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Dexmedetomidine for modified electroconvulsive therapy: a dose-optimized treatment study

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Abstract

Objectives To determine the optimal dexmedetomidine dose for hemodynamic stability and recovery quality in modified electroconvulsive therapy (MECT).

Methods In this randomized trial, 252 patients receiving MECT were allocated to six groups (placebo, D1–D5; 42/group). Groups D1–D5 received dexmedetomidine (0.2–1.0 μg/kg) 10 min pre-anesthesia, while controls received saline. Hemodynamic parameters heart rate (HR), mean arterial pressure (MAP), seizure duration, propofol requirements, recovery times, and adverse events were analyzed.

Results Doses ≥ 0.4 μg/kg (D2–D5) significantly reduced HR and MAP versus control ($P < 0.05$), with prolonged recovery in D4–D5 ($P < 0.05$). Seizure duration remained unchanged across groups. Propofol use decreased dose-dependently (D2–D5, $P < 0.05$). The D2 group (0.4 μg/kg) achieved optimal hemodynamic stability without excessive recovery delays.

Conclusions Dexmedetomidine pretreatment at 0.4 μg/kg optimizes MECT anesthesia by balancing hemodynamic control, reduced propofol use, and rapid recovery, establishing it as the recommended dose.

Keywords Dexmedetomidine, Modified electroconvulsive therapy, Anesthesia, Preconditioning, Optimal dose

Introduction

Modified electroconvulsive therapy (MECT) is frequently utilized for the treatment of severe major depressive disorder, mania, anxiety disorders, and schizophrenia [1]. MECT demonstrates significant improvements in safety and efficacy compared to traditional electroconvulsive therapy [2]. The therapeutic efficacy of MECT correlates positively with electroencephalogram (EEG) waveform amplitude during both ictal and postictal phases. However, since most anesthetic agents exhibit anticonvulsant properties that may suppress seizure induction, careful consideration of their inhibitory effects on seizure duration is imperative during the anesthesia induction phase [3–5].

Dexmedetomidine, a potent sedative, alleviates anxiety and stress by suppressing central nervous system activity and modulating neurotransmitters [6]. During the preoperative anesthesia induction phase, dexmedetomidine can facilitate a smooth and rapid transition to anesthesia in conjunction with other anesthetic agents, minimizing patient discomfort [7].

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Prior studies demonstrate that dexmedetomidine pretreatment effectively attenuates acute hyperdynamic responses during MECT [8, 9]. However, dose-dependent prolongation of recovery times has been reported, which may compromise therapeutic efficacy and elevate adverse event risks [10, 11]. Therefore, this study aims to investigate the optimal dose of dexmedetomidine pretreatment in MECT, providing a basis for the rational and effective selection of dexmedetomidine dosage.

Materials and methods

Study subjects

The study subjects were patients admitted to the Affiliated Hospital of Wuxi Health Vocational College from January 2022 to August 2023, who were scheduled to receive MECT. This study was approved by the Medical Ethics Committee of the hospital (Approval Number: LAEY-2021-010). All participants were comprehensively informed of the trial's purpose, procedures, potential risks, benefits, and their rights/responsibilities prior to enrollment. Written informed consent was obtained after confirming their voluntary participation and full understanding of the study. This study was conducted following the CONSORT guidelines, and all experimental conditions conformed to the Declaration of Helsinki.

Inclusion criteria: 1. Diagnoses of schizophrenia, depression, mania, or bipolar disorder per ICD-10 criteria, with a documented illness duration of 1 month to 20 years. 2. Aged 18–58 years, regardless of gender. 3. Failure of pharmacological therapies or presence of treatment-refractory symptoms (e.g., impulsivity, hallucinations, delusions). 4. Absence of medications with significant cardiovascular effects within 48 h pre-procedure and no history of psychoactive substance dependence. 5. Normal cardiovascular/pulmonary function and electrolyte balance confirmed via preoperative assessment.

Exclusion criteria: 1. Allergies to dexmedetomidine or any other anesthetic agents used in this study. 2. Heart failure; patients with second-to-third-degree atrioventricular block, atrial fibrillation, or preoperative heart rate < 55 beats per minute. 3. History of cerebral infarction, severe hypertension, coronary heart disease, bronchial asthma, or liver and kidney dysfunction. 4. Complicated with diabetes, pheochromocytoma, hyperthyroidism, Cushing's syndrome. 5. Psychoactive substance or non-addictive substance-induced psychiatric disorders, organic mental disorders, or psychiatric conditions secondary to other diseases.

Sample size estimation

The sample size was calculated using the formula for comparing means:

$$N = \left(\frac{(Z_{\alpha/2} + Z_{\beta}) \cdot \sigma}{\delta} \right)^2$$

Based on a pilot study ($\sigma = 8.5 \sigma = 8.5$ for HR) and prior literature ($\delta = 5 \delta = 5$), $N = 38 N = 38$ per group was required ($\alpha = 0.05$, power = 90%). To accommodate a 10% attrition rate, 42 patients were enrolled per group. Post hoc analysis confirmed > 90% power for primary outcomes.

Experimental grouping

Patients were randomly assigned to six groups via block randomization (block size = 6, D1–D5 and control group) using a computer-generated sequence (SPSS v13.0). To ensure allocation concealment, assignments were placed in sealed, opaque envelopes opened sequentially by the anesthesiologist post-enrollment. An independent statistician generated the sequence, and baseline characteristics were balanced across groups (Table 1). The D1 to D5 groups received intravenous infusion of dexmedetomidine at doses of 0.2 μg/kg, 0.4 μg/kg, 0.6 μg/kg, 0.8 μg/kg, and 1.0 μg/kg, dexmedetomidine was administered as a single intravenous bolus 10 min pre-induction, consistent with prior MECT studies [8, 12]. This protocol prioritizes hemodynamic stabilization during the brief peri-stimulation phase, avoiding prolonged sedation associated with continuous infusions. The control group received an equivalent volume of saline (30 ml) over the same period.

Table 1 Demographic information of the studied patients

Variables	Control ($n = 42$)	D1 ($n = 42$)	D2 ($n = 42$)	D3 ($n = 42$)	D4 ($n = 42$)	D5 ($n = 42$)	F/χ^2	P
Gender (Male/Female)	23/19	24/18	22/20	23/19	21/21	22/20	0.526	0.991
Age (years)	34.56 ± 7.56	35.82 ± 6.59	36.86 ± 7.15	35.83 ± 7.33	34.95 ± 7.69	35.19 ± 7.67	0.520	0.761
Body Mass Index (kg/m^2)	22.68 ± 1.59	22.86 ± 1.88	22.66 ± 1.91	23.13 ± 1.07	23.21 ± 1.13	22.77 ± 1.43	0.967	0.438
ASA classification								
I	16	14	15	13	14	15	0.579	0.989
II	26	28	27	29	28	27		

MECT treatment

Patients were fitted with an electrocardiogram monitor for continuous monitoring of heart rate, ECG, non-invasive blood pressure, and oxygen saturation. Ten minutes before anesthesia induction, patients received intravenous infusion of either dexmedetomidine or normal saline as per their group assignment.

During anesthesia induction, patients received intravenous propofol (1.5–2 mg/kg) and succinylcholine (0.5–1.0 mg/kg). Following loss of eyelid reflexes, an oropharyngeal airway was inserted and face mask ventilation initiated. MECT device was configured with a frequency of 60–90 Hz, pulse width of 0.5 ms, and energy dose equivalent to 0.5 × patient age (years) to elicit therapeutic seizures. Post-treatment, continuous hemodynamic and respiratory monitoring was maintained until spontaneous breathing and consciousness fully recovered.

Observational parameters

Heart rate (HR) and mean arterial pressure (MAP) were recorded at nine timepoints: immediately after dexmedetomidine pretreatment, and at 5, 10, 15, 20, 25, and 30 min post-treatment. The duration of seizures and the amount of propofol administered were recorded for each group. The time to recovery of spontaneous breathing, time to awakening, and incidence of adverse events were documented to assess the quality of anesthesia recovery across groups.

Statistical analysis

Data analysis and processing were performed using SPSS version 13.0. Descriptive statistics were used for continuous variables, with inter-group comparisons conducted using the *t* test. Categorical data were described using frequency and percentage, with inter-group comparisons assessed using the Chi-square (χ^2) test. For comparisons among multiple groups, one-way ANOVA was employed, and pairwise comparisons were conducted using Tukey's test. $P < 0.05$ indicates a statistically significant difference.

Results

A total of 252 patients were included in this study. There were no statistically significant differences in baseline characteristics among the groups ($P > 0.05$) in Table 1.

Comparison of HR and MAP between different groups

HR in the D2–D5 groups demonstrated significant reductions compared to the control group at five post-pretreatment intervals ($P < 0.05$; Table 2). While the D1 group exhibited a transient HR decrease versus placebo at 15 min post-pretreatment, sustained reductions were observed in the D4 and D5 groups, with HR values at 30 min post-treatment significantly lower than baseline levels ($P < 0.05$).

MAP in groups D3–D5 significantly decreased compared to the placebo group at all timepoints after pretreatment (Table 3). MAP in the D2 group demonstrated statistically significant reductions versus the control group at 15, 20, 25, and 30 min post-treatment ($P < 0.05$). MAP in groups D3–D5 significantly reduced in 30 min by pretreatment ($P < 0.05$). The results are presented in Figs. 1 and 2.

Comparison of seizure duration and propofol consumption in different groups

There were no significant differences in seizure duration among the groups ($P > 0.05$). However, the propofol consumption in groups D2–D5 significantly decreased compared to the placebo group, with the differences being statistically significant ($P < 0.05$). The results are presented in Table 4.

Comparison of anesthesia recovery quality among different groups

As shown in Table 5, the time to the return of spontaneous respiration in groups D4 and D5 significantly increased compared to the placebo group. The awakening time in groups D3–D5 also significantly increased compared to the control group. The incidence of adverse

Table 2 Comparison of heart rate at different time periods in each group during MECT

Time (minutes)	Control ($n = 42$)	D1 ($n = 42$)	D2 ($n = 42$)	D3 ($n = 42$)	D4 ($n = 42$)	D5 ($n = 42$)	F	*P*
0	85 ± 7.34	86 ± 7.55	85 ± 8.28	87 ± 7.78	85 ± 7.36	86 ± 8.27	0.463	0.803
5	80 ± 7.67	78 ± 7.14	71 ± 7.12*	66 ± 7.42*	64 ± 6.89*	60 ± 6.93*	51.336	< 0.001
10	78 ± 6.57	75 ± 7.08	66 ± 7.11*	63 ± 6.39*	61 ± 6.82*	57 ± 6.97*	60.77	< 0.001
15	119 ± 10.73	107 ± 10.37*	96 ± 9.96*	90 ± 9.16*	88 ± 8.36*	82 ± 8.28*	87.052	< 0.001
20	103 ± 8.56	98 ± 9.36	86 ± 8.15*	83 ± 8.58*	82 ± 7.93*	75 ± 7.77*	66.399	< 0.001
25	92 ± 8.12	87 ± 8.25	83 ± 8.48*	80 ± 7.94*	77 ± 7.26*	74 ± 7.19*	29.541	< 0.001
30	88 ± 7.34	84 ± 7.39	81 ± 8.05*	77 ± 7.29*	75 ± 7.20*[a]	73 ± 7.58*[a]	24.52	< 0.001

*Compared with the control group $P < 0.05$. [a]Compared with the same group treatment immediately (0 min), $P < 0.05$

Table 3 Comparison of mean arterial pressure at different time periods in each group during MECT

Time (minutes)	Control ($n=42$)	D1 ($n=42$)	D2 ($n=42$)	D3 ($n=42$)	D4 ($n=42$)	D5 ($n=42$)	F	*P*
0	92 ± 10.56	91 ± 10.34	92 ± 9.35	91 ± 9.45	90 ± 8.58	90 ± 9.69	0.358	0.877
5	89 ± 8.38	86 ± 8.37	84 ± 9.28	80 ± 7.89*	79 ± 9.49*	76 ± 9.36*	12.681	*< 0.001*
10	86 ± 9.33	83 ± 8.34	81 ± 8.37	75 ± 8.24*	72 ± 7.89*	68 ± 7.28*	29.701	*< 0.001*
15	104 ± 10.34	101 ± 10.47	96 ± 10.46*	88 ± 9.73*	86 ± 9.77*	82 ± 9.34*	32.483	*< 0.001*
20	98 ± 8.36	96 ± 8.67	92 ± 9.16*	84 ± 8.92*	78 ± 8.43*	76 ± 8.3*	49.147	*< 0.001*
25	94 ± 9.84	93 ± 8.95	88 ± 8.37*	80 ± 8.79*	74 ± 8.32*	70 ± 8.17*	55.286	*< 0.001*
30	92 ± 8.93	91 ± 8.77	86 ± 8.49*	78 ± 8.33*[a]	72 ± 7.59*[a]	66 ± 7.62*[a]	68.308	*< 0.001*

*Compared with the control group $P < 0.05$, [a]Compared with the same group treatment immediately (0 min), $P < 0.05$

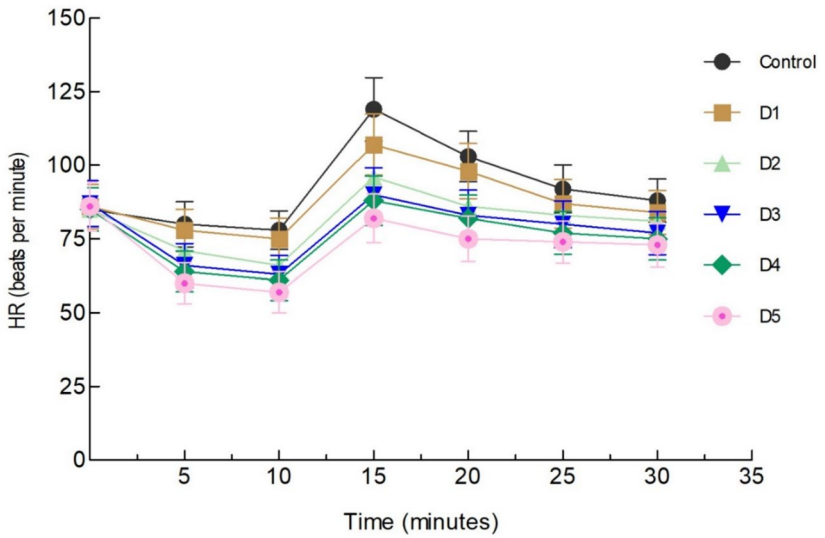

Fig. 1 Statistical line chart of heart rate changes in patients with different doses of dexmedetomidine pretreatment in each group

events significantly decreased in groups D1–D5, with the lowest incidence observed in group D5.

Discussion

MECT, as a commonly used psychiatric treatment method, has significant advantages in improving safety and effectiveness, and expanding indications, but still has limitations. Although electrical stimulation methods are gradually being optimized and improved, there are still some patients who may experience discomfort, such as headaches, agitation, nausea, and vomiting during treatment [13]. Reducing the incidence of adverse events is currently an urgent problem that needs to be addressed [14]. Anesthetic agents, through their anticonvulsant properties, may suppress seizure induction in MECT [15, 16]. Dose-dependent effects further complicate this balance, as higher doses risk attenuating therapeutic efficacy [17]. Therefore, exploring the use of anesthetic drugs while maintaining sedative effects in MECT treatment is one of the hot topics in clinical.

As a sedative drug with clear efficacy, dexmedetomidine has good tolerability and safety, such as minimal respiratory depression, strong sedation, and auxiliary analgesic effects, thereby reducing the demand for volatile anesthetics and postoperative opioid drugs [18]. Studies have found that pretreatment with a certain amount of dexmedetomidine in MECT can alleviate propofol injection pain without affecting seizure duration or reducing acute hyperdynamic responses [12]. However, the optimal dose of dexmedetomidine infusion has not yet reached a consensus in clinical practice.

Our results align with Subsoontorn et al. [7], who reported dexmedetomidine's role in attenuating hyperdynamic responses during MECT. Furthermore, Kuimoto et al. [16] emphasized that preserving seizure quality requires careful anesthetic titration, a principle reflected in our protocol's reduced propofol doses. Finally, our

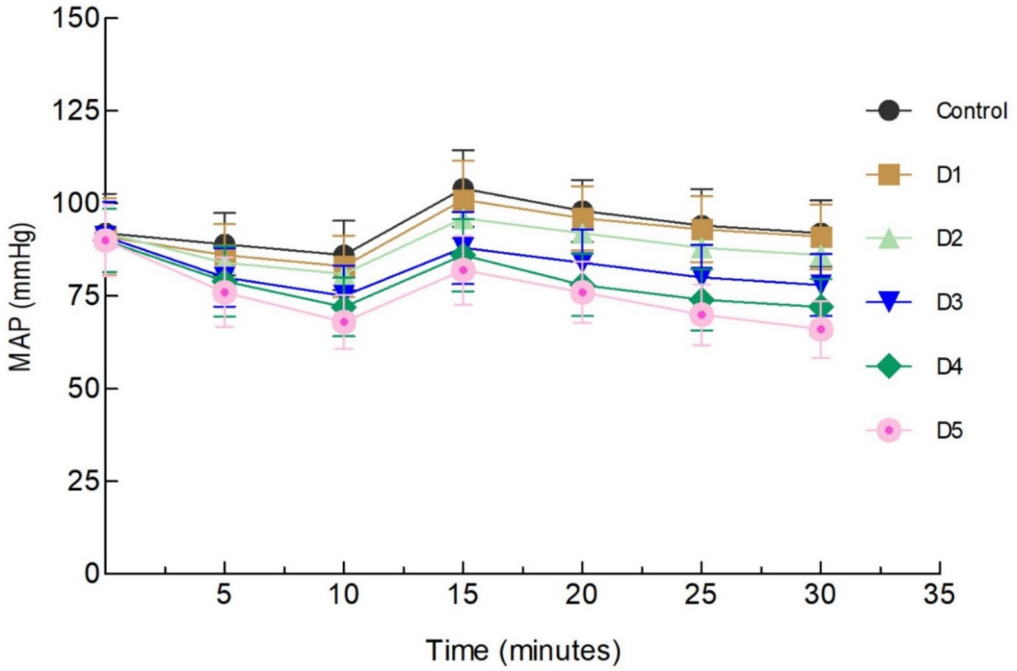

Fig. 2 Statistical line chart of mean arterial pressure changes in patients with different doses of dexmedetomidine pretreatment in each group

Table 4 Comparison of epilepsy duration and propofol dosage between different groups

Groups	Epilepsy duration (seconds)	Propofol dosage (mg/kg)
Control ($n=42$)	44 ± 8	1.20 ± 0.36
D1 ($n=42$)	43 ± 7	1.15 ± 0.33
D2 ($n=42$)	43 ± 8	1.00 ± 0.18*
D3 ($n=42$)	44 ± 7	1.00 ± 0.25*
D4 ($n=42$)	42 ± 6	0.95 ± 0.23*
D5 ($n=42$)	43 ± 8	0.90 ± 0.12*
F	0.438	8.595
P	0.822	*< 0.001*

*Compared with the control group *P < 0.05*

dose optimization findings corroborate Hao et al. [19], reinforcing 0.4 μg/kg as a clinically validated dose.

A study has found that the most common side effects of dexmedetomidine include hypotension and bradycardia, which are usually related to the drug's inhibitory effect on the cardiovascular system [20]. The probability of side effects occurring is directly related to the dosage. The study by Hao et al. [19] pointed out that high-dose dexmedetomidine combined with propofol has a higher risk of hypotension and bradycardia during anesthesia in patients undergoing MECT treatment. While higher dexmedetomidine doses (≥ 0.6 μg/kg) prolonged recovery times, these groups demonstrated lower postoperative agitation rates, likely due to its anxiolytic properties [21]. However, delayed awakening may transiently affect orientation and increase nursing demands, particularly in elderly populations [22]. Importantly, the D2 group (0.4 μg/kg) avoided significant recovery delays while maintaining hemodynamic stability and reducing propofol requirements. This balance supports its recommendation as the optimal dose for MECT, minimizing risks without compromising workflow efficiency.

While seizure duration did not differ among groups, recent literature emphasizes that EEG amplitude and postictal suppression are stronger predictors of MECT efficacy [16]. Although EEG parameters were not measured in this study, the reduced propofol dosage in higher-dose dexmedetomidine groups (D3–D5) may mitigate propofol's anticonvulsant effects, potentially enhancing seizure quality. Future studies should integrate EEG monitoring to validate this hypothesis. Importantly, our findings confirm that dexmedetomidine at 0.4 μg/kg stabilizes hemodynamics without suppressing seizure duration, aligning with its role in optimizing MECT safety.

Studies have shown that dexmedetomidine pretreatment significantly reduces the incidence of postoperative agitation, delirium, hypertension, and tachycardia in elderly patients undergoing MECT treatment [9, 21]. Aksay et al. reported [22] that dexmedetomidine effectively treats

Table 5 Comparison of anesthesia recovery quality indicators among different groups

Groups	Recovery of spontaneous breathing (minutes)	Recovery procedure (minutes)	Adverse event (%)				
			Nausea and vomiting		Restlessness		Headache
Control ($n=42$)	4.8 ± 1.3	13.6 ± 3.6	3 (7.14)		8 (19.05)		9 (21.43)
D1 ($n=42$)	5.0 ± 1.1	14.1 ± 2.9	2 (4.76)		6 (14.29)		9 (21.43)
D2 ($n=42$)	5.1 ± 1.2	14.4 ± 3.3	2 (4.76)		7 (16.67)		8 (19.05)
D3 ($n=42$)	5.4 ± 1.1	15.6 ± 2.8*	1 (2.38)		6 (14.29)		8 (19.05)
D4 ($n=42$)	5.6 ± 1.1*	16.3 ± 3.3*	0 (0)		5 (11.90)		6 (14.29)
D5 ($n=42$)	5.8 ± 1.3*	17.1 ± 3.2*	1 (2.38)		4 (9.52)		6 (14.29)
F/χ^2	3.990	7.728	3.802	1.944		1.489	
P	0.002	< 0.001	0.578	0.857		0.914	

*Compared with the control group $P < 0.05$

refractory postoperative agitation following ketamine anesthesia electroconvulsive therapy. Our research shows that pretreatment with dexmedetomidine significantly reduces the incidence of adverse reactions in patients after MECT treatment.

Conclusion

Pretreatment with dexmedetomidine at 0.4 μg/kg optimizes anesthesia management in MECT by stabilizing hemodynamic parameters, reducing propofol requirements, and enhancing postprocedural recovery quality. This dose demonstrates an optimal balance of efficacy and safety, minimizing adverse events while maintaining therapeutic effectiveness. Based on these findings, 0.4 μg/kg dexmedetomidine is recommended as the standard pretreatment regimen for adult MECT to ensure hemodynamic stability and rapid postoperative recovery.

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Author contributions
The original concept development and compilation are done by corresponding author, JS, MZ and GLZ has played a pivotal role in manuscript design and data interpretation; YZ, JZM and DKL collected the data and did the analysis; LC, QK and AJW did the statistical analysis and created figures; YJ, XML and ZMD drafted the manuscript and revised the article. All authors read and approved the final manuscript.

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Data availability
The data that support the findings of this study are available on request from the corresponding author.

Declarations

Ethics approval and consent to participate
This study was conducted following the CONSORT guidelines, and all experimental conditions conformed to the Declaration of Helsinki. Before starting the project, all participants were asked to complete the written consent form.

Competing interests
The authors declare no competing interests.

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