

Outcome of Four Pretreatment Regimes on Hemodynamics during Electroconvulsive Therapy: A Double-blind Randomized Controlled Crossover Trial

Abstract

Context: Electroconvulsive therapy (ECT) is associated with tachycardia and hypertension. **Aims:** The aim of this study was to compare two doses of dexmedetomidine, esmolol, and lignocaine with respect to hemodynamics, seizure duration, emergence agitation (EA), and recovery profile. **Methodology:** Thirty patients undergoing ECT were assigned to each of the following pretreatment regimes over the course of five ECT sessions in a randomized crossover design: Group D1 (dexmedetomidine 1 µg/kg), Group D0.5 (dexmedetomidine 0.5 µg/kg), Group E (esmolol 1 mg/kg), Group L (lignocaine 1 mg/kg), and Group C (saline as placebo) before induction. Heart rate (HR), mean arterial pressure (MAP), seizure duration, EA, and time to discharge were evaluated. **Results:** Groups D1, D0.5, and esmolol had significantly reduced response of HR, MAP compared to lignocaine and control groups at 1, 3, 5 min after ECT ($P < 0.05$). Motor seizure duration was comparable in all groups except Group L ($P = 0.000$). Peak HR was significantly decreased in all groups compared to control. Total propofol requirement was reduced in D1 ($P = 0.000$) and D0.5 ($P = 0.001$) when compared to control. Time to spontaneous breathing was comparable in all the groups ($P > 0.05$). Time to eye opening and time to discharge were comparable in all groups ($P > 0.05$) except Group D1 ($P = 0.001$). EA score was least in Group D1 ($P = 0.000$). **Conclusion:** Dexmedetomidine 1 µg/kg, 0.5 µg/kg, and esmolol produced significant amelioration of cardiovascular response to ECT without affecting seizure duration, results being best with dexmedetomidine 1 µg/kg. However, the latter has the shortcoming of delayed recovery.

Keywords: Dexmedetomidine, electroconvulsive therapy, esmolol, hemodynamics, lignocaine

Introduction

Electroconvulsive therapy (ECT) is frequently used for the treatment of psychiatric illness.^[1] The cardiovascular response generated by ECT is a brief parasympathetic sequence followed by sympathetic stimulation during the seizure, which markedly increases plasma levels of catecholamines, thereby increasing heart rate (HR) and mean arterial pressure (MAP). These hemodynamic effects can produce cardiovascular stress and could place a patient with coronary or cerebrovascular disease at the risk of an acute coronary or cerebrovascular event.^[1] Several drug regimens have been used to prevent or to attenuate the hemodynamic response to ECT, including nitroglycerine, fentanyl, labetalol, esmolol, clonidine, and dexmedetomidine.^[2-6] Each drug is reported to have its own merits and demerits for ECT in terms of their effects on hemodynamics, seizure duration, and recovery.^[2]

ECTs are frequently performed on an outpatient basis; therefore, a sensitive balance is to be warranted with pretreatment regimens with respect to their effects on cardiovascular hemodynamics, without affecting the efficacy of ECT and also home discharge. Anesthetic drugs used for the procedure should be short-acting with rapid recovery profiles.

We did not find any randomized controlled study which prospectively compared dexmedetomidine, esmolol, and lignocaine in terms of attenuation of hemodynamic effects during ECT in the same group of patients. This prospective randomized placebo-controlled double-blind study with four-way within-patient crossover design primarily compares the effects of two doses of dexmedetomidine, esmolol, and lignocaine on hemodynamic response during ECT under general anesthesia. Return of spontaneous breathing, eye

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opening, seizure duration, and readiness to discharge formed the secondary end-points.

Methodology

This prospective randomized placebo-controlled double-blind study was conducted after the Institutional Ethics Committee approval. Thirty patients of either sex, belonging to the American Society of Anesthesiologists (ASA) Grade I, with ages between 18 and 50 years, requiring 4–8 or more ECTs were invited to participate, and informed consent was obtained from them and their legal guardians. Patients with known sensitivity or allergy to study drugs, pregnant and lactating females were excluded from the study. Each patient's ECT-induced seizure threshold was determined in the first two ECT sessions. These two sessions were not included in the study. After this, patients were studied during five subsequent ECT treatments in a randomized crossover design. The crossover design enabled the patient to receive each of the five treatments. Same patients were included to avoid any patient bias in terms of demographical characteristics and antipsychiatric medications. This was done to remove confounding variables on hemodynamics, seizure duration, and recovery parameters. There was a washout period of at least 48 h between two subsequent ECT treatments. They were assigned to one of the five study groups based on computer-generated random numbers, mentioning the sequence of treatments for each patient. Enrollment and randomization were carried out confidentially by an anesthetist not involved in the study. Random group assigned was enclosed in a sealed envelope to ensure concealment of allocation sequence. Drugs were prepared by the same anesthesiologist who carried out randomization. The patients were subsequently administered an alternative study drug in the next session. The anesthesiologist conducting the case, the patients, and the anesthesiologist in the postanesthesia care unit (PACU) were all blinded to group assignment and drugs used. Data was recorded by a blinded observer. Drugs were prepared in 50 and 10 ml syringe for each patient. Group D1 and Group D0.5 patients had dexmedetomidine 1 µg/kg and 0.5 µg/kg diluted up to 30 ml in their respective 50 ml syringes and normal saline (NS) boluses in their 10 ml syringes. Group E and Group L had esmolol 1 mg/kg and lignocaine 1.5 mg/kg diluted up to 10 ml in their 10 ml syringes and 30 ml NS in their 50 ml syringes.

On arrival in the ECT suite, after confirming adequate starvation, patient's HR, arterial blood pressure, oxygen saturation, respiratory rate, and electrocardiogram were noted (PM-9000^{Express}, Penlon, Abingdon, UK). Intravenous (IV) access was secured with 20G cannula and Ringer's lactate solution at 2 ml/kg was started. IV glycopyrrolate 0.2 mg was administered. Each patient received one infusion over 10 min followed by one 10 ml bolus over 15 s before the induction of general anesthesia.

Oxygen was administered during that time through nasal cannula at 2 L/min. In session D1, the patients received an infusion of dexmedetomidine 1 µg/kg, followed by a bolus of 10 ml NS. In session D0.5, the patients received an infusion of dexmedetomidine 0.5 µg/kg, followed by a bolus of 10 ml NS. In session E, the patients received an infusion of 30 ml NS, followed by a bolus of esmolol (1 mg/kg) in 10 ml NS. In session L, the patients received an infusion of 30 ml NS followed by a bolus of lignocaine (1 mg/kg) in 10 ml NS. In control session, the patients received a 30 ml infusion of NS, followed by a bolus of 10 ml NS.

Thirty seconds after the bolus drug was administered, anesthesia was induced with 1 mg/kg propofol and then additional 0.5 mg/kg boluses, if needed, till lack of response to verbal command and loss of eyelash reflex. The total dose of propofol was recorded. After the patient had become unresponsive, ventilation was assisted by facemask connected to Mapleson A breathing system with oxygen at 8 L/min and an arterial tourniquet was tied on one arm to isolate the limb and allow accurate assessment of the motor seizure. Succinylcholine 0.5 mg/kg was then administered, and a mouth guard was inserted after the disappearance of fasciculations. The patient's lungs were ventilated for 30 s with 100% oxygen, following which ECT was given.

A constant supramaximal stimulus (determined from ECT treatments received before entering the study) was delivered using handheld bitemporal electrodes. The patient was ventilated with oxygen during the procedure till the return of spontaneous respiration. If no seizure occurred after the stimulus or if the duration was <25 s, a second stimulus of higher intensity was administered 1 min later and noted. The duration of motor seizure was recorded as the time from start of the motor seizure to the cessation of tonic-clonic movements in the isolated arm.

The HR, MAP, and oxygen saturation were recorded at baseline, after infusion, after bolus drug, after induction of anesthesia, and at 1, 3, 5, and 10 min after ECT. Peak HR during the seizure was noted. The protocol specified that in case a higher intensity stimulus was required, then the hemodynamics of the second stimulus was also noted at 1, 3, 5, and 10 min after the stimulus, and the average of the two corresponding readings would be taken. In addition, the need for repeat stimulus for ECT, total dose of propofol, time from anesthesia induction to spontaneous breathing and eye opening was noted. The readiness to discharge was assessed by modified Aldrete score^[7] and postprocedure emergence was assessed after the patient was fully awake by emergence agitation (EA) score^[8] (1 = sleeping, 2 = awake and calm, 3 = irritable and crying, 4 = inconsolable crying, 5 = restlessness and disorientation). Adverse events such as bradycardia (HR <45 bpm), tachycardia (HR >20% of baseline), hypotension (MAP <60 mmHg), hypertension (MAP >20% of

baseline, desaturation ($\text{SpO}_2 < 90\%$), respiratory depression (respiratory rate < 10), nausea, vomiting, dry mouth, or any other event till discharge were noted and treated.

In the PACU, routine monitoring was done. The patient was deemed fit to be discharged after assessment by the psychiatrist (which is usually 1–2 h postprocedure) and when a score of 10 was achieved as per modified Aldrete's criteria,^[7] whichever was later. Patients were released into the care of the legal guardian to transport the patient.

Sample size calculation was done by EpiInfo program version 6.02 (U.S. Department of Health & Human Services, USA). The sample size was based on the sample sizes of the previous study^[9] with Type 1 error of 0.05 and power of 0.80. Twenty-seven patients were required in each study group to detect a difference of 25% in HR at 1 min of ECT between the groups. To accommodate for dropouts, thirty patients were recruited. Data analysis was done using SPSS version 16.0 (IBM corporation, USA). Z score normality tests were applied to assess whether variables were normally distributed. Results are expressed as mean \pm standard deviation for parametric data and median (range) for nonparametric data with 95% confidence intervals (CIs). Statistical comparisons were performed using one-way analysis of variance (ANOVA) with Dunnett's *t post hoc* tests to assess the significance of results. Multiple measurements within groups were analyzed by repeated measures ANOVA. Data not normally distributed were evaluated by Mann–Whitney U-test. Categorical data were analyzed using Chi-square test. $P < 0.05$ was considered statistically significant.

Results

The present study included a total of 150 ECT treatments administered to thirty patients with various psychiatric disorders (aged 18–50 years; body mass index 19.3–26.7, 13 males and 17 females). The psychiatric disorders include depression (43%), psychosis (43%), and bipolar disorder (14%). The drugs that these patients were receiving as part of their treatment schedule before and during their ECT sessions included haloperidol, pacitane, oxcarbazepine, olanzapine, clozapine, mirtazapine, fluoxetine, and quetiapine. Every patient was on two or three of the mentioned drugs. The same drugs continued through all the ECT sessions, and if any drug was changed at any point during the study period, then that patient was excluded from the study, to avoid any bias in the results. Even though the patient age group included in the study was 18–50 years (6/30 patients being above age 40), only ASA I patients were included to avoid any potential cardio or cerebrovascular event. The male-female gender difference was statistically insignificant.

This study design resulted in exactly the same number of patients being exposed to each treatment. There were no differences between the groups with respect to baseline HR and MAP. There was a significant reduction in HR

and MAP in all the study groups after the study drug administration and before the ECT. Groups D1, D0.5, and E were found to significantly blunt the increase in HR and MAP following ECT in comparison to the control group. Significant difference in mean HR at 1 min after ECT was found in Groups D1, D0.5, and E in comparison to Group C which is summarized as D1 (–22.83, 95% CI – 32.1, –13.5), D0.5 (–13.63, 95% CI – 22.9, –4.4), and E (–10.23, 95% CI – 19.5, –0.95). Similar readings were recorded at 3, 5, and 10 min post-ECT in Groups D1, D0.5, and E [Figure 1]. No significant difference in mean HR was found with Group L in comparison to Group C ($P > 0.05$).

Significant difference in MAP was found after 1, 3, and 5 min of ECT treatment in Groups D1, D0.5, and E [Figure 2] when compared to Group C. While in Group C and Group L, there was a mean increase in mean blood pressure by 13% and 15%, respectively, from their respective baselines; in D1, there was a 10.6% decrease in MAP after 1 min of ECT treatment. MAP did not change significantly from respective baselines in Groups D0.5 and E.

Peak HR was lower in all the study groups as compared to Group C which was statistically significant [Table 1]. HR and MAP values had returned to their respective baseline in all the groups within ten minutes of shifting to PACU.

Propofol requirement was significantly lower in Groups D1 ($P = 0.000$) and D0.5 ($P = 0.001$) and none of the patients required additional top ups. In contrast, additional top ups of propofol were required in Groups C, L, and E and the propofol requirement was comparable in these groups [Table 1]. The maximum propofol requirement was in control group with average dose being 1.5 mg/kg followed by Groups L and E (1.29 and 1.2 mg/kg, respectively).

There was no significant difference in the number of stimuli requirement and seizure duration between the groups ($P > 0.05$) except for the Group L which had lower seizure duration ($P < 0.05$) as compared to control group [Table 1]. However, the seizure duration in Group L

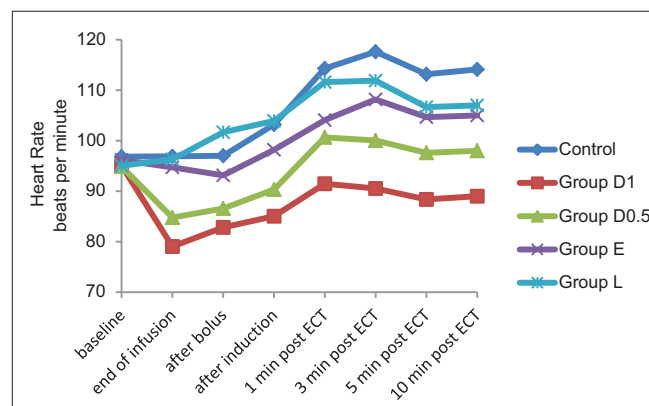


Figure 1: Changes in mean heart rate in all the groups over a period of time

averaged above 25 s, which is clinically acceptable, and hence, no additional stimuli were required in Group L or any other study group.

There were no significant differences between the groups for the time of return of spontaneous respiration in comparison to the control group ($P > 0.05$). However, the time taken for spontaneous eye opening was significantly delayed in Group D1 in comparison to the control group ($P < 0.05$). Furthermore, time required for discharge from PACU was also significantly prolonged in Group D1 ($P < 0.05$) in comparison to control (44.3 ± 16.9 vs. 12.5 ± 5 min) as well as other groups [Table 2].

No patients in any of the groups had emergence agitation though EA scores were statistically lower in Group D1 ($P = 0.006$).

There was no difference in oxygen saturation among the groups, and none of the patients complained of awareness during anesthesia. Two patients in Group E developed coughing. Headache occurred in two patients in Group C.

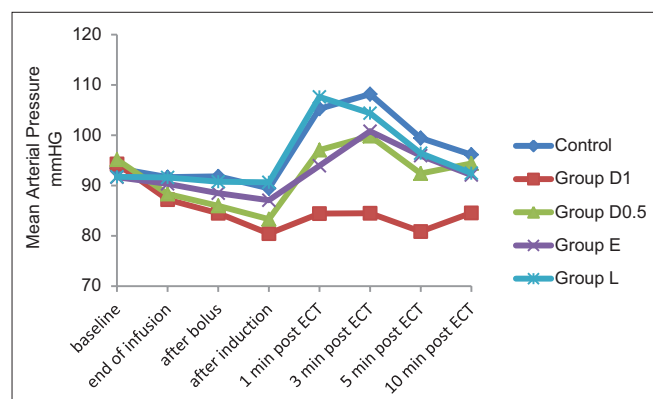


Figure 2: Changes in mean arterial pressure over a period of time

No patient experienced respiratory depression, hypoxemia, bradycardia, hypotension, or hypertension, nausea and vomiting.

Discussion

This prospective randomized placebo-controlled double-blind within-patient crossover trial demonstrates that dexmedetomidine in dose of 1 µg/kg significantly attenuates the cardiovascular response to ECT. A lower dose of dexmedetomidine (0.5 µg/kg) and esmolol (1 mg/kg) also showed significant attenuation in HR and MAP in comparison to control but not in comparison with their respective baseline. In contrast, at the doses studied, Group L did not show any ameliorating effect on HR and MAP. Total propofol requirement was reduced in dexmedetomidine and esmolol groups. However, recovery duration was significantly prolonged Group D1. Nevertheless, we observed a significant difference in EA score in D1 group among all the groups.

The previous studies showed that a variety of antihypertensive drugs have been successful in blocking the profound sympathetic stimulation generated by ECT, but with the disadvantage of prolonged hypotension.^[3,10] Dexmedetomidine is a robust α2 adrenergic agonist and is used to attenuate the hemodynamic stress response.^[9] Rapid administration of dexmedetomidine can lead to sudden exogenous release of catecholamines which may lead to tachycardia, bradycardia, and hypertension, and the minimum time needed to administer dexmedetomidine is 10 min.^[11] Dexmedetomidine, when used in a dose of 1 µg/kg, has been reported to cause prolonged discharge from PACU.^[12] Hence, we decided to compare two doses of dexmedetomidine in the same group of patients with respect to hemodynamics, seizure quality, and recovery

Table 1: Seizure duration, propofol consumption, and peak heart rate of the patients

Characteristics	Group	Mean±SD	95% CI	P
Motor seizure duration (s)	Group C	33.9±6.9	31.3-36.5	
	Group D 1	30±8.2	27-33.1	0.213
	Group D0.5	33.1±9.7	29.5-36.7	0.987
	Group E	30.1±9.4	27.6-33.6	0.226
	Group L	27.30±2.2	25.8-27.8	0.001*
Total propofol (mg)	Group C	81.73±15.0	76.36-87.1	
	Group D 1	54.79±9.8	51.27-58.31	0.001*
	Group D0.5	59.94±16.6	54-65.88	0.001*
	Group E	74.95±18.0	68.51-81.39	0.05
	Group L	76.35±17.6	70.05-82.65	0.09
Peak heart rate (beats/min)	Group C	157.47±27.34	147.26-167.68	
	Group D 1	120.47±18.79	113.45-127.49	0.001*
	Group D0.5	133.3±24	124.34-142.26	0.002*
	Group E	138.73±30.14	133.48-155.99	0.025*
	Group L	137.57±28.67	126.86-148.27	0.013*

* $P < 0.05$. Data expressed as mean±SD with 95% CI. Group C: Control group, Group D 1: Dexmedetomidine 1 µg/kg, Group D0.5: Dexmedetomidine 0.5 µg/kg, Group E: Esmolol, Group L: Lignocaine, SD: Standard deviation, CI: Confidence interval

Table 2: Recovery characteristics

Characteristics	Group	Mean±SD	95% CI	P
Time to spontaneous breathing (s)	Group C	103.6±45.6	86.6-120.7	
	Group D 1	123.3±70.2	97.1-149.5	0.403
	Group D0.5	108.6±57.4	87.2-130	0.989
	Group E	106.1±42.8	90.2-122.1	0.999
	Group L	87.8±41.9	72.2-103.4	0.595
Time to eye opening (s)	Group C	243.4±87.1	210.9-275.9	
	Group D 1	366±167.8	303.4-428.7	0.001*
	Group D0.5	262.4±81.5	232-292.9	0.894
	Group E	231.4±82.1	200.7-262	0.978
	Group L	206.8±83.6	175.6-238	0.474
Readiness for discharge from PACU (min)	Group C	12.5±5	10.6-14.3	
	Group D 1	44.3±16.9	38-50.6	0.001*
	Group D0.5	18.5±9.7	14.9-22	0.057
	Group E	13.5±6	11.3-15.8	0.979
	Group L	10.9±4.8	9.2-12.7	0.928
Emergence agitation score (1-5)	Group C	1.83±0.74	1.57-2.09	
	Group D 1	1.33±0.47	1.16-1.5	0.001*
	Group D0.5	1.7±0.59	1.49-1.91	0.22
	Group E	1.9±0.66	1.66-2.14	0.35
	Group L	1.6±0.53	1.41-1.79	0.08

* $P < 0.05$. Data expressed as mean±SD with 95% CI. Group C: Control group, Group D 1: Dexmedetomidine 1 µg/kg, Group D0.5: Dexmedetomidine 0.5 µg/kg, Group E: Esmolol, Group L: Lignocaine, PACU: Postanesthesia care unit, SD: Standard deviation, CI: Confidence interval

parameters. Esmolol hydrochloride is an ultra-short-acting, β_1 selective blocker with a distribution half-life of 2 min and elimination half-life of 9 min. It appears quite suitable for short-lived stress response like endotracheal intubation and ECT.^[13] However, high doses of esmolol (>1 mg/kg) may reduce seizure duration.^[13-15] Hence, we chose a dose of 1 mg/kg of esmolol to avoid any discrepancy in seizure duration between the groups. Lignocaine is a relatively short-acting drug which blocks the sympathetic response by blocking the voltage-gated sodium channels.^[4] The recommended dose is 1-1.5 mg/kg. Esmolol and lignocaine do not appear to have any effect on recovery parameters.

Dexmedetomidine in doses of 1 and 0.5 µg/kg as well as esmolol significantly attenuated the hemodynamic response which is in corroboration with other studies,^[3,5,8,9,15,16] the results being best with Group D1. However, Fu and White^[11] found no difference in hemodynamics with dexmedetomidine. This could be attributed to their small sample size of six patients. Moreover, they had used labetalol in all the patients which may have influenced their results. Some authors using dexmedetomidine in doses of 0.5 µg/kg^[17,18] also found no difference in hemodynamics between control and study groups which are contrary to our results. Mizrak *et al.*^[18] had studied dexmedetomidine in patients who had severe post-ECT agitation. The sample population of that study is hence different than the ones that we included in our study, which probably explains the contradictory results. Moshiri *et al.*^[17] had used atropine as premedication and thiopentone for induction which

could have probably offset the hemodynamic effects of dexmedetomidine. Lignocaine failed to show any benefit with respect to hemodynamics which is in corroboration with other studies.^[4,19] A significant proportion of patients was on antipsychotic drugs. Antipsychotic drugs alter the response to hypnotic agents and also the postanesthetic recovery period.^[20] The preoperative use of antipsychotics makes schizophrenic patients more susceptible to the hypotensive action of general anesthesia. On the other hand, discontinuation of antipsychotics may increase the episodes of psychotic symptoms such as hallucinations and agitation. Therefore, patients with chronic schizophrenia were made to continue their antipsychotics preoperatively.

The therapeutic efficacy of ECT is subjected to the duration of cerebral seizure. Although the mechanism of ECT is unknown yet, seizure duration more than 25 s is contemplated to be an index of ECT adequacy.^[21] It is recommended to monitor seizure duration by observation of motor activity and by monitoring EEG activity.^[6] We measured motor seizure duration using an isolated limb tourniquet. In the present study, we did not observe seizure duration <25 s in any of the groups. However, lidocaine in a dose of even 1 mg/kg did produce a reduction in seizure duration, though clinically acceptable; a result consistent with previous studies where dose of 1.5 mg/kg was used.^[22,23]

ECT, being mostly an outpatient affair, demands a rapid recovery as well. In the current study, recovery times were comparable in D0.5, E, L, and control groups. In contrast,

Moshiri *et al.*^[17] who used dexmedetomidine 0.5 µg/kg IV dose found a slightly prolonged recovery by approximately 3 min in comparison to control group. In the current study, home readiness discharge was significantly prolonged with D1 group. This finding is similar as observed in study conducted by Fu and White^[12] where dexmedetomidine was used in both the doses 0.5 and 1 µg/kg IV. The possible explanation for the delayed recovery could be the intrinsic sedative effects of the drug.^[12] Even though dexmedetomidine has a short distribution half-life of 5 min, the elimination half-life of 2 h could be responsible for delayed discharge.^[11] Even though Group D1 showed delayed readiness to home discharge in comparison to other groups, the 95% CIs show a duration of 38–50.6 min [Table 2] for discharge. This is well within the acceptable limits for day care procedure in an outpatient setup. Patients in this area are usually sent home one to 2 h after the procedure.

ECT itself causes restlessness and agitation due to stimulation of the central nervous system. Hence, attenuation of the EA is highly recommended. In our study, we observed that EA was significantly attenuated with D1 group in comparison to all other groups. Patients of D1 group were calmer and less agitated which is similar to other studies.^[17,18]

A possible limitation of the study is that the seizure duration of patients was assessed by observation of tonic-clonic activity using isolated limb cuff technique and not EEG basis. Although the isolated cuff technique is reliable, monitoring seizure by electroencephalography may outlast peripheral tonic-clonic manifestation. Usually, the central seizure lasts 5-8 s longer than limb movement.^[19] Furthermore, the effects of pretreatment regimes were studied only in ASA I patients. Further studies need to be carried out recruiting high-risk patients.

Conclusion

Dexmedetomidine in doses of 1 µg/kg and 0.5 µg/kg as well as esmolol in a dose of 1 mg/kg effectively blunts the hemodynamics response to ECT without affecting seizure duration. Dexmedetomidine in doses of 1 µg/kg has advantages in the form of greatest cardiovascular stability and lower EA though associated with the shortcoming of delayed recovery. With the advent of newer drugs, lignocaine may be used only when absolutely indicated.

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Conflicts of interest

There are no conflicts of interest.

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