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Research Article

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Evaluation of the Effects of Low Dose Dexmedetomidine on Hyperdynamic Responses to Electroconvulsive Therapy

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Abstract

Background:

Electroconvulsive therapy (ECT) often causes acute hyperdynamic cardiovascular responses, which may be harmful to patients at risk of cardiac events. Although dexmedetomidine can attenuate these effects, higher doses (0.5–1 mcg/kg) have been linked to delayed anaesthetic recovery.

Objectives:

To evaluate the effects of low-dose dexmedetomidine (0.25 mcg/kg and 0.5 mcg/kg) on hyperdynamic responses to ECT and on recovery from anaesthesia.

Methods:

In a randomized, double-blind study, 75 patients undergoing modified ECT for severe drug-resistant depression were assigned to three groups: D1 (0.25 mcg/kg dexmedetomidine), D2 (0.5 mcg/kg), and D3 (placebo). All patients received standard anaesthetic premedication and induction with propofol, followed by ECT. Outcomes included heart rate (HR), mean arterial pressure (MAP), ST-segment changes, heart rate variability, recovery time, and post-ECT agitation scores.

Results:

HR and MAP were significantly lower in dexmedetomidine groups (D1 and D2) compared to placebo, with the greatest reductions in group D2. ST-segment changes occurred only in the placebo group. Recovery was fastest in the placebo group and slowest in group D2 (mean recovery times: D1 = 9.2 min, D2 = 11.3 min, D3 = 7.2 min). Agitation scores were similar across groups.

Conclusion:

Low-dose dexmedetomidine (0.25 mcg/kg) effectively attenuates the cardiovascular responses to ECT while enabling faster recovery compared to higher doses. It may be a safer alternative for patients requiring modified ECT.

Keywords: Modified ECT, Dexmedetomidine, Premedication, Anaesthesia Recovery, Propofol

1. Introduction

Electroconvulsive therapy (ECT) remains a widely utilized and highly effective treatment modality for severe and treatment-resistant psychiatric disorders, particularly major depressive disorder, bipolar disorder, and schizophrenia [1]. Despite its clinical efficacy, ECT is often associated with transient but significant cardiovascular complications, collectively referred to as hyperdynamic responses, which include abrupt increases in heart rate and blood pressure due to autonomic nervous system stimulation [2]. These responses may pose a substantial risk, particularly for patients with underlying cardiovascular conditions.

To mitigate these adverse effects, various pharmacologic agents have been studied, including beta-blockers, opioids, and alpha-2

adrenergic agonists. Dexmedetomidine, a highly selective alpha-2 adrenoceptor agonist, has garnered attention due to its sympatholytic, sedative, and analgesic properties, which make it a potential candidate for modulating the hemodynamic effects during ECT without significantly affecting seizure duration or quality [3].

Low-dose dexmedetomidine administration prior to ECT has been proposed as a strategy to attenuate hyperdynamic responses while preserving the therapeutic efficacy of the procedure. However, evidence on its safety and effectiveness remains varied, necessitating further evaluation. This study seeks to assess the effects of low-dose dexmedetomidine on the hyperdynamic cardiovascular responses associated with ECT, with the aim of improving peri-procedural safety and patient outcomes.

Electroconvulsive therapy, though effective, induces a biphasic autonomic response, characterized by initial parasympathetic stimulation followed by a robust sympathetic surge, leading to tachycardia and hypertension [1,4]. The hyperdynamic state is particularly concerning in elderly or cardiovascularly compromised patients, and as such, strategies to blunt this response are clinically important.

Dexmedetomidine, by virtue of its central sympatholytic action, has been shown to effectively reduce heart rate and blood pressure in various perioperative settings [5]. Several studies have evaluated its use in ECT. For instance, Shukla et al. demonstrated that premedication with dexmedetomidine significantly blunted the hemodynamic response without significantly altering seizure duration [3]. Similarly, Hooten et al. found that dexmedetomidine provided hemodynamic stability and decreased recovery agitation in ECT patients [6].

However, concerns have been raised about the potential of dexmedetomidine to reduce seizure duration or increase the seizure threshold, potentially compromising ECT efficacy [7]. This has led researchers to focus on optimizing the dose. Low doses (e.g., 0.25–0.5 µg/kg) appear to balance efficacy and safety, with minimal effects on seizure parameters while still attenuating the sympathetic response [8].

Comparative studies have also examined dexmedetomidine alongside agents like esmolol, fentanyl, and labetalol. In general, dexmedetomidine provides a more favorable profile due to its sedative properties and reduced likelihood of causing respiratory depression [9]. Nonetheless, variability in outcomes across populations, dosing strategies, and ECT protocols highlights the need for more targeted studies.

2. Methods

2.1. Study Area and Design

This prospective randomized double-blind controlled study was conducted over six months (September 2023 to February 2024) at the Department of Psychiatry, University College Hospital (UCH), and New World Specialist Hospital and Rehabilitation Centre, both in Ibadan, Nigeria. These centers conduct approximately 70–100 electroconvulsive therapy (ECT) sessions annually.

2.2. Study Population and Sample Size

Patients aged 18–65 years with ICD-10 diagnosed severe or acute medication-resistant depressive disorders scheduled for modified ECT were recruited. A total sample size of 75 (25 per group) was calculated using the modified Kirkwood formula, allowing for a 10% attrition rate.

2.3. Randomization and Blinding

Participants were randomized into three groups:

- Group D1: 0.25 mcg/kg dexmedetomidine
- Group D2: 0.5 mcg/kg dexmedetomidine
- Group D3: Placebo(10mlsaline)

Block randomization was performed using computer-generated sequences. The study was double-blinded; the pharmacist prepared the study drugs in identical syringes, and both anaesthetists and patients were unaware of group allocations.

2.4. Inclusion and Exclusion Criteria

Included were adult patients (18–65 years), ASA I–II, who consented and met ICD-10 criteria for severe depression. Exclusion criteria included other Axis I psychiatric diagnoses, organic brain disease, cardiovascular or ocular disorders, and known allergies to study drugs.

2.5. Ethical Approval and Consent

Ethical approval was obtained from UCH/University of Ibadan Ethics Committee and New World Hospital Ethics Committee. Written informed consent was obtained from all participants or their next of kin.

2.6. Procedure

Standard pre-anaesthetic evaluations were conducted. Participants fasted for 6 hours for solids and 2 hours for clear fluids. On ECT day, patients received dexamethasone and glycopyrrolate, followed by the study drug via syringe pump (2 ml/min). Anaesthesia was induced with propofol (1 mg/kg), with additional boluses as needed. Suxamethonium (0.75 mg/kg) was administered for muscle relaxation. ECT was delivered using a bitemporal approach with 120 mC current.

Motor seizure duration was measured using the tourniquet cuff method. ECG and heart rate variability (HRV) were recorded pre- and post-ECT using the Contec 8000G ECG system. Recovery time was noted as time to spontaneous respiration, eye opening, and obeying commands. Patients were monitored post-procedure for vital signs, recovery, and agitation using a standardized agitation scale.

2.7. Outcome Measures

- **Primary:** Attenuation of post-ECT hyperdynamic response (HR

and MAP changes).

• **Secondary:** ST-segment changes, HRV, anaesthetic recovery time, and incidence of post-ECT agitation.

2.8. Data Collection and Analysis

Data on demographics, clinical parameters, haemodynamics, ECG, recovery time, and complications were collected using a standardized proforma. HRV indices (RMSSD, pNN50) were assessed by a blinded cardiologist. Data were analyzed using IBM SPSS v26. Categorical variables were expressed as frequencies and percentages; continuous variables as means ± SD. ANOVA and chi-square tests were used for group comparisons.

3. Results

A total of seventy-five patients met the eligibility criteria and were recruited over a six-month period (September 2023 to February 2024). They were randomly assigned to three groups: D1, D2, and D3. Group D1 received 0.25 mcg/kg dexmedetomidine, group D2 received 0.5 mcg/kg dexmedetomidine, and group D3 (placebo group) received normal saline. All participants completed the study.

3.1. Demographic Profile

All three groups included patients diagnosed with severe depression and were comparable in terms of age, weight, height, BMI, gender, and ASA Physical Status. The mean ages were 29.4±5.0 (D1), 31.1±8.3 (D2), and 31.7±7.1 years (D3), with no significant difference (p = 0.095) [Table I]. The study population comprised 41 males (54.75%) and 34 females (45.35%), with no significant gender difference among the groups (p = 0.686). The mean weights were 59.6±7.9 kg (D1), 63.9±11 kg (D2), and 61.9±6.7 kg (D3), (p = 0.246). The mean heights were 1.6±0.13 m (D1), 1.6±0.12 m (D2), and 1.6±0.08 m (D3), (p = 0.291). The mean BMIs were 23±2.7 (D1), 24.2±2.8 (D2), and 24.6±2.9 (D3), (p = 0.117). ASA classification showed that 80% were ASA I and 20% were ASA II, with no significant differences among groups.

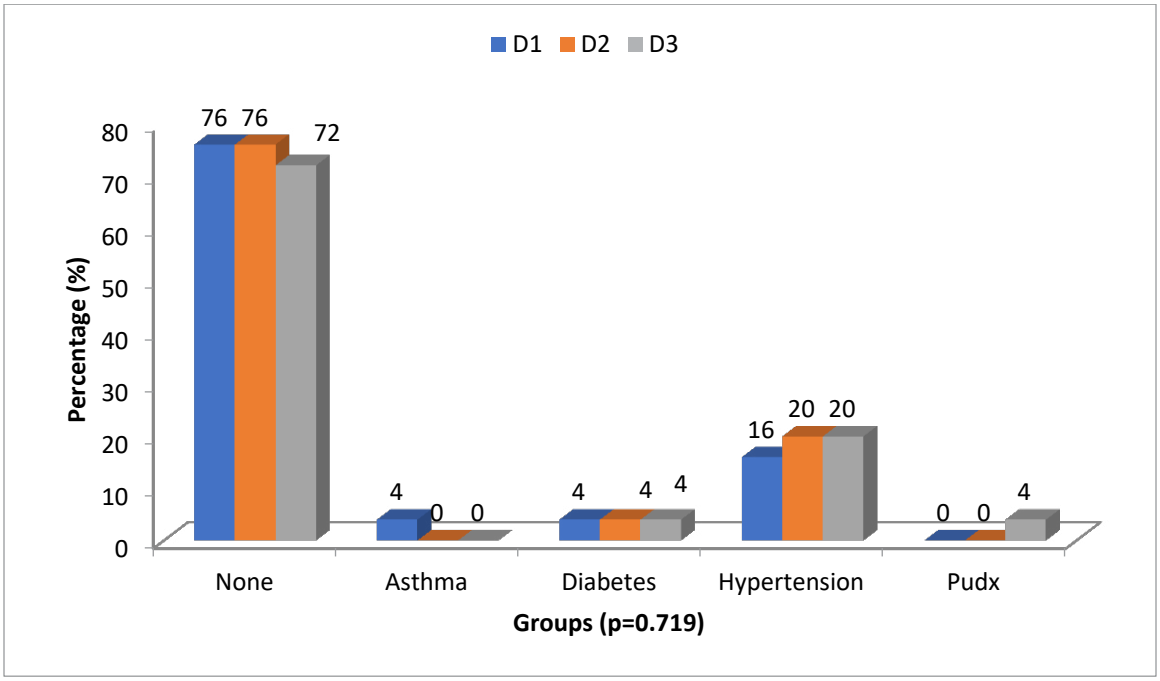
Variables	Statistics	D1 (N =25)	D2 (N =25)	D3 (N=25)	Total (N=75)	f-test/χ^2	p-value
Age (years)	Mean ±SD	29.4±5.0	31.1±8.3	31.7±7.1	31.4±7.1	2.435	0.095
	Range	22.0-42.0	22.0-60.0	18.0-46.0	18.0-60.0		
Weight (Kg)	Mean ±SD	59.6±7.9	63.9±11	61.9±6.7	61.8±8.9	1.429	0.246
	Range	44.0-75.0	43.0-85.0	54.0-81.0	43.0-85.0		
Height (Metre)	Mean ±SD	1.6±0.13	1.6±0.12	1.6±0.08	1.6±0.11	1.255	0.291
	Range	1.28-1.85	1.38-1.86	1.45-1.69	1.28-1.86		
BMI (Kg/M^2)	Mean ±SD	23.0±2.7	24.2±2.8	24.6±2.9	23.9±2.8	2.212	0.117
	Range	19.64-29.47	20.22-30.45	19.64-29.47	19.64-30.45		
DOS(seconds)	Mean ±SD	18.2±3.6	18.9±3.6	17.3±4.2	18.1±3.8	1.044	0.357
	Range	13.0-25.0	12.0-25.0	10.0-25.0	10.0-26.0		
Sex N(%)	Male	14(56.0)	15(60.0)	12(48.0)	41(54.75)	0.753	0.686
	Female	11(44.0)	10(40.0)	13(52.0)	34(45.35)		
ASA,N(%)	I	21(84.0)	21(84.0)	18(72.0)	60(80.0)	1.500	0.472
	II	4(16.0)	4(16.0)	7(28.0)	15(20.0)		

DOS Duration of motor seizure

Table I: Comparison of Demographic Profile

3.2. Distribution of Co-Morbidity

There was no significant difference in the prevalence of co-morbidities among the groups (p = 0.719) [Figure 1]. Hypertension was seen in 18.7% of patients (16% in D1, 20% in D2, 20% in D3). Diabetes mellitus was present in 4% of each group. Asthma occurred in 4% of D1 but was absent in D2 and D3. Peptic ulcer disease was present in 4% of D3 only.



Pudx Peptic ulcer disease

Figure 1: Distribution of Comorbidity among the Study Groups

3.3. Duration of Motor Seizure

The mean motor seizure durations were comparable across groups: 18.2±3.6 (D1), 18.9±3.6 (D2), and 17.9±4.2 seconds (D3), with no significant difference (p = 0.357) [Table I].

3.4. Total Dose of Propofol Used for Induction of Anaesthesia

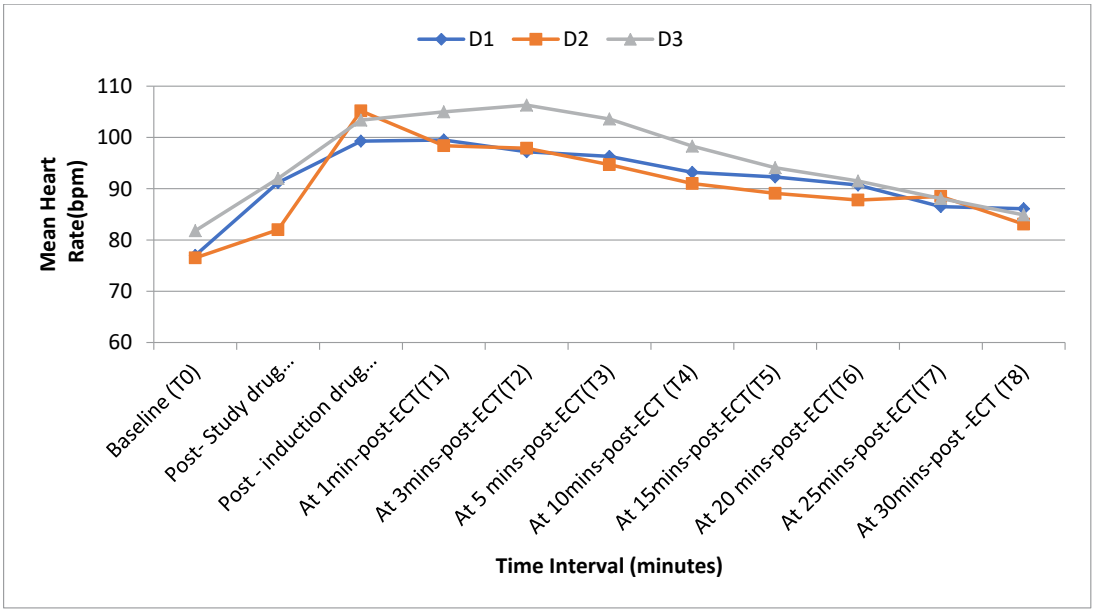
There was a significant difference in the total propofol dose among the groups (p = 0.0001). Group D3 required the highest dose (105.0±16.2 mg), followed by D1 (68.8±12.6 mg), and D2 required the least (64.6±8.3 mg).

3.5. Haemodynamic Changes

3.5.1. Pre-Induction Vital Signs: Baseline vital signs (T0) were similar across all groups. After administration of study drugs, significant group differences were observed in HR, SBP, DBP, and MAP. Group D3 consistently showed the highest values, while D2 had the lowest (except for DBP). Mean HR post-drug: 91.2±13.1 (D1), 82.0±20.7 (D2), 92.0±9.4 bpm (D3) (p = 0.040). SBP: 113.2±13.8 (D1), 108.4±9.4 (D2), 132.8±17.2 mmHg (D3) (p = 0.008). DBP: 67.3±10.3 (D1), 69.4±14.6 (D2), 78.8±14.6 mmHg (D3) (p = 0.040). MAP: 80.6±12.9 (D1), 78.1±7.9 (D2), 91.7±12.5 mmHg (D3) (p = 0.039). No significant differences were observed in RR and SpO2.

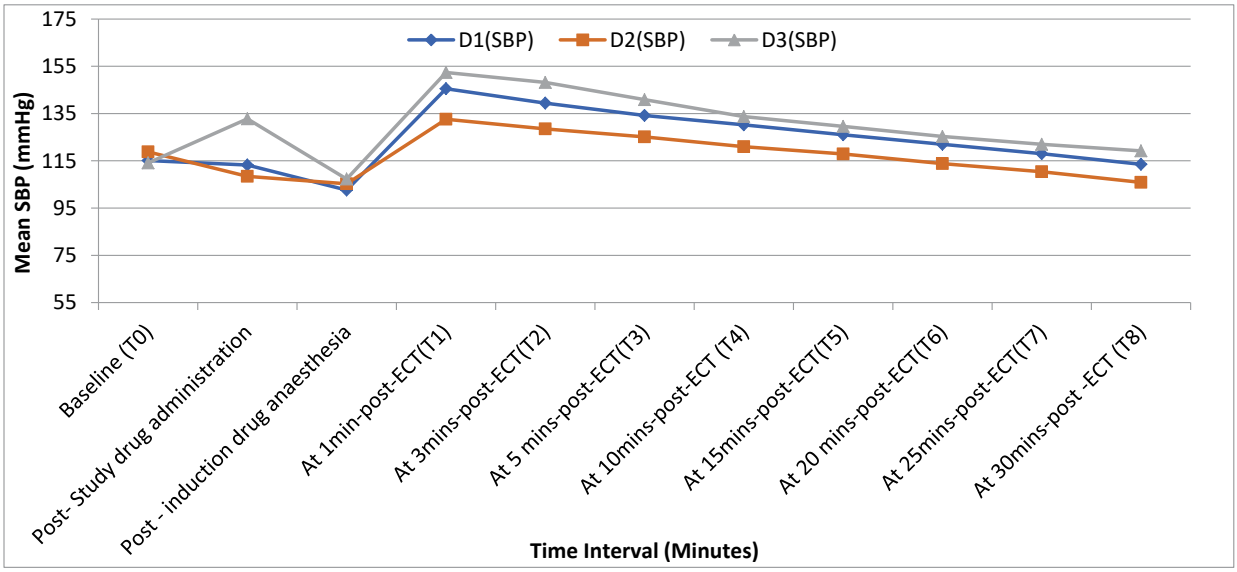
3.5.2. Post-Induction Vital Signs: HR increased post-induction across all groups, while SBP, DBP, and MAP decreased. Only DBP showed a significant between-group difference (p = 0.029). Mean HR: 99.3±11.3 (D1), 105.2±9.4 (D2), 103.4±13.8 bpm (D3) (p = 0.186). MAP: 73.0±8.7 (D1), 73.6±1.7 (D2), 70.6±11.4 mmHg (D3) (p = 0.598). No significant differences were observed in SBP, RR, and SpO2.

3.5.3. Post-ECT Vital Signs: Vital signs post-ECT (T1–T8) are shown in Figures 2–7. HR increased in the first 30 minutes, with significant group differences in the first 10 minutes post-ECT (except T2), with D2 having the lowest and D3 the highest HR. SBP, DBP, and MAP were lowest in D2 and highest in D3. RR and SpO2 were similar across groups.



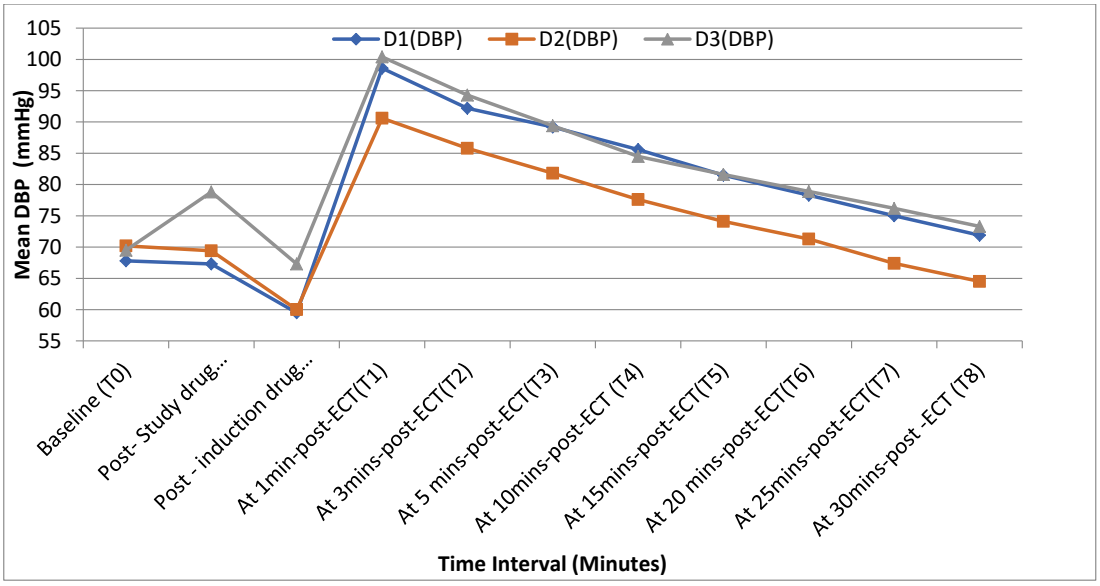
Time	T0	Post-stud.	Post-induct.	T1	T2	T3	T4	T5	T6	T7	T8
p-value	0.242	0.040*	0.186	0.015*	0.049*	0.033*	0.046*	0.135	0.227	0.316	0.333

Figure 2: The Trend in the Mean HR Among the Study Groups



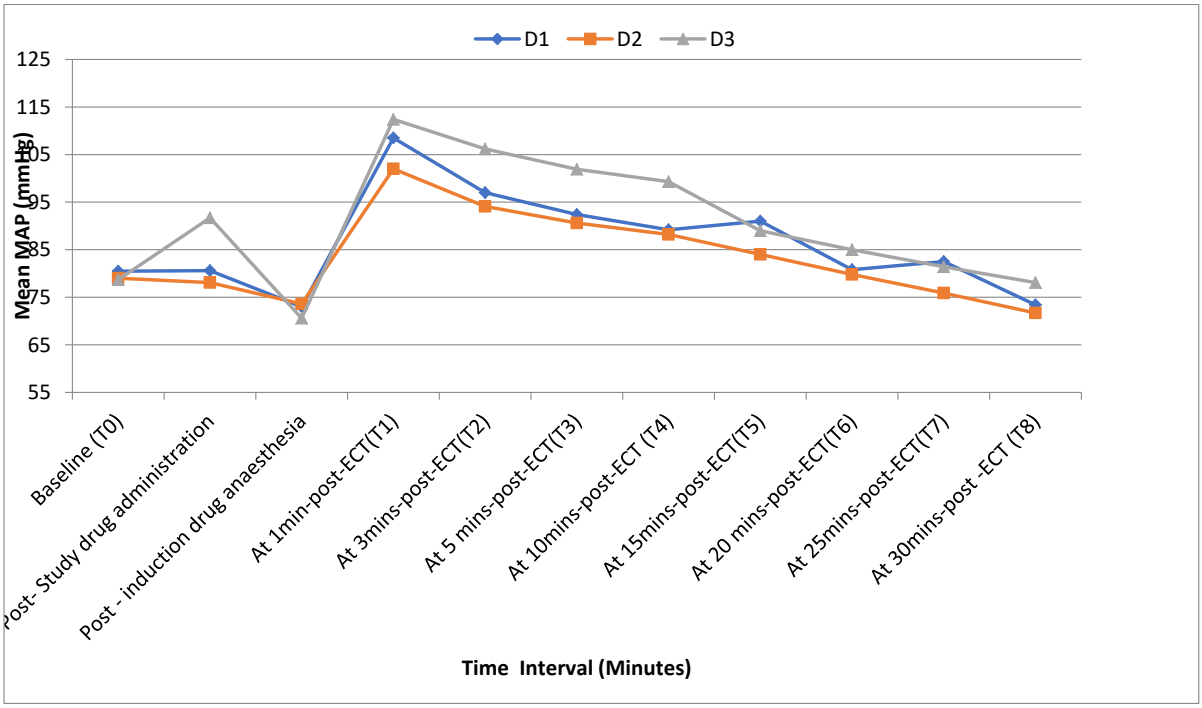
Time	T0	Post stud.	Post-indu.	T1	T2	T3	T4	T5	T6	T7	T8
p-value	0.201	0.008*	0.278	0.0001*	0.0001*	0.0001*	0.0001*	0.0001*	0.0001*	0.0001*	0.0001*

Figure 3: The Trend in the Mean SBP among the Study Groups



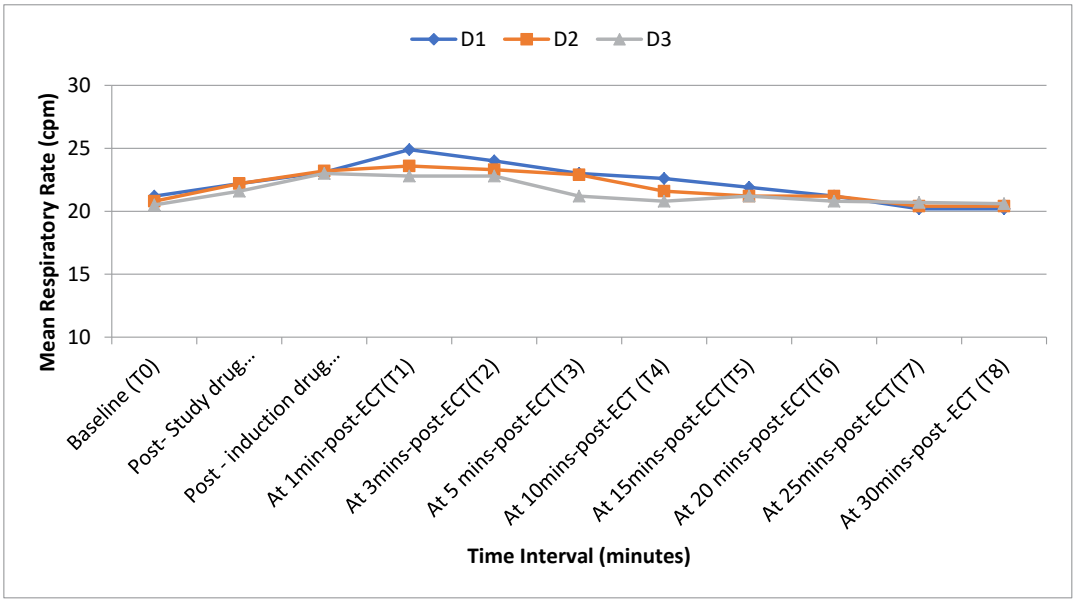
Time	T0	Post stud.	Post induct.	T1	T2	T3	T4	T5	T6	T7	T8
p-value	0.321	0.040*	0.029*	0.006*	0.008*	0.018*	0.006*	0.003*	0.005*	0.001*	0.0001*

Figure 4: The Trend in the Mean DBP among the Study Groups



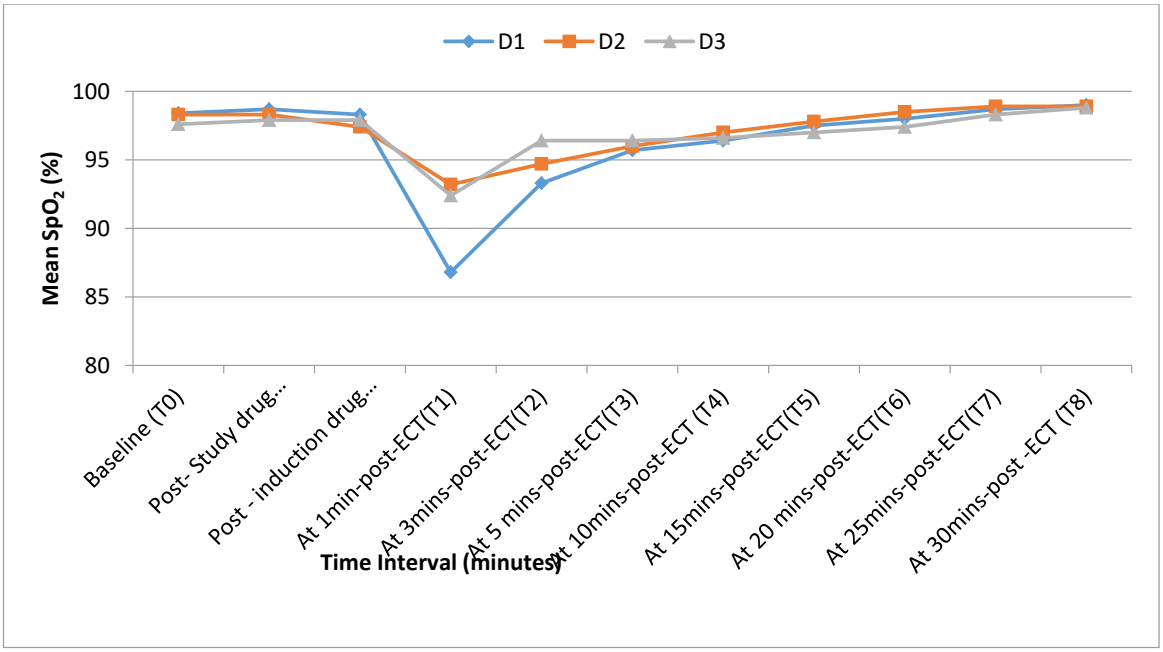
Time	T0	Post study.	Post induct.	T1	T2	T3	T4	T5	T6	T7	T8
p-value	0.121	0.039*	0.598	0.038*	0.048*	0.043*	0.045*	0.042*	0.040*	0.032*	0.019*

Figure 5: The Trend in the Mean MAP among the Study Groups



Time	T0	Post stud.	Post induct.	T1	T2	T3	T4	T5	T6	T7	T8
p-value	0.107	0.536	0.904	0.300	0.117	0.044*	0.080	0.147	0.573	0.290	0.597

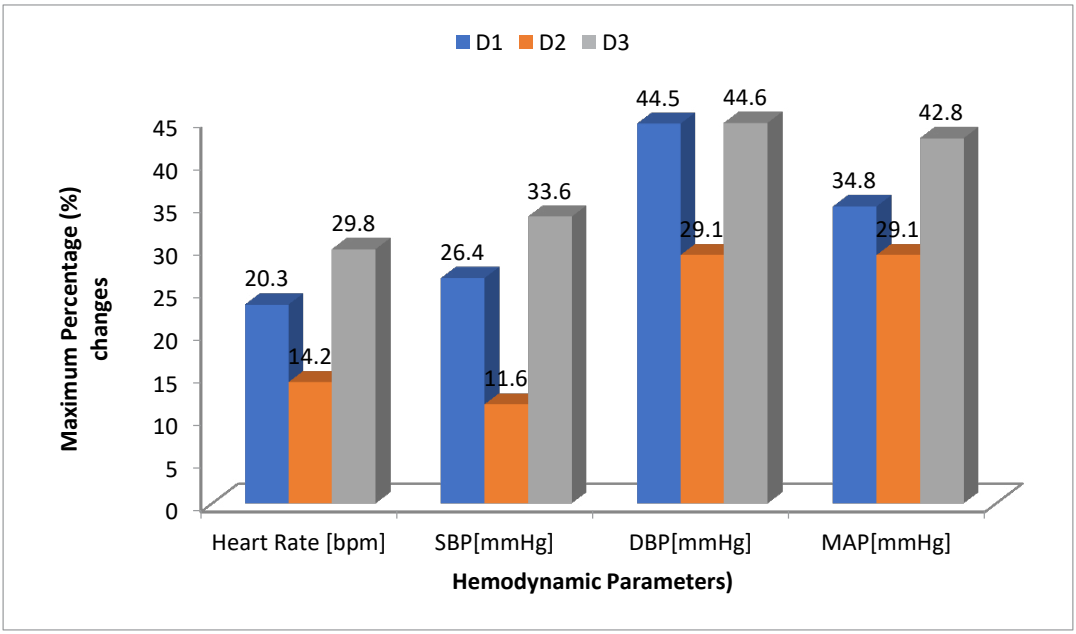
Figure 6: The Trend in the Mean RR among the Groups



Time	T0	Post stud.	Post induct.	T1	T2	T3	T4	T5	T6	T7	T8
p-value	0.092	0.342	0.209	0.286	0.108	0.787	0.751	0.343	0.066	0.087	0.733

Figure 7: The Trend in the Mean SpO_2 among the Groups

3.6. Maximum Changes in Haemodynamic Parameters Above Baseline: [Figure 8] shows maximum percentage increases in HR and BP parameters. HR increased most at 1 minute in D1 (20.3%), and at 3 minutes in D2 (14.2%) and D3 (29.8%) (p = 0.015). MAP increases: 34.8% (D1), 29.1% (D2), 42.8% (D3) (p = 0.038). SBP increases: 26.4% (D1), 11.6% (D2), 33.6% (D3) (p = 0.001). DBP increases: 44.5% (D1), 29.1% (D2), 44.6% (D3) (p = 0.006). D3 had the highest increases.



HR (p value =0.015) SBP (p value =0.001) DBP (p value=0.006) MAP (p value =0.038)

Figure 8: Maximum Changes in Haemodynamic Parameters above Baseline.

3.7. Comparison of Ect-Associated St Segment Changes

[Table II] shows that prior to ECT, only 1 patient (4%) in D3 had 2.0 mm ST elevation. Post-ECT, 1 patient in D3 had ST elevation and 4 (16%) had ST depression (1.5–2.5 mm). No ST changes were noted in D1 and D2. No arrhythmias occurred in any group. Group D3 had the highest percentage increases in haemodynamic parameters.

Complication	Items	D1 N = 25 No (%)	D2 N = 25 No (%)	D3 N = 25 No (%)	Total N=75 No (%)	X^2	p-value
Pre-ECT ST Segment changes	ST segment elevation	0	0	1(4.0)	1(4.0)	NA	NA
	ST segment Depression	0	0	0	0		
Post-ECT ST Segment changes	ST segment elevation	0	0	1(4.0)	1(4.0)	NA	NA
	ST segment Depression	0	0	4(16.0)	4(16.0)		

N Number of patients

Table II: Incidence of ECT associated ST-Segment Changes on ECG

3.8. Heart Rate Variability (Hrv) Pre- and Post-Ect

[Table III] shows HRV parameters. Significant post-ECT decreases in rMSSD and increases in pNN50 were observed in all groups. rMSSD decreased by 53.1% (D1), 53.8% (D2), and 43.3% (D3). pNN50 increased by 221.8% (D1), 190.5% (D2), and 186.2% (D3). D2 had the greatest rMSSD reduction; D1 had the highest pNN50 increase.

Groups	HRV Variables	Period	Mean ± SD	Pair difference Mean/ %Change	SD	Pair t-test	p-value
D1	rMSSD (ms)	Pre	81.7±34.3	43.39/ -53.1%	17.23	12.589	0.0001*
		Post	38.3±23.0				
	pNN50(%)	Pre	9.6±8.0	21.29/ +221.8%	10.84	9.820	0.0001*
		Post	30.9±16.5				
D2	rMSSD (ms)	Pre	67.6±21.9	36.32/ -53.8%	13.24	13.714	0.0001*
		Post	31.2±15.7				
	pNN50(%)	Pre	13.7±10.3	26.14/ +190.5%	10.50	12.443	0.0001*
		Post	39.8±16.5				
D3	rMSSD (ms)	Pre	76.2±45.3	33.03/ -43.3%	46.88	3.523	0.0002*
		Post	43.2±27.4				
	pNN50(%)	Pre	6.5±7.8	12.18/ +186.2%	7.99	7.622	0.0001*
		Post	18.6±11.4				

* Statistically significant

Table III: Comparison of Pre- and Post-ECT Heart Rate Variability

3.9. Recovery from Anaesthesia

Tables IV and V show significant differences in recovery times. Time to spontaneous breathing was longest in D2 (3.4±1.7 min), followed by D1 (2.1±1.2 min), and shortest in D3 (1.9±1.0 min) (p = 0.0001). Time to eye opening: D2 (8.2±2.3 min), D1 (6.7±1.4 min), D3 (4.8±1.4 min) (p = 0.0001). Time to obey command: D2 (11.3±2.3 min), D1 (9.2±1.1 min), D3 (7.2±1.4 min) (p = 0.001). Table IV and V confirms significantly longer recovery in D2 for all parameters.

Duration (minute)	Statistics	D1(N=25)	D2(N=25)	D3(N=25)	Total(N=75)	f-test	p-value
Time from the end of motor seizure to spontaneous breathing.	Mean ±SD	2.1±1.2	3.4±1.7	1.9±1.0	2.5±1.4	8.801	0.001*
	Range	1-5	1-8	1-5	1-8		
Time from the end of motor seizure to spontaneous eye opening.	Mean ±SD	6.7±1.4	8.2±2.3	4.8±1.4	6.6±2.3	23.502	0.001*
	Range	3.0-9.0	4.0-13.0	2.0-8.0	2.0-13.0		
Time from the end of motor seizure to obeying verbal command.	Mean ±SD	9.2±1.1	11.3±2.3	7.2±1.4	9.2±2.3	35.046	0.001*
	Range	7.00-12.0	8.0-16.0	5.00-10.0	5.00-16.0		

* Statistically significant

Table IV: Comparison of the Mean Time of Recovery from Anaesthesia between Group

Duration (Minute)	Statistics	D1(N=25)	D2(N=25)	t-test	p-value
Time from the end of motor seizure to spontaneous breathing.	Mean ±SD Range	2.1±1.2 1-5	3.4±1.7 1-8	3.692	0.001*
Time from the end of motor seizure to spontaneous eye opening.	Mean ±SD Range	6.7±1.4 3.0-9.0	8.2±2.3 4.0-13.0	2.794	0.007*
Time from the end of motor seizure to obeying verbal command.	Mean ±SD Range	9.2±1.1 7.00-12.0	11.3±2.3 8.0-16.0	2.794	0.008*

* Statistically significant

Table V: Comparison of the Mean Time of Recovery from Anaesthesia between Group D1 and D2

3.10. Post-Ect Agitation and Other Complications

All patients had an agitation score of 2 at 30 minutes post-ECT [Table VI]. Hypertension occurred in 31% overall, highest in D3 (56%) and lowest in D2 (16%) (p = 0.003). Tachycardia was observed in 36% (D3: 64%, D2: 20%) (p = 0.002). Bradycardia occurred in 1% without significant group difference. Desaturation affected 28%, highest in D3 (44%) and lowest in D1 (16%) (p = 0.03). No patients had shivering, nausea, or vomiting post-ECT.

Complication	D1 (N=25) N(%)	D2 (N=25) N(%)	D3 (N=25) N(%)	Total (N=75) N(%)	X^2	p-value
Hypertension (S.B.P>20% of baseline)	5(20.0)	4(16.0)	14(56.0)	23(30.7)	11.413	0.003
Bradycardia (HR<60bpm)	0(4.0)	1(4.0)	0	1(1.3)	2.027	0.363
Tachycardia (Post ECT)	6(24.0)	5(20.0)	16(64.0)	27(36.0)	12.847	0.002
Desaturation(SpO_2<94 %)	4(16.0)	6(24.0)	11(44.0)	21(28.0)	2.280	0.032
Emergence agitation score of 2 at 30min post-ECT	25(100.0)	25(100.0)	25(100.0)	75(100.0)	NA	NA

N Number of patients % Percentage per group

Table VI: Comparison of the Incidence of Complications among the Groups

4. Discussion

4.1. Summary and Comparative Discussion

This study demonstrated that low-dose dexmedetomidine (0.25 mcg/kg and 0.5 mcg/kg) effectively attenuated hyperdynamic cardiovascular responses associated with electroconvulsive therapy (ECT), without significantly affecting seizure duration. The dexmedetomidine groups (D1 and D2) experienced significantly lower mean increases in heart rate, systolic, diastolic, and mean arterial pressures compared to the placebo group (D3). These findings affirm the sympatholytic efficacy of dexmedetomidine via central α2-adrenergic receptor activation [5]. In line with the findings, Sharan et al. showed that dexmedetomidine (1 mcg/kg) significantly blunted the post-ECT rise in heart rate and blood pressure, more effectively than esmolol or placebo [10]. The present study's lower doses (0.25–0.5 mcg/kg) produced similar effects but with less intensity, confirming a dose-dependent relationship. Subsoontorn et al. also found that both 0.5 and 1 mcg/kg dexmedetomidine significantly reduced SBP and DBP post-ECT compared to placebo, similar to this study, though their longer infusion time (15 minutes) may have enhanced hemodynamic control [11-13].

Interestingly, Li et al. observed no significant attenuation in MAP with a single low dose of 0.2 mcg/kg, possibly due to a shorter 5-minute infusion window—highlighting the importance of infusion duration alongside dosage [14]. Delayed recovery was observed in the dexmedetomidine groups in this study, consistent with findings from Sannakki et al. and Begec et al., who attributed prolonged recovery to higher sedative doses of dexmedetomidine [15,16]. Conversely, Sharan et al. reported no significant delay, suggesting that recovery differences may stem from varying definitions of recovery endpoints [11]. In your study, timing began at seizure cessation, while others timed from muscle relaxant administration. Seizure duration was not significantly altered by dexmedetomidine, corroborating the conclusions of Bagle et al., Subsoontorn et al., and Li et al. [12,15,17]. Since seizure quality is critical to ECT efficacy, this finding supports the safety of dexmedetomidine use in maintaining therapeutic outcomes. This study found ST segment changes only in the placebo group, consistent with the cardioprotective role of dexmedetomidine. Rumi et al. reported a 6.4% incidence of post-ECT ST depression, while Takada et al. and Rasmussen et al. also noted sympathetic surge–linked ECG changes, which were minimized in younger, healthier populations like yours [14,18,19].

HRV analysis in this study revealed increased parasympathetic activity (higher pNN50, lower rMSSD drop) in dexmedetomidine groups, indicating a moderated autonomic response. Bozkurt et al. found no significant HRV change, likely due to delayed ECG monitoring and absence of dexmedetomidine [20]. Your findings are consistent with the immediate autonomic modulation expected from α2-agonists. Despite its known sedative effects, dexmedetomidine did not significantly reduce agitation scores in this study. This contrasts with Shams et al., Subsoontorn et al., and Bagle et al., where higher doses led to better agitation control [12,21,22]. Differences in agitation scoring tools and patient characteristics may account for the discrepancy.

A key finding of this study is the significantly reduced propofol requirement in dexmedetomidine groups, consistent with Parikh et al. and Shams et al., who reported lower induction agent use when dexmedetomidine was co-administered [9,22] .

5. Conclusion

This study supports the effectiveness of low-dose dexmedetomidine (0.25–0.5 mcg/kg) in reducing hyperdynamic cardiovascular responses to ECT without compromising seizure duration. However, delayed anaesthetic recovery and equivocal agitation control were observed, likely dose-related. Compared with similar studies, this work aligns with existing literature in confirming dexmedetomidine's autonomic modulatory and anaesthetic-sparing properties, while highlighting the need for dose optimization to balance safety and efficacy.

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