Original Article

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Anaesthesia Section

Efficacy of Intravenous Dexmedetomidine in Attenuating Haemodynamic Response and Postictal Agitation in Patients undergoing modified Electroconvulsive Therapy (m-ECT): A Randomised Controlled Trial

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ABSTRACT

Introduction: Electroconvulsive Therapy (ECT) is an established and effective treatment for severe psychiatric disorders, including major depressive disorder and bipolar disorder. However, ECT is often associated with significant haemodynamic fluctuations and Postictal Agitation (PIA), which may pose safety concerns. Dexmedetomidine, a selective alpha-2 adrenergic agonist, has shown promise in stabilising haemodynamics and reducing PIA in various clinical settings.

Aim: To evaluate the efficacy of intravenous dexmedetomidine in attenuating haemodynamic responses and PIA in patients undergoing modified ECT (m-ECT).

Materials and Methods: This double-blinded, randomised controlled study was conducted at Pt. B.D. Sharma PGIMS, Rohtak, Haryana, India, from July 2023 to September 2024, and included 100 patients undergoing m-ECT. Patients were randomly allocated to receive either intravenous 0.9% normal saline (Group C) or intravenous dexmedetomidine at a dose of 1 µg/kg (Group D), administered over 10 minutes prior to m-ECT. Haemodynamic parameters, including Heart Rate (HR), Mean Arterial Pressure (MAP), and Rate Pressure Product (RPP), were measured at multiple intervals. PIA was assessed using the emergence agitation score. Additional parameters recorded included seizure duration, time to spontaneous breathing, and time to obey verbal commands. Data were analysed using Statistical Package for the Social Sciences (SPSS) software version 26.0. The Independent t-test and Chi-square test were applied where appropriate, with a p-value <0.05 considered statistically significant.

Results: Demographic characteristics, including age, gender distribution, and American Society of Anaesthesiologists (ASA) physical status, were comparable between both groups, with no statistically significant differences. The mean age in Group C was 36.64±11.71 years, and in Group D was 38.28±12.10 years (p-value=0.467). The mean peak HR was significantly lower in Group D (97.46±9.50 bpm) compared to Group C (124.10±20.54 bpm). MAP was also significantly lower in Group D (94.6±12.2 mmHg) than in Group C (123.96±15.94 mmHg). The RPP was reduced in Group D (9154.64±1547.45) compared to Group C (10113.84±1706.19). Overall, Group D demonstrated significantly lower levels of emergence agitation, suggesting that dexmedetomidine was more effective in promoting calm recovery. The statistically significant p-value (0.006) supports this conclusion.

Conclusion: Intravenous dexmedetomidine effectively reduces haemodynamic fluctuations and PIA in patients undergoing modified ECT. It provides better cardiovascular stability, lowers HR and blood pressure, and improves the quality of recovery. Although it slightly prolongs the time to obey verbal commands, the overall benefits make dexmedetomidine a safe and valuable adjunct in the anaesthetic management of m-ECT.

Keywords: Anaesthesia, Haemodynamic parameters, Sedation

INTRODUCTION

The ECT has been a pivotal treatment for severe psychiatric disorders since its inception in the 1930s [1]. Initially met with skepticism, ECT has evolved into a well-validated intervention, particularly for conditions such as major depressive disorder, bipolar disorder, schizophrenia, and catatonia, which often resist conventional pharmacotherapy [2]. The procedure involves delivering a controlled electrical current to the brain to induce a seizure, which can provide rapid relief from debilitating psychiatric symptoms—especially in acute situations where immediate intervention is crucial [3].

The advent of modified ECT (m-ECT), performed under general anaesthesia, has significantly enhanced patient safety and comfort [4]. Despite its proven efficacy, ECT is associated with notable physiological responses that may pose risks to patients. These responses are characterised by marked haemodynamic fluctuations, involving an initial parasympathetic response followed by a sustained sympathetic response [5]. The initial phase can cause bradycardia and hypotension, which are particularly concerning in patients with pre-existing cardiovascular conditions [6]. The subsequent sympathetic phase may result in significant increases in HR and blood pressure, predisposing patients to cardiovascular complications [7].

Another important concern following ECT is PIA a transient state of confusion and agitation affecting approximately 12% of patients [8]. PIA can complicate recovery and may necessitate additional interventions, raising safety concerns in the post-anaesthetic care setting. To address these issues, pharmacological agents such as dexmedetomidine have been investigated for their potential to mitigate the adverse effects associated with ECT.

Dexmedetomidine is a highly selective alpha-2 adrenergic agonist known for its sedative, anxiolytic, and sympatholytic properties, making it particularly effective in maintaining haemodynamic stability during the procedures [9]. This study aimed to evaluate the efficacy of intravenous dexmedetomidine in attenuating haemodynamic responses and PIA in patients undergoing modified ECT.

The primary outcomes of the study were HR, MAP, RPP, and PIA (assessed using the emergence agitation score). The secondary outcomes included seizure duration, time to spontaneous breathing, and time to obey verbal commands. Additional observations involved monitoring for potential side-effects such as bradycardia, hypotension, nausea, and respiratory depression.

MATERIALS AND METHODS

This double-blind, randomised controlled trial was conducted in the Department of Anaesthesiology and Critical Care, Pt. B.D. Sharma PGIMS, Rohtak, Haryana, India over a period of 15 months, from July 2023 to September 2024. The study was approved by the Institutional Ethics Committee (Approval No.: EC/NEW/INST/2022/HR/0189) and registered with the Clinical Trial Registry of India (CTRI/2024/05/067681). Written informed consent was obtained from all participants. Patients were selected based on their clinical diagnosis and the requirement of ECT as part of their treatment plan.

Inclusion criteria:

- Patients aged between 18 and 60 years.
- Diagnosed with mood disorders requiring m-ECT.
- Classified as ASA physical status I or II.
- Provided written informed consent (or consent from a legal guardian).

Exclusion criteria:

- Refusal to provide consent by the patient or relatives.
- History of underlying renal, hepatic, cardiac, or pulmonary disease.
- History of head trauma.
- Patients who did not experience seizures following m-ECT.

Sample size calculation: The sample size was calculated based on a previous study [9] that demonstrated a significant effect of dexmedetomidine in attenuating haemodynamic responses during ECT. Assuming an alpha error of 0.05, a power of 80%, and the ability to detect a clinically significant difference in HR between groups, it was estimated that 50 patients per group would provide adequate statistical power to detect meaningful differences. Therefore, a total of 100 patients were included in the study. No patients were excluded after screening.

Study Procedure

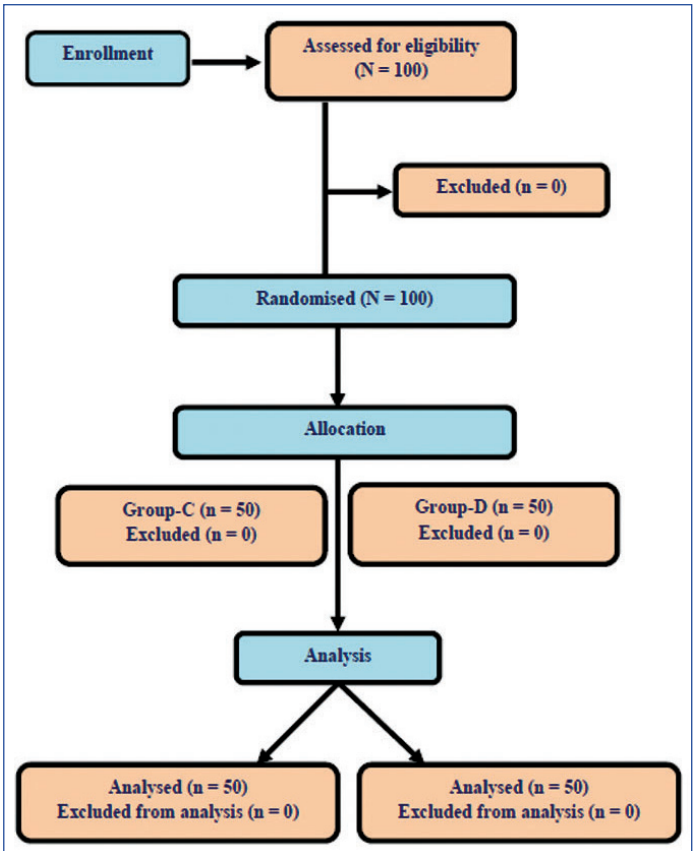
Upon arrival in the ECT suite, all patients underwent standard monitoring, including pulse oximetry, Electrocardiography (ECG), and Non Invasive Blood Pressure (NIBP) measurements. A 20-gauge Peripheral Intravenous (IV) cannula was inserted into a dorsal hand vein for drug administration. Baseline haemodynamic parameters—HR, Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), and MAP—were recorded before any intervention.

Patients were randomly assigned to one of two groups using a sealed envelope technique based on a computer-generated randomisation list [Table/Fig-1]:

Group C (Control Group): Received 0.9% normal saline intravenously over 10 minutes prior to m-ECT.

Group D (Dexmedetomidine Group): Received intravenous dexmedetomidine at a dose of 1 µg/kg body weight, diluted in 10 mL of normal saline, administered slowly over 10 minutes prior to m-ECT.

The random allocation sequence was generated using a computer-based randomisation table by an independent statistician not involved in patient enrolment or outcome assessment. Patient enrolment was performed by a designated anaesthesiologist who was not involved in group allocation or data analysis. Group assignments were concealed using Sequentially Numbered, Opaque, Sealed Envelopes (SNOSE) to ensure allocation concealment.



[Table/Fig-1]: A Consolidated Standards of Reporting Trials (CONSORT) diagram that visually represents the progress of participants through each stage.

This study followed a double-blind design: both the patients and the investigators responsible for monitoring and recording outcomes were blinded to group assignments. The anaesthesiologist administering the study drug was not involved in post-procedure assessments, thereby minimising observer and performance bias.

Intravenous dexmedetomidine was administered at a dose of 1 µg/kg over 10 minutes, as supported by previous literature demonstrating its efficacy in attenuating haemodynamic responses during ECT without significantly affecting seizure duration [9]. Following drug administration, standard anaesthesia protocols were followed. Patients in both groups were premedicated with atropine, induced with thiopental, and given muscle relaxation with succinylcholine. The m-ECT procedure was performed using a constant-current ECT device, and the electrical stimulus parameters were determined by the attending psychiatrist.

Data collection: Following ECT, various parameters were monitored and recorded. Haemodynamic parameters (HR, SBP, DBP, and MAP) were measured at baseline and at 1, 3, 5, 10, and 20 minutes post-ECT. The duration of the motor seizure was recorded in seconds. The time to spontaneous breathing was measured from the end of muscle relaxant administration until the resumption of spontaneous respiration. The time to obey verbal commands was measured from the end of muscle relaxant administration until the patient responded to verbal commands. The level of agitation was assessed using the Emergence Agitation Score at 30 minutes post-m-ECT. In addition to evaluating PIA, the study monitored potential side-effects, including bradycardia, hypotension, nausea, and respiratory depression.

STATISTICAL ANALYSIS

Data were analysed using SPSS software version 26.0. Continuous variables were compared using the Independent t-test, while categorical variables were analysed using the Chi-square test. A p-value <0.05 was considered statistically significant.

RESULTS

The demographic profiles of both groups were comparable. There were no statistically significant differences between the groups in terms of age, body weight, or ASA physical status. Patients were similarly distributed across both groups, with a slightly higher proportion of ASA Grade I patients in the dexmedetomidine group. This ensured that both groups were well matched at baseline, allowing for an unbiased comparison of clinical outcomes [Table/Fig-2,3].

Parameters	Mean±SD		p-value
	Group C	Group D	
Age (years)	36.64±11.71	38.38±12.1	0.467
Weight (Kg)	67.66±7.92	66.14±10.43	0.414

[Table/Fig-2]: Comparison of mean age and weight in the two groups (N=100).

Mallampati grade	Group C, n (%)	Group D, n (%)	p-value
Grade 1	5 (10)	9 (18)	0.249
Grade 2	32 (64)	22 (44)	
Grade 3	9 (18)	13 (26)	
Grade 4	4 (8)	6 (12)	

[Table/Fig-3]: Comparison of Mallampati grade in the two groups (N=100).

Haemodynamic parameters: Group D demonstrated significantly lower HR, MAP, and RPP values compared to Group C at all measured intervals, indicating better cardiovascular stability with dexmedetomidine [Table/Fig-4,5].

Haemodynamic variables	Interval	Mean±SD		p-value
		Group C	Group D	
Systolic BP (mm Hg)	Baseline	120.48±12.08	115.8±11.80	0.053
	1 min	141.88±20.73	138±14.19	0.278
	3 mins	143.46±21.5	136.2±15.73	0.057
	5 mins	129.62±15.83	120.26±15.04	0.003
	10 mins	118.02±11.86	112.7±13.03	0.035
	20 mins	115.62±12.27	111.34±11.21	0.072
Diastolic BP (mm Hg)	Baseline	74.44±6.59	71.26±7.77	0.030
	1 min	90.6±16.13	85.48±10.6	0.064
	3 mins	88.88±14.79	83.14±11.96	0.035
	5 mins	79.88±11.89	75.18±9.41	0.031
	10 mins	74.66±9.26	70.52±9.33	0.028
	20 mins	73.7±7.8	69.94±7.42	0.015
Mean Arterial Pressure (MAP) (mm Hg)	Baseline	87.46±7.81	81.2±8.57	<0.001
	1 min	106.84±16.53	96.08±12.12	<0.001
	3 mins	103.28±14.49	92.22±12.64	<0.001
	5 mins	95.42±11.41	84.9±11.45	<0.001
	10 mins	86.56±8.57	79.24±9.91	<0.001
	20 mins	84.4±7.46	78.3±8.66	<0.001
Heart Rate (HR) (bpm)	Baseline	93.44±18.7	81.08±10.04	<0.001
	1 min	116.08±25.86	101.98±16.01	0.001
	3 mins	113.54±20.91	97.98±13.41	<0.001
	5 mins	104.82±16.02	89.5±13.04	<0.001
	10 mins	92.96±13.91	83.36±10.02	<0.001
	20 mins	87.72±11.36	82.08±10.44	0.011
Rate-Pressure Product (RPP)	Baseline	11492.1±2821.04	9417.72±1683.2	<0.001
	1 min	16547.56±4744.13	14259.12±3298.68	0.006
	3 mins	16416.98±4573.1	13399.86±3021.65	<0.001
	5 mins	13559.5±2768.97	10820.86±2521.6)	<0.001
	10 mins	10983.6±2131.43	9382.4±1685.31	<0.001
	20 mins	10113.84±1706.19	9154.64±1547.45)	0.004

[Table/Fig-4]: Comparison of mean haemodynamic parameters in the two groups (N=100).

Parameter	Group C (Mean±SD)	Group D (Mean±SD)	p-value
Heart Rate (HR) (bpm)	124.1±20.54	97.46±9.5	<0.001
Mean Arterial Pressure (MAP) (mmHg)	123.96±15.94	94.6±12.2	<0.001
Rate-Pressure Product (RPP)	10113.84±1706.19	9154.64±1547.45	<0.001

[Table/Fig-5]: Comparison of overall mean haemodynamic parameters in the two groups (N=100).

Duration of motor seizures: There was no statistically significant difference in seizure duration between the two groups, confirming that dexmedetomidine did not interfere with seizure quality or duration [Table/Fig-6].

Parameters	Group C	Group D	p-value
Duration of motor seizure (seconds)	49.68±18.92	52.72±15.96	0.387
Time to spontaneous breathing (seconds)	184.62±77.73	211.5±76.09	0.084
Time to obey verbal commands (seconds)	331.4±151.25	389.56±84.3	0.020

[Table/Fig-6]: Duration of motor seizures and recovery parameters.

Although Group D showed a slightly longer time to spontaneous breathing compared to Group C, the difference was not statistically significant and had no clinical implications. Group D exhibited a significantly longer time to obey verbal commands than Group C, suggesting a mild prolongation in recovery due to the sedative effect of dexmedetomidine [Table/Fig-6].

Agitation score: The incidence of emergence agitation was significantly lower in Group D compared to Group C, indicating that dexmedetomidine effectively reduced PIA [Table/Fig-7].

Agitation score	Group C, n (%)	Group D, n (%)	p-value
Sleeping	9 (18)	14 (28)	0.006
Awake and calm	32 (64)	36 (72)	
Irritable and crying	9 (18)	0	

[Table/Fig-7]: Distribution of cases according to emergence score in two groups (N=100).

No significant side-effects were observed in either group. While a few patients in the dexmedetomidine group experienced mild hypotension and bradycardia, these episodes were transient, did not require intervention, and were clinically insignificant.

DISCUSSION

This randomised, double-blind trial evaluated the efficacy of intravenous dexmedetomidine in attenuating haemodynamic responses and PIA in patients undergoing m-ECT.

Patient characteristics and group comparison: The demographic characteristics, including age and gender, were comparable between the two groups, with no statistically significant differences. However, a higher proportion of ASA Grade I patients in Group D may have contributed to improved haemodynamic stability. This aligns with previous protocols that identified ASA grading as a potential confounding variable when assessing anaesthetic drug efficacy during ECT [9].

Haemodynamic effects: Present study findings demonstrated a significant reduction in peak HR, MAP, and RPP in the dexmedetomidine group, indicating a clear sympatholytic effect. These results are consistent with the findings of Bagle AA et al., and Begec Z et al., who reported that dexmedetomidine effectively blunts hyperdynamic cardiovascular responses following ECT [9,10]. Heidarbeigi K et al., also confirmed the stabilising effect of dexmedetomidine when used with thiopental [11].

In contrast, Fu W and White PF, observed no significant haemodynamic benefit, suggesting that responses may vary depending on patient co-morbidities or concomitant anaesthetic agents. More recent studies further support present study results [12]. Sriramka B et al., demonstrated improved cardiovascular stability and reduced succinylcholine-induced myalgia [13], while Subsoontorn P et al., reported significant reductions in SBP and HR during ECT, confirming dexmedetomidine's reliable autonomic modulation [14]. Espinoza RT and Kellner CH, also emphasised the importance of managing cardiovascular stress as a key safety goal in modern ECT—an outcome supported by present study [15].

Seizure duration and therapeutic efficacy: There was no statistically significant difference in seizure duration between the two groups, confirming that dexmedetomidine does not suppress seizure threshold. This finding was consistent with the observations of Begec Z et al., and Qiu Y et al., who also reported preserved seizure durations [10,16]. Present study results align with those of Li X et al., whose meta-analysis concluded that dexmedetomidine does not adversely affect seizure efficacy, even when combined with other anaesthetics [17]. Similarly, Kumar R et al., found adequate seizure quality despite dexmedetomidine use [18].

Recovery metrics: While the time to spontaneous breathing was slightly prolonged in Group D, the difference was not statistically significant, which aligns with the studies of Bagle AA et al., and Qiu Y et al., [9,16]. However, the time to obey verbal commands was significantly longer in Group D, a finding supported by Li X et al., who attributed the delay to dexmedetomidine's sedative properties [17]. Kumar R et al., and Cohen MB and Stewart JT, reported similar outcomes, noting mild but acceptable delays in emergence without major safety concerns [18,19].

PIA and emergence behaviour: A key finding of present study was the significant reduction in PIA in the dexmedetomidine group. This supports previous reports by Narang P et al., and Mizrak A et al., both of whom demonstrated effective agitation control with dexmedetomidine [7,8]. Rehm J and Moliner M, in their 2023 meta-analysis, confirmed dexmedetomidine's superiority in preventing PIA compared to other agents [20]. Tzabazis A et al., further noted that alternatives such as propofol were less effective, often requiring adjunctive measures [21].

Mechanism of action and safety profile: Dexmedetomidine, an alpha-2 adrenergic agonist, provides sedation, anxiolysis, and sympatholysis without causing respiratory depression [6]. This pharmacological profile contributes to the balanced emergence observed in present study. No serious adverse effects were noted; only mild, transient hypotension and bradycardia occurred in Group D—findings consistent with those of Heidarbeigi K et al., and Sriramka B et al., [11,13].

Limitation(s)

Several limitations were identified in this study. It did not compare low-dose dexmedetomidine with other agents such as short-acting opioids or beta-blockers, which could also influence haemodynamic responses. Only motor seizure duration was recorded; EEG seizure duration was not assessed, which might have provided a more comprehensive evaluation of dexmedetomidine's effects. Additionally, the study population was highly preselected, with a higher prevalence of intractable psychiatric illnesses compared to the general population. The single-centre design also limits the generalisability of the results, and specific psychiatric diagnoses were not analysed.

CONCLUSION(S)

This study demonstrated that intravenous dexmedetomidine significantly attenuates haemodynamic responses and reduces PIA in patients undergoing m-ECT. Compared to the control group, patients receiving dexmedetomidine exhibited lower HR, MAP, and RPP throughout the periprocedural period, reflecting superior cardiovascular stability. Importantly, dexmedetomidine did not compromise the duration of motor seizures, indicating preserved therapeutic efficacy of ECT. Although recovery parameters such as time to obey verbal commands were slightly prolonged in the dexmedetomidine group, the difference was clinically acceptable and attributable to the drug's sedative properties. The incidence of PIA was markedly reduced, reaffirming dexmedetomidine's efficacy in improving patient emergence behaviour. No serious adverse events were reported, with only transient and self-limiting hypotension and bradycardia observed. Overall, the findings support the use of dexmedetomidine as a valuable anaesthetic adjunct during m-ECT to enhance safety and patient comfort. This study adds to the growing body of evidence favouring dexmedetomidine's role in ECT settings and highlights its potential to optimise perioperative outcomes. Further multicentric research is recommended to validate and generalise these findings across broader patient populations.

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