Review Article

Anaesthesia for electroconvulsive therapy: An overview with an update on its role in potentiating electroconvulsive therapy

Address for correspondence:
Dr. Pavan Kumar Kadiyala,
Siddhartha Medical College,
Vijayawada - 522 008,
Andhra Pradesh, India.
E-mail: drkadiyala2@gmail.com

Pavan Kumar Kadiyala, Lakshmi Deepthi Kadiyala[1]
Department of Psychiatry, Siddhartha Medical College, Vijayawada, Andhra Pradesh, [1]Department of Anaesthesia, JJM Medical College, Davanagere, Karnataka, India

Access this article online
Website: www.ijaweb.org
DOI: 10.4103/ija.IJA_132_17
Quick response code

ABSTRACT

Despite advances in pharmacotherapy, electroconvulsive therapy (ECT) remains a mainstay treatment option in psychiatry since its introduction in 1930s. It can be used primarily in severe illnesses when there is an urgent need for treatment or secondarily after failure or intolerance to pharmacotherapy. The 'unmodified' technique of ECT was practised initially, with a high incidence of musculoskeletal complications. Several modifications including general anaesthesia and muscle relaxation are used to increase the safety and patient acceptability of ECT. Various anaesthetic techniques including medications are considered to provide adequate therapeutic seizure, simultaneously controlling seizure-induced haemodynamic changes and side effects. A brief review of literature on choice of these anaesthetic techniques is discussed. This article is intended to reinforce the knowledge of clinicians, who may have limited exposure to ECT procedure. Importance is given to the recent updates on the role of induction agents in potentiating therapeutic response to ECT in psychiatric disorders.

Key words: Anaesthesia, electroconvulsive therapy, synergistic effect

INTRODUCTION

Electroconvulsive therapy (ECT), introduced by Cerlitti and Bini in 1937, is the induction of a generalised seizure by electrical stimulation of one or both cerebral hemispheres. It has become a highly sophisticated and precise procedure with passage of time. Initially, 'unmodified' technique was practised in which patients were conscious and without muscle relaxation. This resulted in musculoskeletal complications in as many as 40% patients. Beginning in the 1950s and 1960s, however, several refinements including anaesthetic medications and muscle relaxants were introduced to increase the safety and patient acceptability.[1-3]

Initially, ECT was used to treat several types of psychiatric disorders and to calm disruptive inpatients in psychiatric wards, regardless of their diagnosis. In recent years, its use is restricted primarily to severe mental illnesses when there is an urgent need for treatment or secondarily after failure or intolerance to pharmacotherapy. Although there are no absolute contraindications to ECT, some medical conditions are known to increase the risk due to the significant cardiovascular and cerebrovascular changes associated with ECT. The cardiovascular effects result from autonomic nervous system (ANS) activation during ECT procedure. This leads to an initial 10–15 s of parasympathetic discharge resulting in bradycardia and occasional asystole (the tonic phase). This is followed by a pronounced sympathetic response of hypertension, tachycardia and other arrhythmias peaking 1 min after ECT stimulation and generally resolving within 5–10 min thereafter (the clonic phase). There is increased cerebral metabolic rate (CMR) which results in a marked increase in cerebral

How to cite this article: Kadiyala PK, Kadiyala LD. Anaesthesia for electroconvulsive therapy: An overview with an update on its role in potentiating electroconvulsive therapy. Indian J Anaesth 2017;61:373-80.

blood flow (CBF) and intracranial pressure (ICP). Furthermore, there is increased intraocular and intragastric pressure. Short-term memory loss is also common.[1-3]

The anaesthetic requirements for ECT include control of these haemodynamic changes and related complications, together with the primary requirements of amnesia and muscle relaxation. Although these are essential, the level of anaesthesia should not be so deep as to overly suppress the seizure activity which is the goal of the treatment. The clinician must be well versed on the anaesthetic management of patients undergoing ECT. This article is a narrative overview of existing literature intended to reinforce the knowledge of clinicians, who may have limited exposure to ECT procedure. Relevant information was extracted from searches of computerised databases, hand searches and authoritative texts from inception through 18th February 2017, and the search was limited to the English language. Recent updates on the role of induction agents in potentiating therapeutic response to ECT in the treatment of psychiatric disorders are discussed in detail.

ADMINISTRATION OF ANAESTHESIA

Pre-medication

Anticholinergic agents such as glycopyrrolate and atropine are often given to antagonize the initial parasympathetic discharge. Glycopyrrolate (0.01 mg/kg) is preferred and administered either intramuscularly at least 3 min prior the scheduled procedure or intravenously just before injecting the induction agent. It may decrease the likelihood and severity of bradycardia or asystole and the risk of aspiration due to vagal effects of ECT. Routine use of anticholinergics, however, has been criticised as unnecessary. They may be particularly useful in patients who are receiving sympathetic blocking agents, such as beta-blockers or to whom a seizure threshold has not yet been established (i.e., for those undergoing their first ECT session). Beta blockers such as esmolol (1 mg/kg) and labetalol (0.3 mg/kg) can be used to attenuate the sympathetic response of surge in systolic pressure and heart rate. However, they should be considered after a detailed evaluation of each patient's cardiovascular risk. Esmolol has a lesser effect on seizure duration than labetalol. Calcium channel blockers and alpha 2 agonists can also be used. Clonidine and dexmedetomidine (1 mcg/kg over 10 min before induction of anaesthesia) control blood pressure without affecting seizure duration. Medications that increase the morbidity (lithium) or decrease the efficacy (benzodiazepines, anticonvulsants) of ECT should be appraised.[1-5]

Oxygenation

Many patients became hypoxic and cyanotic with loss of sphincter control with the practice of unmodified ECT. The introduction of modified ECT with muscle relaxation results in reduced oxygen requirement. Despite this, however, cerebral oxygen consumption increases almost 200% during the seizure. It is, therefore, a standard recommendation that the lungs should be ventilated with 100% oxygen at a rate of 15–20 breaths/min, beginning approximately 1 min before the induction, continued until the resumption of spontaneous breathing. Hyperventilation may prolong the seizure.[2,6]

INDUCTION AGENTS

Induction agents provide amnesia for the brief period of electrical stimulation and the action of the muscle-relaxing agent. A variety of induction agents may be used depending on clinical characteristics of the patient. An ideal induction agent should have a short half-life with rapid onset and recovery, maintain haemodynamic stability and have no interference with seizure duration or seizure threshold.[1,2,4]

Availability and preference of induction agent

Sodium pentothal (2–4 mg/kg) was the first induction agent used because of its availability at that time. Methohexital (0.5–1.0 mg/kg), a newer barbiturate, became more popular after its development. It remained the most widely used general anaesthetic for ECT for many decades and is considered the 'gold standard'. However, it's unlicensed status in the United Kingdom and problems with its supply present practical difficulties to those who wish to use it.[1,2,7] Ketamine (1.5–2 mg/kg) and etomidate (0.15–0.6 mg/kg) might seem preferable to other agents in the light of their lack of anticonvulsant properties, however, other aspects such as drug's safety require consideration.[8] Ketamine-propofol mixture ("ketofol") and ketofol-dexmedetomidine combination (ketofol-dex mixture) can be used as alternative induction agents, which overcome disadvantages of individual agents.[9,10]

Sevoflurane (5%–8% for induction, followed by 1–2 minimal alveolar concentration [MAC] is the only inhalational agent in widespread use for induction in

ECT, with comparable effects to intravenous (IV) agents. It is preferred in patients not cooperative for IV access. It has the advantage of attenuating uterine contractions following ECT and is used in the third trimester of pregnancy.[1,11] Opioids, such as remifentanil in higher doses, can be used as a sole agent in patients refractory to seizure induction. However, they are not usually recommended as sole agents and are combined with other anaesthetic agents. They offer an advantage of attenuating haemodynamic responses to ECT and also increase the seizure duration by an induction agent dose-sparing effect.[7,12]

In the current health-care environment, use of general anaesthetic techniques with a rapid onset and recovery is essential to facilitate the discharge of the patients within 1–2 hours after the ECT. Since the half-life of propofol is shorter than that of anaesthetic barbiturates and, with the advantage of minor haemodynamic effects, it is universally becoming the induction agent of choice, in spite of higher cost. It is also considered as reference agent due to its wider use and advantages over others.[4,8,9] Because of 'smoother' anaesthesia experience and relatively greater anticonvulsant action than other induction agents, it may be the agent of choice for ECT in children and adolescents, many of whom may have prolonged seizures early in their treatment course.[12]

There is no robust evidence to recommend a particular induction agent for ECT and all currently available induction agents are suitable for ECT. They should be chosen on the basis of their effect on seizure quality, adverse effect profile and emergence time.[13] A careful balance will need to be struck between the clinical condition of the patient and the induction agent utilised. Whichever drug is used, it is preferable to utilise the same one throughout a course of ECT and rarely may need to be changed during the course of treatment.[4]

ACTION ON SEIZURE: DURATION OF SEIZURE

ECT seizure duration provides, at best, a moderate predictor of the efficacy of treatment. It is monitored by both motor and electroencephalogram (EEG) activity as each has its own limitations. The goal is to induce a motor and a central EEG seizure of at least 25 and 40 s respectively.[11]

Induction agents in the descending order of seizure duration after their use are:[1,4,7,14,15]
Etomidate > ketamine > methohexital > sevoflurane > thiopental > propofol.

The durations of EEG and motor seizures are longest after etomidate and shortest after propofol. Thus, etomidate and also ketamine are effective in patients in whom it has been found to be difficult to produce any seizure activity. Although the seizure duration of propofol is shortest, this does not alter seizure quality indicators and does not affect efficacy. Therefore, it may be useful in the presence of prolonged seizures. Methohexital produce dose-dependent decreases in seizure duration and can be minimised by giving divided doses. Alternatively, induction agents such as methohexital, propofol and etomidate are combined with a short acting, highly potent opioids, such as alfentanil (10–25 mcg/kg) or remifentanil (1 mcg/kg) but not fentanyl, to increase seizure duration.[1,7,14,16] Another useful adjunct to augment seizure duration is moderate hyperventilation (approximately twenty breaths) with an end-tidal carbon dioxide of around 30 mmHg. It is given immediately after the succinylcholine (SCh) is injected and continued (with a break for stimulus delivery) until the desired seizure duration is achieved. It is generally considered safe with an advantage of more rapid orientation following the treatment. However, there is increased chance of prolonged seizures (>120 s) and diminished drive to breathe postictally, particularly for patients with chronic obstructive pulmonary disease.[2,4,11,12]

SEIZURE THRESHOLD

Initiation of ECT routinely involves the estimation of the seizure threshold as it varies in different individuals. Stimulus should be suprathreshold to ensure therapeutic seizure. Most of the patients have seizure threshold below specified stimulation levels. However, older patients or those on anticonvulsants, have higher threshold levels. In addition, the cognitive side-effects of ECT are proportional to how much the stimulus dose is above threshold. To reduce the threshold, the medication regime should be modified or hyperventilation may be considered.[11]

Induction agents in descending order of seizure threshold reducing property are:[1,4,8,12,17-19]
Etomidate > ketamine > methohexital > thiopental > propofol.

Many anaesthetics exhibit dose-dependent anticonvulsant and/or proconvulsant properties. All agents have been reported to produce EEG excitatory activity on induction of anaesthesia. The highest

incidence appears to be with etomidate, followed by methohexital, thiopental and propofol.[1,17] A systematic review by Hooten and Rasmussen on all currently available induction agents (except ketamine) reported etomidate as the only induction agent that may reduce the seizure threshold.[14] At higher doses, all agents act as anticonvulsants. At these doses, the barbiturates (thiopental and methohexital), ketamine and propofol are well established as agents for the treatment of refractory status epilepticus.[17] To reduce the anticonvulsant effects on seizure expression, the anaesthetic-ECT time interval should be extended to as long as practically possible to facilitate the production of better quality seizures.[15] Combining opioids such as remifentanil or alfentanil with these agents reduce seizure threshold by an induction agent dose-sparing effect. Remifentanil, for instance, is the medication recommended in patients who when treated with methohexital remain refractory despite maximum stimulus.[1] Sevoflurane, similarly, has both proconvulsant and anticonvulsant properties at different doses. High concentration of sevoflurane has seizure-provoking activity, particularly in children and when used in conjunction with hypocapnea.[4,17]

EFFECTS ON CARDIOVASCULAR FUNCTION

All induction agents used in ECT, except etomidate and ketamine, blunt the acute haemodynamic response of ECT procedure.[1] Etomidate maintains cardiovascular stability compared to others and produces minimal changes in heart rate and cardiac output. Because of its reduced cardiovascular depressant properties, the acute haemodynamic response to ECT become more prominent, when compared to barbiturates and propofol.[20] Ketamine, through its sympathomimetic action, produces tachycardia and hypertension. However, they are manageable and does not impede its use.[19] Thus, all induction agents used in ECT can be used safely in patients with normal cardiovascular function.

The anaesthetic barbiturates produce dose-dependent decreases in mean arterial pressure (MAP), and an elevation in heart rate, provided that the baroreceptor reflex is active. Propofol also produces a dose-dependent decrease in blood pressure. This decrease is significantly greater than thiopental. It simultaneously blunts the baroreceptor reflex or is directly vagotonic; in some patients, this may result in significant bradycardia and even asystole. Therefore, thiopental and propofol should be used with caution in patients at risk for or intolerant of decreases in blood pressure. However, propofol should be considered in patients who have problematic pre-existing hypertension during ECT.[20,21] Unlike other anaesthetics, ketamine typically increases blood pressure, heart rate, cardiac output and myocardial oxygen consumption. Thus, it can be used in patients at risk for hypotension during anaesthesia; however, it is not an ideal drug for patients at risk for myocardial ischaemia. Sevoflurane produces a concentration-dependent decrease in arterial blood pressure, without reflex tachycardia. Thus, it may be a preferable agent in patients prone to myocardial ischaemia. Etomidate, at induction doses, typically produces a small increase in heart rate and little or no decrease in blood pressure. It has little effect on coronary perfusion pressure and reduces myocardial oxygen consumption. Thus, of all induction agents, etomidate is best suited to maintain cardiovascular stability in patients with coronary artery disease, cardiomyopathy, valvular heart disease, cerebral vascular disease or hypovolemia.[20,21] Opioids are known to blunt autonomic responses to noxious stimuli. Remifentanil and alfentanil can be combined with other agents to reduce haemodynamic response associated with ECT.[7,21]

Induction agents in the descending order of ability to increase heart rate:[22,23]
Ketamine > methohexital > thiopental > etomidate (no effect) > propofol (decreased heart rate).

Induction agents in descending order of ability to decrease blood pressure (MAP):[22,23]
Propofol > thiopental > methohexital > etomidate (no increase) > ketamine (increased MAP).

CARDIAC ARRHYTHMIAS: QT PROLONGATION

Major depression may alter ANS activity in a patient and may increase the risk of arrhythmias and sudden cardiac death. In addition, ECT may cause an acute rise in QT dispersion, which may predispose to arrhythmias. All induction agents used for ECT prolong the QT interval. Sevoflurane should be avoided in patients at risk of QT prolongation. Ketamine is not recommended because of its sympathomimetic properties. Propofol, etomidate and thiopental can be used safely.[14,24]

CEREBRAL HAEMODYNAMICS

Barbiturates, propofol and etomidate reduce CBF, ICP and CMR, as measured by cerebral oxygen

consumption (cerebral metabolic rate of oxygen [$CMRO_2$]). Because they lower cerebral metabolism, they have been used for neuroprotection in patients at risk for cerebral ischaemia. Ketamine, however, increases CBF and ICP with minimal alteration of cerebral metabolism. It is relatively contraindicated for patients with increased ICP or those at risk for cerebral ischaemia as it may aggravate a "tight" brain. Total IV anaesthesia accomplished by propofol is often used for the "tight" brain, including moderate to severe brain oedema. Volatile anaesthetics like sevoflurane also reduce $CMRO_2$ like IV agents. However, their effect on cerebral physiology is different from IV agents as they possess intrinsic cerebral vasodilatory activity, which is least with sevoflurane. At 1 MAC, CBF and ICP remain unchanged in patients with normal intracranial compliance. In patients with poor intracranial compliance and with MAC >1, sevoflurane dilates the cerebral vasculature, producing increased CBF and ICP. The increase in ICP may be prevented by hyperventilation as the response to hypocapnia is preserved during sevoflurane anaesthesia. Opioids have relatively little effect on CBF and CMR in the normal, unstimulated nervous system.[20,25,26]

Induction agents in the descending order of $CMRO_2$ reducing ability:[20,25,26]
Propofol > sevoflurane > thiopental and methohexital > etomidate > ketamine.

Induction agents in the descending order of CBF and ICP reducing ability:[20,25,26]
Propofol > thiopental and methohexital > etomidate > ketamine.

EMERGENCE TIME

Emergence time is the time from drug administration for general anaesthesia till eye opening or following commands. The differences in emergence time among induction agents suitable for ECT are small, and these small variations in emergence should not govern drug choice.[14]

Induction agents in the descending order of emergence time:[14,27]
Ketamine > etomidate > barbiturates > propofol > sevoflurane.

OTHER EFFECTS

Etomidate, in a dose-dependent manner, inhibits the adrenal enzyme 11-β-hydroxylase, important for steroidogenesis. Single induction doses of etomidate may mildly and transiently reduce cortisol levels, but no significant differences in outcome after short-term administration have been found. In addition, it can cause nausea and vomiting resulting in delayed recovery. A unique and undesirable effect of ketamine is its association with psychomimetic emergence delirium. This can be particularly prominent after rapid awakening from induction doses. Patients can experience vivid dreams, illusions, hallucinations and delusions. The risk is lower in children and in those pre-medicated with benzodiazepines.[20]

MUSCLE RELAXATION

Muscle relaxation is used to eliminate musculoskeletal injury and aid in airway management. Succinylcholine (0.5–1.5 mg/kg) remains the relaxant of choice due to its rapid onset and short duration. Atracurium, mivacurium or rocuronium may be acceptable alternatives, although their relatively prolonged action will need continued anaesthesia and/or active reversal after treatment. Sevoflurane, the only inhalational induction agent used in ECT, has an advantage as it produces skeletal muscle relaxation and enhances the effects of neuromuscular blocking agents.[3,7,20]

ROLE OF INDUCTION AGENTS IN POTENTIATING THERAPEUTIC EFFICACY OF ELECTROCONVULSIVE THERAPY

In the early days of modified ECT, barbiturates were the only choice for induction, and it did not occur to psychiatrists to be involved in decision making regarding anaesthetic agents. Over time, many anaesthetic agents have been developed. Furthermore, there has been increasing literature regarding the influence of induction agent on the therapeutic efficacy of ECT, which led psychiatrists to liaise with the anaesthesiologist in making the choice of the induction agent.[28]

Ketamine has intrinsic antidepressant properties as it is an N-methyl-D-aspartate antagonist.[12] Many studies assessed whether its antidepressant effect might be synergistic with ECT and showed mixed results.[19,29,30] It may speed the onset of antidepressant response to ECT but does not result in greater efficacy at the end of the ECT course.[27] This could be due to the potential development of tolerance due to the repeated use. Administering other induction agent (like thiopental) in alternate

treatment sessions with ketamine may prevent the development of tolerance.[31] The enhancement of efficacy early in the course by ketamine, may be particularly advantageous in two situations: (a) in right unilateral ultra-brief ECT where the onset of antidepressant effect is slower, (b) in cases of high suicide risk or high clinical severity (e.g., catatonia), when a very rapid response is required.[15,27]

Propofol or barbiturates do not have any intrinsic antidepressant properties, however, they are quite comparable to ketamine in ultimate antidepressant efficacy of ECT, though ketamine produces a quicker response. Furthermore, combining propofol with ketamine (ketofol) retains the early antidepressant property of latter, while reducing the adverse effects. This suggests that propofol combined with ketamine anaesthesia might be the technique of first-choice in patients with depressive disorders undergoing ECT.[28,32] Adding dexmedetomidine to this combination (ketofol-dex) has added anti-depressive effect following first ECT session, but not at the end. However, ketofol-dex combination has advantages in the form of increased seizure duration, lower incidence of agitation, more patient satisfaction and acceptable decrease in heart rate and blood pressure when compared to ketofol and without any significant side effects.[10]

Etomidate may improve major depressive disorder more than sodium thiopental.[28] However, depression is associated with stress-related hypothalamic pituitary adrenal axis dysregulation, and there is concern regarding the consequences of etomidate's unwanted suppression of adrenal function on depression, particularly on the course of illness.[8] However, the findings of Wang *et al*. in 2011, through measurements of serial cortisol levels at various time points during ECT, found no worrisome reductions with etomidate.[28,33] There is no literature regarding the outcomes of depression with sevoflurane or with the combination of induction agents with opioids or hyperventilation.[28]

A recent meta-analysis on the role of IV induction agents in ECT for major depression showed that after excluding trials responsible for heterogeneity, the depression scores after the ECT course were lower with methohexital compared to propofol, and lower with propofol compared to thiopental. However, it concluded based on overall data, that it was not possible to highlight the superiority of any anaesthetic agent with regards to response rate at the end of treatment. These results also show that seizure duration, as a single factor, may not explain the superiority of one anaesthetic agent over other. Furthermore, it highlighted that ketamine administration cannot be recommended to date as an anaesthetic of choice for ECT for depression, alone or in combination. Anaesthetic agents should be chosen on the basis of adverse event profile and emergence, along with effect of these medications on seizure duration.[34]

There are no specific studies on the influence of induction agents on ECT when used for mania or psychosis. Switch from depression to mania can happen in bipolar patients with ketamine's use in ECT. However, as manic switches may also be a side-effect of ECT treatment, further studies are needed.[34] Ketamine can induce psychotic symptoms, however, practitioners have used it without encountering such problems.[12] Although barbiturates can be used for the treatment of catatonia, no studies compared them with other induction agents in ECT for catatonia. Transient cognitive deficits are common after ECT which are often a reason for terminating a course of ECT before remission is achieved. Ketamine was shown to be preferable to thiopental, methohexital and etomidate in this aspect.[8]

Neuroleptic malignant syndrome (NMS) is a serious side-effect produced by some antipsychotic drugs with some clinical similarities to malignant hyperthermia (MH). Although evidence is lacking to support NMS and MH having a similar pathophysiology, caution is advised when administering general anaesthesia to patients with NMS. Agents such as sevoflurane, suxamethonium (SCh) or their combination, known to trigger MH should be avoided, though the administration of SCh to patients receiving ECT for NMS is safe. Therefore, in patients with a history of NMS, a more promising method of muscle relaxation is to use rocuronium-sugammadex as an alternative to SCh.[16]

CONCLUSION

Anaesthesia not only enables the ECT procedure but may also have a significant influence on its clinical efficacy and tolerability through the impact on electrophysiological variables and seizure parameters. Psychiatrists used to be in charge of administration and recovery from ECT, many years ago. Nowadays, the procedure is performed in an ECT administration

room where an anaesthesiologist is in charge of general anaesthesia and a psychiatrist administers ECT. Thus, anaesthesiologists and psychiatrists must be aware of not only the physiological responses to ECT and how to modify these but also understand the anaesthetic factors that may influence the efficacy of ECT.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Ross L. Electroconvulsive therapy. In: Urman R, Gross WL, Philip BK, editors. Anesthesia Outside of the Operating Room. 1st ed. New York: Oxford University Press; 2011. p. 251-9.
2. Mankad MV, Weiner RD. Anesthesia and other medications. In: Mankad MV, Beyer JL, Weiner RD, Krystal AD, editors. Clinical Manual of Electroconvulsive Therapy. 1st ed. Arlington: American Psychiatric Publishing Inc.; 2010. p. 81-93.
3. Lee M. Anesthesia for electroconvulsive therapy. In: Atlee JL, editor. Complications in Anesthesia. 2nd ed. Philadelphia: Elsevier Health Sciences; 2006. p. 903-5.
4. Gih DE, Taylor E, Biermann B. Anesthesia. In: Ghaziuddin N, Walter G, editors. Electroconvulsive Therapy in Children and Adolescents. 1st ed. New York: Oxford University Press; 2013. p. 125-39.
5. Souter KJ. Anesthesia provided at alternate sites. In: Barash PG, Cullen BF, Stoelting RK, Cahalan MK, Stock MC, editors. Clinical Anesthesia. 6th ed. Philadelphia: Lippincott Williams and Wilkins; 2009. p. 871-2.
6. Abrams R, editor. Technique of electroconvulsive therapy : Theory. In: Electroconvulsive Therapy. 4th ed. New York: Oxford University Press Inc.; 2002. p. 142-71.
7. Walker SC, Bowley J, Walker HA. Anesthesia for ECT. In: Waite J, Easton A, editors. The ECT Handbook. 3rd ed. London: Royal College of Psychiatrists Publications; 2013. p. 14-27.
8. Hoyer C, Kranaster L, Janke C, Sartorius A. Impact of the anesthetic agents ketamine, etomidate, thiopental, and propofol on seizure parameters and seizure quality in electroconvulsive therapy: A retrospective study. Eur Arch Psychiatry Clin Neurosci 2014;264:255-61.
9. Firouzian A, Tabassomi F. Is ketamine-propofol mixture (ketofol) an appropriate alternative induction agent for electroconvulsive therapy? Saudi J Anaesth 2013;7:476-7.
10. Shams T, El-Masry R. Ketofol-dexmedetomidine combination in ECT: A punch for depression and agitation. Indian J Anaesth 2014;58:275-80.
11. Dunne RA, McLoughlin DM. ECT prescribing and practice. In: Waite J, Easton A, editors. The ECT Handbook. 3rd ed. London: Royal College of Psychiatrists Publications; 2013. p. 28-44.
12. Kellner CH, editor. Electroconvulsive therapy (ECT): Technique. In: Brain Stimulation in Psychiatry: ECT, DBS, TMS and Other Modalities. 1st ed. Cambridge: Cambridge University Press; 2012. p. 56-60.
13. Lihua P, Su M, Ke W, Ziemann-Gimmel P. Different regimens of intravenous sedatives or hypnotics for electroconvulsive therapy (ECT) in adult patients with depression. Cochrane Database Syst Rev 2014;4:CD009763.
14. Hooten WM, Rasmussen KG Jr. Effects of general anesthetic agents in adults receiving electroconvulsive therapy: A systematic review. J ECT 2008;24:208-23.
15. Gálvez V, McGuirk L, Loo CK. The use of ketamine in ECT anaesthesia: A systematic review and critical commentary on efficacy, cognitive, safety and seizure outcomes. World J Biol Psychiatry 2016:1-21. Available from: https://www.ncbi.nlm.nih.gov/pubmed/27892759. [Last accessed on 2016 Nov 28].
16. Kadoi Y. Selection of anesthetics and muscle relaxants for electroconvulsive therapy. In: Saito S, editor. Anesthesia Management for Electroconvulsive Therapy: Practical Techniques and Physiological Background. 1st ed. Japan: Springer; 2016. p. 49-66.
17. Perks A, Cheema S, Mohanraj R. Anaesthesia and epilepsy. Br J Anaesth 2012;108:562-71.
18. Lee TA, Byrne RW, Sturaitis MK. Anesthetic considerations in cortical mapping and awake surgery. In: Byrne RW, editor. Functional Mapping of the Cerebral Cortex: Safe Surgery in Eloquent Brain. 1st ed. New York: Springer International Publishing; 2016. p. 77-90.
19. Kellner CH, Bryson EO. Ketamine Anesthesia for Electroconvulsive Therapy. New York, USA: Psychiatry Times; c2014. Available from: http://www.psychiatrictimes.com/electroconvulsive-therapy/ketamine-anesthesia-electroconvulsive-therapy/page/0/2. [Last cited on 2014 May 09].
20. Patel PM, Patel HH, Roth DM. General anesthetics and therapeutic gases. In: Brunton LL, Chabner BA, Knollmann BC, editors. Goodman & Gilman's the Pharmacological Basis of Therapeutics. 12th ed. New York: McGraw-Hill Companies; 2011. p. 527-64.
21. Alwardt CM, Redford D, Larson DF. General anesthesia in cardiac surgery: A review of drugs and practices. J Extra Corpor Technol 2005;37:227-35.
22. Loushin MK. The effects of anesthetic agents on cardiac function. In: Iaizzo PA, editor. Handbook of Cardiac Anatomy, Physiology, and Devices. 2nd ed. New York: Springer Science & Business Media; 2009. p. 231-41.
23. Ebert TJ, Lindenbaum L. Anesthetic agents, adjuvants and drug interaction. In: Barash P, Cullen BF, Stoelting RK, Cahalan M, Stock CM, Ortega R, editors. Clinical Anesthesia. 7th ed. Philadelphia: Lippincott Williams and Wilkins; 2013. p. 445-580.
24. Fazio G, Vernuccio F, Grutta G, Re GL. Drugs to be avoided in patients with long QT syndrome: Focus on the anaesthesiological management. World J Cardiol 2013;5:87-93.
25. Patel PM, Drummond JC, Lemkuil BP. Cerebral physiology and the effects of anesthetic drugs. In: Miller RD, Cohen NH, Eriksson LI, Fleisher LA, Weiner-Kronish JP, Young WL, editors. Miller's Anesthesia. 8th ed. Philadelphia: Elsevier Saunders; 2015. p. 388.
26. Ikeda Y, Uchino H, Miyashita R. Role and management of intracranial pressure in neuroanesthesia. In: Uchino H, Ushijima K, Ikeda Y, editors. Neuroanesthesia and Cerebrospinal Protection. 1st ed. Japan: Springer; 2015. p. 153-62.
27. Loo CK, Galvez V. Ketmaine and electroconvulsive therapy. In: Mathew SJ, Zarate CA Jr., editors. Ketamine for Treatment-Resistant Depression: The First Decade of Progress. 1st ed. Cham: Springer International Publishing; 2016. p. 123-36.
28. Rasmussen KG. Improving ECT efficacy and decreasing cognitive side effects. In: Reti I, editor. Brain Stimulation: Methodologies and Interventions. 1st ed. New Jersey: Wiley-Blackwell; 2015. p. 96-104.
29. Fond G, Loundou A, Rabu C, Macgregor A, Lançon C, Brittner M, *et al.* Ketamine administration in depressive disorders: A systematic review and meta-analysis. Psychopharmacology (Berl) 2014;231:3663-76.
30. McGirr A, Berlim MT, Bond DJ, Neufeld NH, Chan PY, Yatham LN, *et al.* A systematic review and meta-analysis of randomized controlled trials of adjunctive ketamine in electroconvulsive therapy: Efficacy and tolerability. J Psychiatr Res 2015;62:23-30.

31. Rybakowski JK, Bodnar A, Krzywotulski M, Chlopocka-Wozniak M, Michalak M, Rosada-Kurasinska J, *et al.* Ketamine anesthesia, efficacy of electroconvulsive therapy, and cognitive functions in treatment-resistant depression. J ECT 2016;32:164-8.
32. Wang X, Chen Y, Zhou X, Liu F, Zhang T, Zhang C. Effects of propofol and ketamine as combined anesthesia for electroconvulsive therapy in patients with depressive disorder. J ECT 2012;28:128-32.
33. Wang N, Wang XH, Lu J, Zhang JY. The effect of repeated etomidate anesthesia on adrenocortical function during a course of electroconvulsive therapy. J ECT 2011;27:281-5.
34. Fond G, Bennabi D, Haffen E, Brunel L, Micoulaud-Franchi JA, Loundou A, *et al.* A Bayesian framework systematic review and meta-analysis of anesthetic agents effectiveness/tolerability profile in electroconvulsive therapy for major depression. Sci Rep 2016;6:19847.

Announcement

CALENDAR OF EVENTS OF ISA 2017

The cut off dates to receive applications / nominations for various Awards / competitions 2017 is as below. Hard copy with all supportive documents to be sent by Regd. Post with soft copy (Masking names etc.) of the same by E Mail to secretaryisanhq@gmail.com. The masked soft copy will be circulated among judges. Only ISA members are eligible to apply for any Awards / competitions. The details of Awards can be had from Hon. Secretary & also posted in www.isaweb.in

Cut Off Date	Name of Award / Competition	Application to be sent to
30 June 2017	Bhopal Award for Academic Excellence	Hon. Secretary, ISA
30 June 2017	Late Prof. Dr. A .P. Singhal Life Time Achievement Award	Hon. Secretary, ISA
30 June 2017	Rukmini Pandit Award	Hon. Secretary, ISA
30 June 2017	Dr. Y. G. Bhoj Raj Award Award	Hon. Secretary, ISA
30 Sept. 2017	Kop's Award	Chairperson, Scientific Committee ISACON 2017 copy to Hon. Secretary, ISA
30 Sept. 2017	ISACON Jaipur Award	Chairperson, Scientific Committee ISACON 2017 copy to Hon. Secretary, ISA
30 Sept. 2017	Prof. Dr. Venkata Rao Oration 2017	Hon. Secretary, ISA
30 Sept. 2017	Ish Narani Best poster Award	Chairperson, Scientific Committee ISACON 2017
30 Sept. 2017	ISA Goldcon Quiz	Chairperson, Scientific Committee ISACON 2017
10 Nov. 2017	Late Dr. T. N. Jha Memorial Award & Dr. K. P. Chansoriya Travel Grant	Hon. Secretary, ISA, copy to Chairperson Scientific Committee of ISACON 2017
20 Oct. 2017	Awards (01 Oct 2016 to 30 Sept 2017)	Hon. Secretary, ISA

(Report your monthly activity online every month after logging in using Secretary's log in ID)

1. Best City Branch
2. Best Metro Branch
3. Best State Chapter
4. Public Awareness – Individual
5. Public Awareness – City / Metro
6. Public Awareness - State
7. Ether Day (WAD) 2017 City & State
8. Membership drive
9. Proficiency Awards

Send hard copy (where ever applicable) to
Dr. Venkatagiri K.M.
Hon Secretary, ISA National
"Ashwathi"' Opp. Ayyappa temple,
Nullippady, Kasaragod 671 121.
secretaryisanhq@gmail.com / 9388030395.