Original Research Article

Indian Journal of Anesthesia and Analgesia
2019;6(4) (Part - II): 1355-1360
DOI: http://dx.doi.org/10.21088/ijaa.2349.8471.6419.41

A Comparison of Dexmedetomidine with Thiopentone Sodium Versus Esmolol with Thiopentone Sodium to Attenuate the Hemodynamic Stress Responses after Electroconvulsive Therapy

B Krishna Chaitanya[1], A Prashanth[2], Shaik Hala[3], Kanth Pavan Kumar[4], U Suresh Babu[5]

[1]Assistant Professor, Dept. of Anesthesia [2]Assistant Professor, Dept. of Psychiatry, [3]Postgraduate, Dept. of Anesthesia, [4]Postgraduate, Dept. of Anesthesia, [5]Assistant Professor, Dept. of Preventive Social Medicine, Maharajah's Institute of Medical Sciences (MIMS), Vizianagaram, Andhra Pradesh 535217, India.

Abstract

Modified electroconvulsive therapy (ECT) under anesthesia is an important in the treatment of severe, persistent depression; bipolar disorder and schizophrenia; especially resistant cases. However, it is commonly associated with acute hyper dynamic responses. *Aims:* To compare the effects of dexmedetomidine and esmolol on patients' haemodynamics, motor seizure duration, and recovery times following ECT. *Study Design:* Randomised Prospective Double Blinded Study. *Materials and Methods:* 90 cases aged between 18 to 50 years belonging to ASA grade I and II were randomly divided into three groups with 30 each. Group A received normal saline (placebo), Group B received dexmedetomidine 1 µg/kg, and Group C received esmolol 1 mg/kg; before induction with thiopentone sodium 2 mg/kg and muscle relaxation with succinylcholine 0.75 mg/kg. Hemodynamic parameters were recorded at different time intervals. The seizure duration using arm isolation method and recovery times using post-anesthesia discharge scoring system (PADSS) were noted. *Analysis:* Data analysis was done using SPSS (Statistical product and service solutions) software trial version 21 for windows. Results were expressed as mean ±SD, proportions and percentages. One way ANOVA test was used to assess the significant differences between groups. *Results:* PostECT rise in hemodynamic parameters was significantly less in dexmedetomidine group as compared to esmolol and control group at 2, 4, 6, and 8 min using ANOVA test. There was no significant difference in seizure duration, emergence, and recovery among the three groups. *Conclusions:* Both drugs reduce the hyperdynamic response to ECT without affecting the seizure duration, but dexmedetomidine has more favourable response in view of stable vitals, smooth emergence and no adverse effect on recovery duration.

Keywords: Dexmedetomidine; Esmolol; Modified electroconvulsive therapy.

How to cite this article:

B Krishna Chaitanya, A Prashanth, Shaik Hala. A Comparison of Dexmedetomidine with Thiopentone Sodium Versus Esmolol with Thiopentone Sodium to Attenuate the Hemodynamic Stress Responses after Electroconvulsive Therapy. Indian J Anesth Analg. 2019;6(4):1355-1360.

Introduction

Electroconvulsive therapy (ECT) is a well known treatment for severe depression in patients who are resistant to pharmacotherapy. Nowadays, almost all the ECT procedures are performed under general anesthesia; also known as modified ECT. However, it is commonly associated with acute hyper dynamic responses, including initial parasympathetic response followed by transient hypertension and tachycardia due to catecholamines release in the body**.** During the sympathetic stimulation**,** systolic blood pressure may increase by 30%–40% and heart

Corresponding Author: A Prasanth, Assistant Professor, Dept. of Psychiatry, Maharajah's Institute of Medical Sciences (MIMS), Vizianagaram, Andhra Pradesh 535217, India.
E-mail: Krrish22002@gmail.com

Received on 07.05.2019, **Accepted on** 08.06.2019

rate (HR) may increase by 20% (or more).[1] These responses may be harmful to patients with ischemic heart disease, hypertension, and cerebrovascular disease. To decrease this Sympathetic stress response many pharmacological agents like beta blockers, calcium channel blockers, α2 agonists, direct acting vasodilators, and local anaesthetics were tried.[2-6]

Dexmedetomidine is a centrally acting α agonist having high affinity for α-2 receptors with α_2:α_1 binding selectivity ratio of 1620:1 as opposed to to 220:1 for clonidine. The intravenous (IV) dexmedetomidine used as a premedicant in anesthesia as it provides sedation, analgesia, anxiolysis, and improved hemodynamic stability. It also effectively reduces the requirement of anaesthetics.[7] Esmolol hydrochloride is an ultra short acting cardio selective β1 adrenergic receptor antagonist having a rapid onset of action which is administered through intravenous route only and has a distribution half life of 2 min and an elimination half life of 9 min. After an initial dose of 0.5 mg/kg intravenously; over 60 sec, its full therapeutic effect comes in 5 min, and its action ceases within 10–30 min following the discontinuation of drug. Thus use of esmolol appears suitable to reduce short lived stress response associated with laryngoscopy, tracheal intubation, or ECT. As per psychiatry point of view, an adequate motor seizure is defined as the one that lasts more than 25–30s. The objectives of anesthesia to be kept in mind for modified ECT include rapid loss of consciousness, attenuation of hemodynamic responses, avoidance of gross movements, minimal interference with seizure, prompt, smooth and early recovery of spontaneous ventilation and consciousness. Furthermore, early ambulation and discharge to home should be considered. This study was attempted to compare the effects of dexmedetomidine and esmolol after induction with Thiopentone sodium and muscle relaxation by succinylcholine in controlling the hemodynamic stress responses, motor seizure duration and time for recovery in patients who were administered ECT.

Materials and Methods

After ethical committee clearance 90 cases, belonging to the American Society of Anaesthesiologists Classes I and II, aged 18–50 years diagnosed with major depressive disorder (suicidal patients), schizophrenia, catatonia (in which first line treatment failed), or bipolar disorder were included in the study. Patients with atrio-ventricular conduction block greater than first degree, history of major illness such as tuberculosis, bronchial asthma, hypertension, recent history of stroke, acute respiratory disorders, raised intracranial tension from any cause, systolic blood pressure (SBP) < 90 mmHg, Heart rate < 50 bpm or history of drug allergy to interventional drugs and pregnant females were excluded from the study. The study population was randomly divided into three groups (Groups A, B, and C) with thirty cases in each group. Pre-anaesthetic evaluation was done thoroughly. Airway assessment using Mallampati grading; eye examination to rule out any signs of raised intracranial tension (papilledema) and other routine investigations were done. Chronic anti depressant medications were continued on the day of surgery. The patient was kept nil per oral for 6 h. In the ECT suite, 20 G cannula was inserted, and normal saline infusion was started. Multi parameter monitors were attached to record HR, non-invasive measurements of SBP, diastolic blood pressure (DBP), mean arterial blood pressure (MAP), SPO_2 and electrocardiogram. Baseline vitals were taken after giving 5 min for stabilizing the patient. Group A (n=30) received normal saline (placebo); Group B (n=30) received Dexmedetomidine 1 μg/kg (total volume of 20 ml over a period of 10 min); and Group C (n=30) received Esmolol 1 mg/kg (total volume of 20 ml over a period of 3 min) before induction using syringe pumps. Preoxygenation was done for 3 min through face mask with Bain's circuit. General anesthesia was induced with IV Thiopentone Sodium 2 mg/kg until eyelash reflexes lost. Then, after inflating tourniquet of the other arm, succinylcholine 0.75 mg/kg IV was administered. When the fasciculations subsided, and adequate muscle relaxation was obtained, an oral soft bite block was placed in the mouth.

Psychiatrist was allowed to place bi-fronto temporal electrodes over forehead and a brief pulse stimulus of 90–120 volts maintenance electroconvulsive therapy current for 1 millisecond was given to produce seizures. The effectiveness of ECT current was determined by the appearance of tonic–clonic seizures.The ventilation was assisted with 100% O_2, until the patient resumed adequate spontaneous breathing. Following the ECT current, the Heart rate, Systolic BP, Diastolic BP, and Mean arterial pressure were recorded at 0, 2, 4, 6, 8, 10 min, and thereafter every 5 min till 30 min and then every 15 min. The duration from the begining of stimulus (ECT) to the cessation of clonic tonic motor activity in the isolated arm was recorded using clinical method. The duration of recovery from the succinylcholine especially

spontaneous breathing was recorded. Patients were assessed for side effects such as nausea, vomiting, hypotension/hypertension, respiratory depression after the electrical stimulus and were discharged from the post-anesthetic care unit to the psychiatry department according to postanesthesia discharge scoring system (PADSS) criteria.

Analysis

Data analysis was done using SPSS (Statistical product and service solutions) software trial version 21 for windows. Results were expressed as mean ± standard deviation, proportions and percentages. One way ANOVA test was used to assess the significant differences between groups. For all statistical analysis $p< 0.05$ was considered as statistically significant.

Results

There was no significant difference in the baseline variables like heart rate and mean arterial pressure among the three groups ($p>0.05$). (Table 2) There was also a significant increase in the heart rate and MAP while immediately after giving ECT and up to 2nd and 4th minute in the groups A and C ($p<0.05$) unlike Group B which showed no significant change in the HR and MAP throughout the observation period ($p>0.05$) (Tables 3 and 5).

Post Hoc tests showed that there was a significant difference ($p<0.05$) in the heart rate and MAP between Group A and B and Group A and C during infusion of the drug and at 2, 4, 6 and 8 minutes. A comparison between Group B and group C revealed significant difference regarding HR and

Table 1: Shopwing the Demographic data

	Group A (control) Mean ± SD	GroupB (dexmedetomidine) Mean ± SD	Group C (esmolol) Mean ± SD
Age (years)	33.4 ± 9.05	34 ± 8.21	34.2 ± 8.41
Sex ratio (male:female)	20:10	21:9	19:11
Weight (kg)	56.12 ± 14	55.2 ± 5.12	56.5 ± 7.16

SD- *standard deviation*

Table 2: Showing The intergroup comparison of baseline vitals

Baseline	Group A (control) Mean±SD	Group B (dexmedet-omidine) Mean±SD	Group C (esmolol) Mean±SD
HR (/min)	76.5 ± 4.52	77.03 ± 3.84	76.2 ± 4.10
SBP (mmHg)	119.93 ± 3.77	121.0 ± 3.29	120.26 ± 3.53
DBP(mmHg)	76.4+3.51	76.93 ± 3.08	77.26 ± 2.75
MAP(mmHg)	90.86 ± 3.06	91.6 ± 2.44	91.53 ± 2.36
SpO_2(%)	98.93 ± 0.77	98.86 ± 0.80	98.86 ± 0.76

HR – Heart rate; SBP – Systolic blood pressure; MAP – Mean arterial pressure; DBP – Diastolic blood pressure; SpO_2 – Oxygen saturation

Table 3: showing the Post electroconvulsive therapy changes in heart rate in Group A, B, and C at different time interval

Time(min)	Group A (beats/min)	Group B (beats/min)	Group C (beats/min)
Infusion	79.60	83.43	80.53
Induction	76.93	72.17	78.43
0	108.97	76.03	92.83
2	106.03	81.17	94.53
4	98.80	80.63	86.87
6	89.23	81.43	82.87
8	80.10	81.80	81.33
10	80.03	81.80	81.33
15	80.10	81.80	81.33
20	80.03	81.80	81.33
25	80.10	81.80	81.33
30	80.03	81.80	81.33

HR of three groups with time.

Table 4: Showing the comparison of HR among the Groups.

Time	Group A versus Group B (P value with significance)	Group A versus Group C (P value with significance)	Group B versus Group C (P value with significance)
Baseline	0.63 (NS)	0.30 (NS)	0.63 (NS)
After study drug infusion	<0.001 (HS)	<0.001 (HS)	0.06 (NS)
After induction	0.08 (NS)	0.92 (NS)	0.18 (NS)
After ECT			
0 min	0.001 (HS)	0.280 (NS)	0.000 (HS)
2 min	0.000 (HS)	0.000 (HS)	0.000 (HS)
4 min	0.000 (HS)	0.000 (HS)	0.000 (HS)
6 min	0.000 (HS)	0.000 (HS)	0.000 (HS)
8 min	0.000 (HS)	0.000 (HS)	0.349 (NS)
10 min	0.224 (NS)	1.388 (NS)	0.738 (NS)
15 min	1.383 (NS)	1.383 (NS)	1.383 (NS)
20 min	1.383 (NS)	1.383 (NS)	1.383 (NS)
25 min	1.383 (NS)	1.383 (NS)	1.383 (NS)
30 min	1.383 (NS)	1.383 (NS)	1.383 (NS)

(S – Significant (p<0.05); NS – Nonsignificant (p>0.05); HS – Highly significant (p<0.001). ECT – Electroconvulsive therapy)

Table 5: Showing Postelectroconvulsive therapy changes in MAP in Group A, B, and C at different time interval MAP 3 groups

Time(min)	Group A (mm of Hg)	Group B (mm of Hg)	Group C (mm of Hg)
infusion	93.87 ± 3.27	93.77 ± 6.50	92.73 ± 5.90
induction	89.40 ± 3.28	89.83 ± 6.40	90.53 ± 5.87
Ect	119.40 ± 3.50	92.97 ± 8.33	104.70 ± 6.59
2	117.27 ± 3.62	94.70 ± 7.78	102.73 ± 6.48
4	109.57 ± 3.29	92.43 ± 7.23	97.00 ± 6.22
6	99.47 ± 3.41	91.97 ± 6.59	93.20 ± 6.42
8	93.60 ± 3.21	93.50 ± 5.69	92.93 ± 6.11
10	93.60 ± 3.21	93.17 ± 6.06	92.93 ± 6.11
15	93.60 ± 3.21	93.17 ± 6.06	92.93 ± 6.11
20	93.60 ± 3.21	93.17 ± 6.06	92.93 ± 6.11
25	93.60 ± 3.21	93.17 ± 6.06	92.93 ± 6.11
30	93.60 ± 3.21	93.17 ± 6.06	92.93 ± 6.11

Table 6: Showing comparison of MAP among the 3 groups

Time	Group A versus Group B (P value with significance)	Group A versus Group C (P value with significance)	Group B versus Group C (P value with significance)
After study drug infusion	<0.001 (HS)	<0.001 (HS)	0.06 (NS)
After induction	0.08 (NS)	0.92 (NS)	0.18 (NS)
After ECT			
0 min	0.756	0.417	0.616
2 min	0.000	0.000	0.000
4 min	0.000	0.000	0.000
6 min	0.000	0.000	0.003
8 min	0.000	0.000	0.402
10 min	0.940	0.619	0.672
15 min	0.753	0.628	0.865
20 min	0.753	0.628	0.865
25 min	0.753	0.628	0.865
30 min	0.753	0.628	0.865

(S – Significant (p<0.05); NS – Nonsignificant (p>0.05); HS – Highly significant (P<0.001). ECT – Electroconvulsive therapy)

MAP at 2, 4 and 6 minutes only. There was no significant difference between among the 3 groups after 8 minutes.

Discussion

ECT induces generalized tonic–clonic epileptic seizure. The patients undergoing for ECT might be on a number of anti psychotic medications which can cause exaggerated cardiovascular responses. In our study, postECT hyper dynamic responses were significantly less in the dexmedetomidine group at 0, 2, 4, 6, and 8 min as compared with Group A and Group C. similar observations were noted by Shams and ElMasry[7] and Begec *et al*.[8]. Although, Fu and White[9] have found that dexmedetomidine extended the seizure activity duration during ECT, Shams and ElMasry,[7] Mizrak *et al*.,[10] Cohen and Stewart,[11] and Dodawad[12] have found that there was no significant differences in the duration of seizure activity when using dexmedetomidine with the control population, Similar findings were noted in our study. Saito *et al*.,[13] Howie *et al*.[14] and Weinger *et al*.[15] found no significant differences in the duration of seizures, when comparing esmolol versus control group before ECT which was similar in our group. Motor seizure duration and recovery from anesthesia were similar in all the groups. In our study we observed that dexmedetomidine reduced emergence agitation following recovery from anesthesia for ECT, in contrast the recovery was smooth.

The duration of Motor seizure activity was not effected and recovery was not delayed. There were no side effects such as headache, respiratory depression, hypoxemia, bradycardia, hypotension, jaw pain, and muscle spasms. Esmolol has a very fast onset of action (2 min) while dexmedetomidine has a little delayed onset of action and has to be given by infusion. To make double blinding possible, we have given esmolol also in same fashion as dexmedetomidine, but this may reduce the efficacy of esmolol as it is rapidly eliminated from the body. To avoid this bias and rather than making esmolol less effective, we have changed the anaesthesiologist, after giving esmolol in bolus form and dexmedetomidine in an infusion, which is a preferred technique.[16] As ECT procedures are performed frequently in the outpatient setting, the anaesthetic agents used for these procedures should have rapid recovery profiles. In the our study, none of these two drugs (esmolol and dexmedetomidine) prolong the recovery times. These drugs may be superior to other drugs for ECT because of their short halflife and wide therapeutic indices. However, the implications of these findings require further investigation.

Conclusion

Both esmolol and dexmedetomidine attenuate the hyper dynamic response to ECT without affecting the seizure duration, but dexmedetomidine has a more favourable response in view of stable vitals, smooth emergence, and no adverse effect on recovery duration.

Limitations

The monitoring of seizure duration by observing tonic–clonic activity and not using electroencephalogram (EEG) was a limitation of our study because EEG seizure duration activity may be longer than motor seizure activity.

Financial Support and Sponsorship: Nil.

Conflicts of Interest: There are no conflicts of interest.

References

1. Ding Z, White PF. Anesthesia for electroconvulsive therapy. Anesth Analg. 2002;94:135164.
2. Pal R, Pal VS. Anesthesia for electroconvulsive therapy: A noble approach. Int J Med Res Health Sci. 2015;4:8905.
3. Locala JA, Irefin SA, Malone D, Cywinski JB, Samuel SW, Naugle R. The comparative hemodynamic effects of methohexital and remifentanil in electroconvulsive therapy. J ECT.
4. 2005;21:125.
5. Kumar A, Sharma DK, Mani R. A comparison of propofol and thiopentone for electroconvulsive therapy. J Anaesthesiol Clin Pharmacol. 2012;28:3537.
6. Van den Broek WW, Groenland TH, Mulder PG, *et al.* Betablockers and electroconvulsive therapy: A review. Tijdschr Psychiatr 2008;50:20515.
7. Boere E, Birkenhäger TK, Groenland TH, *et al.* Betablocking agents during electroconvulsive therapy: A review. Br J Anaesth. 2014;113:4351.
8. Shams T, ElMasry R. Ketofol dexmedetomidine combination in ECT: A punch for depression and agitation. Indian J Anaesth. 2014;58:27580.
9. Begec Z, Toprak HI, Demirbilek S, *et al.* Dexmedetomidine blunts acute hyperdynamic

responses to electroconvulsive therapy without altering seizure duration. Acta Anaesthesiol Scand. 2008;52:3026.

10. Fu W, White PF. Dexmedetomidine failed to block the acute hyperdynamic response to electroconvulsive therapy. Anesthesiology. 1999;90:4224.
11. Mizrak A, Koruk S, Ganidagli S, *et al.* Premedication with dexmedetomidine and midazolam attenuates agitation after electroconvulsive therapy. J Anesth. 2009;23:610.
12. Cohen MB, Stewart JT. Treatment of postelectroconvulsive therapy agitation with dexmedetomidine. J ECT. 2013;29:e234.
13. Dodawad R, Sumalatha GB, Sandeep P, *et al.* Dexmedetomidine at a dose of 0.6microgram/kg in attenuation of hemodynamic stress response of electroconvulsive therapy. J Evid Based Med Health. 2015;2:47718.
14. Saito S, Nishihara F, Akihiro T, *et al.* Landiolol and esmolol prevent tachycardia without altering cerebral bloodflow. Can J Anaesth. 2005;52:102734.
15. Howie MB, Hiestand DC, Zvara DA, Kim PY, McSweeney TD, Coffman JA. Defining the dose range for esmolol used in electroconvulsive therapy hemodynamic attenuation. Anesth Analg. 1992;75:80510.
16. Weinger MB, Partridge BL, Hauger R, *et al.* Prevention of the cardiovascular and neuroendocrine response to electroconvulsive therapy: I. Effectiveness of pretreatment regimens on hemodynamics. Anesth Analg. 1991;73:55662.
17. Moshiri E, Modir H, Bagheri N, *et al.* Premedication effect of dexmedetomidine and alfentanil on seizure time, recovery duration, and hemodynamic responses in electroconvulsive therapy. Ann Card Anaesth. 2016;19:2638.