neurologic and cardiovascular effects, ECT may result in increase in intraocular pressure and intragastric pressure, nausea, salivation, myalgia, emergence agitation, and rarely sudden death [(5, 6).] Other complications include tongue and gums damage, damage to crowns, veneers, bridges, implants or intraosseous denture supports.

4. Mortality

ECT is a low-risk procedure and mortality rate is about 1 per 10,000 patients (1 per 80,000 treatments) which is similar to that of mortality during anaesthesia for minor surgical procedures. Cardiovascular (arrhythmia and myocardial infarction) and, to a lesser extent, pulmonary (laryngospasm and aspiration) complications are the most common causes of morbidity and mortality.

Preoperative anaesthetic considerations

Ideally preanaesthetic assessment should be carried out in all the patients, along with preoperative optimization of concurrent diseases. Usually, these patients are poor historians, and may have neglected regular medications, so a thorough history and physical examination of the patient should be carried out, to weight the relative risk of the untreated psychiatric illness against the risk of anaesthesia and ECT. It includes a detailed medical history, thorough physical examination and a review of laboratory data.

- **History and Physical Examination**
  It includes systemic examination of all systems, particularly central nervous and cardiovascular systems. The aim is to screen patients for ischemic heart disease, congestive heart failure, hypertension, arrhythmias, intracranial mass lesions, recent stroke, chronic obstructive pulmonary disease, and gastro-oesophageal reflux. Documents of previous anaesthetic history, allergies, and current medications are of great help. Airway evaluation including dentition should be thoroughly examined and any artificial teeth should be documented. A record of the patient's baseline vital signs and weight should be made. Preoperative fasting must be confirmed along with informed consent prior to general anaesthesia administration.
- **Investigations**
  Minimum pre-treatment laboratory data guidelines have not been suggested for preparing patients for ECT. Nevertheless, young, medically healthy patients may not require any laboratory tests. Laboratory testing can be tailored to the patient's medical history and medication. Lithium levels should be checked if the patient has been on long term lithium treatment.

Anesthetic management

Objective of anaesthesia for ECT is to make patient unconscious and adequately paralyzed for brief duration and rapid return of consciousness and orientation, while minimizing physiological and physical effects. Anesthesia for ECT is generally provided at remote centers. The Royal College of Anaesthetists, United Kingdom and the Association of Anaesthetists of Great Britain and Ireland's standards recommends that anaesthesia for ECT should be administered by experienced anaesthetists and that assistance should also be suitably trained.[7] All equipment and drugs for airway management and resuscitation should be immediately available.

Induction agents

The pharmacodynamic properties of most of induction agents are such that they may decrease the duration of ECT in a dose dependant manner, so overdose can adversely affect the effectiveness of the ECT treatment. Hence,induction agent for ECT should be chosen to ensure rapid onset and offset of consciousness, minimal hemodynamic effects, and minimum effect on seizure quality and be inexpensive. For the first session of ECT, dose is titrated against patient's weight but for subsequent treatments it is modified depending upon clinical response, haemodynamic stability, and seizure threshold.

Methohexital has been regarded as the "gold standard" drug for ECT by the American Psychiatry Association as it has many properties of ideal induction agent for ECT, however due to lack of easy availability, other induction agents have been more widely used.[8]A recent systematic review concluded that no induction agent except for

ketamine has overwhelming advantages or disadvantages over one another and all are suitable for ECT and the small variations in emergence and recovery times should not govern drug choice. [9]Whichever drug is used, it is preferable to utilize the same one throughout the course of treatment to avoid interference with the seizure threshold. Advantages and disadvantages of commonly used induction agents are summarized in table1

Neuromuscular blocking agents

Though it is not essential to have complete muscular paralysis, moderate degree of muscular relaxation is required to prevent injuries to the musculoskeletal system and to improve airway management. The reduced or 'modified' muscular contractions in response to the electrical stimulation, along with EEG monitoring, are used to monitor seizure duration.

Succinylcholine (0.5 mg/kg) has been used for ECT since 1950's and is still the muscle relaxant of choice. Larger doses of upto 1.5 mg/kg may be required, particularly in cases of severe cachexia, osteoporosis, or pre-existing skeletal injury.[10, 11] When succinylcholine is contraindicated, ultra-short acting non-depolarizing muscle relaxants (NDMR) like mivacurium is a valuable option for muscle relaxation.[12-14] Because of long duration of action, other NDMR are not routinely used for ECT.

A study by Hoshi et al[15] showed that rocuronium-sugammadex produced longer durations of seizure activity, illustrating the potential benefits of rocuronium-sugammadex as an alternative to succinylcholine. Recently Mirzakhani et al conducted a study to determine minimum effective dosing (lowest dose to provide a predefined qualitative measure of acceptable control of muscle strength during induced convulsions) of succinylcholine and rocuronium for ECT. They concluded that the initial ECT dose of succinylcholine should be selected based on each patient's preprocedural condition, ranging between 0.77 and 1.27 mg kg-1 to produce acceptable muscle blockade and rocuronium–neostigmine combination is a safe alternative if appropriately dosed (0.36–0.6 mg kg-1) and monitored.

Adjuvant drugs

Acute cardiovascular responses secondary to ECT may be life threatening, so several drugs have been tried to attenuate acute sympathetic and parasympathetic responses and also to decrease requirement of concomitantly administered anesthetic agents especially in high risk population.[16-19] Some of these drugs can affect ECT induced seizure quality, therefore selection of adjuvants should be tailored to the needs of the individual patient.

- **Anti-cholinergic agents:** To counter parasympathetic reponses to ECT,glycopyrrolate (100-600 mcg) or atropine (300-600mcg) are used. In routine practice glycopyrrolate is preferred over atropine it has more antisialogogue action, lesser tachycardia, and fewer CNS side effects and decrease myocardial work as compared with atropine . [20]
- **Beta-blockers:** Pretreatment with short acting beta-blockers such as esmolol (1.0- 2.0mg/kg), labetolol (0.05-0.4mg/kg) can attenuate sympathetic stimulation. However, there is controversy about the relative effects of esmolol and labetalol on the duration of seizure activity. Therefore to minimize the potential adverse effect on the duration of seizure activity, these can be administered immediately before or after the electrical stimulation is applied.[21-23]
- **Opioid analgesics:** Opioids like fentanyl,alfentanyl and remifentanil