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# Effect of Dexmedetomidine on Haemodynamic Response and Agitation Score in Modified Electro Convulsive Therapy (ECT): A Prospective Double-Blind, Randomized Controlled Trial

Meenu Sharma, Komal S. Shah, Venu Gandhi and Maya Damor

Department of Anaesthesiology, Sir Takhtsinhji General Hospital, Government Medical College, Bhavnagar-364001, Gujarat, India

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**Key Words**
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**Corresponding Author**
Maya Damor,
Department of Anaesthesiology, Sir Takhtsinhji General Hospital, Government Medical College, Bhavnagar-364001, Gujarat, India

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**ABSTRACT**

Electroconvulsive therapy (ECT) is a beneficial treatment for individuals with psychological disorders who may reject medication. This study aimed to investigate the effects of dexmedetomidine compared to a placebo as pre-treatment options on the hemodynamic parameters and emergence of patients undergoing ETC. A prospective, randomized and double-blinded trial was conducted, involving a total of 60 participants who were equally divided into two groups. Group N received a 10 min intravenous infusion of 100 mL of normal saline. Group D received a 10 min intravenous infusion of dexmedetomidine at a dose of 1 μg $kg^{-1}$, diluted to a total volume of 100 mL. Various parameters, including heart rate (HR), mean arterial pressure (MAP), seizure duration, agitation score and time to discharge, were evaluated by the researchers. In Group N, the recorded heart rates at 5 and 10 min after the electrical stimulus were 113.30±18.79 and 111.37±15.58 beats per minute, respectively. In contrast, Group D exhibited heart rates of 93.63±14.83 and 94.57±20.15 beats per minute at the same time points, showing a significant difference ($p<0.0001$). Systolic blood pressure measurements after 5 min of ECT were 116.53±26.09 mmHg in Group D and 138.03±19.32 mmHg in Group N, indicating a significant difference ($p<0.001$). Diastolic blood pressure and mean arterial pressure were notably reduced in Group D following the induction and electrical stimulus. The duration of seizures was similar between both groups. Additionally, Group D showed an improvement in the Richmond Agitation-Sedation Score. Dexmedetomidine effectively mitigates the impact on hemodynamic parameters during ECT, resulting in decreased emergence agitation without adverse effects on seizure duration or other complications. The administration of dexmedetomidine successfully attenuates changes in hemodynamic responses during ECT while also alleviating emergence agitation, without affecting the duration of seizures or introducing additional complications.

## INTRODUCTION

Electroconvulsive therapy (ECT) is acknowledged as a therapeutic alternative for individuals with schizophrenia, major depression and instances where patients refuse medication. ECT involves the stimulation of the central nervous system to trigger seizures at the beginning of treatment[1]. The cardiovascular reaction during ECT consists of a short parasympathetic phase followed by sympathetic stimulation throughout the seizure. This stimulation causes a notable rise in catecholamine levels, leading to an elevation in heart rate (HR) and mean arterial pressure (MAP).These hemodynamic changes can induce cardiovascular stress and potentially pose a risk of acute coronary or cerebrovascular events for patients with underlying coronary or cerebrovascular disease[2]. Various drug regimens, such as nitroglycerine, fentanyl, labetalol, esmolol, clonidine and dexmedetomidine (DEX), have been used to prevent or mitigate the hemodynamic response during ECT[3-7]. Each of these drugs has its own advantages and disadvantages regarding their impact on hemodynamics, seizure duration and recovery in the context of ECT[3].

In individuals classified as ASA I or II with normal health conditions, the observed hemodynamic changes during ECT generally do not pose a significant risk. However, in elderly patients and those with cardiac or neurological conditions, these hemodynamic variations can potentially raise the risk of myocardial ischemia, pulmonary edema, rare cases of asystole, intracranial haemorrhage, or cerebrovascular accidents. Among patients receiving modified ECT, cardiovascular complications are the primary cause of mortality, with a mortality rate of 0.03%[8,9]. Consequently, anaesthesiologists are highly cautious about attenuating hemodynamic changes and ensuring post-treatment recovery, necessitating the use of optimal pre-treatment regimens. The ideal anaesthetic agent for ECT should act rapidly, not interfere with seizure duration or recovery time and contribute to maintaining the patient's hemodynamic stability[10]. Regrettably, there is currently no universally accepted pre-treatment regimen and the available research on post-procedure recovery and emergence agitation is limited. Consequently, various agents are utilized to improve the comfort and safety of modified ECT. These agents include local anesthetics (such as lidocaine), ganglionic blockers (such as trimethephan), β-blockers (such as esmolol and labetalol), calcium channel blockers (such as nifedipine and nicardipine), α-2 agonists (such as clonidine and dexmedetomidine), direct vasodilators (such as nitroglycerine and sodium nitroprusside) and opioids (such as fentanyl, remifentanil, alfentanil and others)[4,6,11-13].

Among the various options, dexmedetomidine (DEX) is considered an α2-adrenergic agonist that exhibits sedative and antihypertensive properties. When administered, DEX is known to reduce heart rate (HR), systemic vascular resistance and blood pressure (BP)[14]. It has emerged as an anaesthetic agent with a central sympatholytic effect, contributing to the maintenance of the patient's hemodynamic stability. DEX possesses potent anaesthetic and analgesic properties, which can lead to reduced opioid requirements, lower complication rates, decreased stress response and improved recovery quality[14,15]. DEX's anaesthetic effects appear to be unique and can result in mild cognitive impairment, facilitating clear communication between medical staff and patients[16].

The main aim of this study was to compare the cardiovascular effects of intravenous dexmedetomidine at a dose of 1 μg $kg^{-1}$ as a pre-treatment regimen with placebo normal saline and to assess its effectiveness in reducing hemodynamic changes during ECT. The secondary objective was to evaluate postoperative recovery, agitation and any potential complications.

**Materials and Methodology:** This prospective, randomized, double-blinded study was conducted at Sir T. Hospital Bhavnagar, Gujarat, India, after obtaining approval from the Ethics Committee and informed written consent from patients undergoing ECT under general anesthesia and their relatives in the psychiatric ward. The study took place over a period of 9 months in 2020. A total of 30 patients were enrolled in each group, making a total of 60 patients. An additional 10 patients were included to account for potential dropouts. The sample size was determined based on the ability to detect a 10% difference in hemodynamic parameters between the two groups, with a type 1 error of 0.05 and a study power of 90%. Randomization was achieved through simple random sampling using computer-generated random numbers. The inclusion criteria consisted of patients aged 18 to 60 years, of any gender, with ASA physical status I & II and patients with Mallampati grade I & II. Exclusion criteria included patients who refused to provide consent, had known sensitivity to general anesthetic drugs and dexmedetomidine, were pregnant or lactating, had bradycardia (heart rate <50 bpm) or hypotension (systolic blood pressure <100 mm Hg or diastolic blood pressure <50 mm Hg), had a history of myocardial infarction or congestive heart failure within the last 6 months, or had respiratory illnesses such as asthma or COPD.

**One day before the procedure:** A careful history was obtained. A thorough general and systemic

examination was carried out. The patient's preliminary data were recorded. Advice regarding continuation and omission of the dose of antipsychotic medications was given. Inclusion and Exclusion criteria were checked. Written informed consent from patients and relatives of patients was obtained.

**On the day of surgery:** The patients underwent re-evaluation in the preoperative holding area to confirm their Nil By Mouth (NBM) status. The computer-generated randomization chart was used to determine the assigned serial number, which in turn determined the planned administration of the study drug. The anesthesiologist who administered the drug and monitored the patient was not aware of the specific study drug being used. Additionally, the patients themselves were unaware of the type of drug solution they received. Subsequently, the patient was transferred to the procedure table, where monitors were attached and baseline parameters were recorded. The patient received pre-medication consisting of intravenous injection of Ondansetron at a dose of 0.08 mg $kg^{-1}$ and intravenous injection of Glycopyrrolate at a dose of 5 µg $kg^{-1}$.

**Monitoring of the patient included:**

- Pulse Oximeter for Oxygen saturation (SpO2)
- Respiratory rate by manual count
- Blood pressure monitoring by noninvasive BP monitor

**Group N:** The patient received normal saline 100 mL IV over 10 min.

**Group D:** The patient received Dexmedetomidine 1 µg $kg^{-1}$ diluted to the total volume of 100 mL IV over 10 min

Oxygen was supplemented with Bain's Circuit with a face mask at 10 L $min^{-1}$ flow. During the infusion of the study drug, the patient was monitored with the following parameters at 1 min, 5 min and 10 min of infusion.

Pulse rate, Systolic blood pressure, Diastolic blood pressure, Mean arterial pressure, Oxygen saturation ($SpO_2$)

As the volume of the study drug was over, induction was done with inj. Thiopentone 4 mg $kg^{-1}$ slowly IV. After the loss of eyelash reflex, Succinylcholine 0.5 mg $kg^{-1}$ IV was then administered and ventilation was assisted with a bag and mask. An oral soft bite block was placed.

The ECT-induced seizure threshold for each patient was assessed during the initial ECT session. This involved delivering successive stimuli of increasing intensity at 30 sec intervals until a generalized motor seizure activity was observed. The electrical stimulus was administered using bi-frontotemporal electrodes. The duration of the motor seizure was visually observed from the onset of the electrical stimulus until the cessation of tonic-clonic motor activity. The parameters mentioned above, including heart rate, mean arterial pressure and seizure duration, were recorded immediately after induction, as well as at 1, 5 and 10 min after the electrical stimulus.

Immediately after the procedure, the recovery of the patient was assessed with the following parameters in minutes:

- The ability of the patient
- To breathe spontaneously
- To open eyes

**To shift to the post-anesthetic care unit(PACU):** After shifting to PACU, the patient was monitored for the next 50 min, observing for vital parameters and recovery. RASS score was used to assess the recovery at 0, 15, 30, 45 and 60 min. The patient was watched for adverse effects like hypotension, hypertension, tachycardia, bradycardia, arrhythmias, dry mouth, nausea and vomiting.

**Richmond Agitation Sedation Scale (RASS)**
**Target RASS Description:**

| | |
|---|---|
| +4 | Combative, violent, danger to staff |
| +3 | Removes tube(s) or catheters; aggressive |
| +2 | The frequent non-purposeful movement fights the ventilator |
| +1 | Anxious, apprehensive, but not aggressive |
| 0 | Alert and calm |
| -1 | Awakens to voice (eye-opening/contact)>10 sec |
| -2 | Light sedation, briefly awakens to voice (eye-opening/contact) <10 sec |
| -3 | Moderate sedation, movement, or eye-opening. No eye contact |
| -4 | Deep sedation, no response to voice, but movement or eye-openingto physical stimulation |
| -5 | Unarousable, no response to voice or physical Stimulation |

**Procedure for RASS assessment**

- Observe the patient

| | | |
|---|---|---|
| A | The patient is alert, restless, or agitated | Score 0-4 |

- If not alert, state the patient's name and say to open your eyes and look at the speaker

| | | |
|---|---|---|
| B | The patient awakens with sustained eye opening and eye contact | Score-1 |
| C | The patient awakens with eye-opening but not sustained | Score-2 |
| D | Has any movement in response to voice but no eye contact | Score-3 |

- When no response to verbal stimulus, physically stimulate by shaking the shoulder and/or rubbing the sternum

| | | |
|---|---|---|
| E | The patient has any movement to physical stimulation | Score-4 |
| F | The patient has no response to any stimulation | Score-5 |

**Statistical analysis:** The collected data were subjected to statistical analysis using the following tests: The distribution of age, height, weight and diagnosis of the disease were assessed using a chi-square test. Vital parameters such as heart rate, systolic blood pressure, diastolic blood pressure and mean arterial blood pressure were analyzed using an unpaired Student's t-test. The duration of seizure, time taken for spontaneous eye opening, breathing and shifting to the Post-Anesthesia Care Unit (PACU) were evaluated using an unpaired t-test. The sedation scores recorded after the procedure were analyzed using the Mann-Whitney test. A p-value of less than 0.05 was considered statistically significant.

## RESULT

We compared intravenous dexmedetomidine 1 μg $kg^{-1}$ (Group D) versus normal saline (Group N) as a placebo as a premedication drug. A total of 60 cases were enrolled in the study.

In this study, 43.33% (13 patients) suffered from Schizophrenia, 33.33 % (10 patients) suffered from bipolar disorder and the rest 23.33% (7 patients) suffered from Depression shown in Table 1.

The baseline heart rates between group N and group D were similar and not statistically different (p>0.05). However, after 5 min of infusion, the heart rate in group N was 95.27±18.50 beats per minute, while in group D, it was 87.40±16.20 beats per minute, showing a significant difference (p<0.05). Furthermore, after 60 min of undergoing ECT, the heart rate in group D was significantly lower compared to group N (p<0.001) (Table 2).

Before the induction of anesthesia, the systolic and diastolic blood pressures were similar in both groups. However, following induction and the electric stimulus, the systolic and diastolic blood pressures were significantly lower in Group D compared to placebo Group N (p<0.05) (Table 3).

Regarding mean arterial pressure, there were no significant differences between the two groups until the induction of anesthesia. However, in Group D, the mean arterial pressure was significantly lower after induction and the electric stimulus compared to Group N (p<0.05) (Table 4).

Seizure duration was compared between the two groups after the electric stimulus was given. It is evident in the above table, the two groups had similar duration and p-value was not significant. After the convulsions had stopped, immediate recovery of the patients was noted with the above-mentioned parameters. When compared between Group N and Group D. There was no significant difference observed in the time taken for spontaneous breathing between the two groups. However, in group D, there was a significant delay in the other three parameters (p<0.05).

Table 1: Demographic data

| Study parameter | Mean |
|---|---|
| Age (Year), Mean±SD | 31.96±10.14 |
| Weight (Kg), Mean±SD | 60.33±7.42 |
| Height (cm), Mean±SD | 161.46±6.48 |
| Gender | |
| Male (n) | 18 |
| Female (n) | 12 |
| Diagnosis | |
| Depression | 7 (23.33%) |
| Schizophrenia | 13 (43.34%) |
| Bipolar disorder | 10 (33.33%) |

Table 2: Comparison of heart rate among study groups

| Heart Rate (Per min) | Normal saline | Dexmedetomidine | p-value |
|---|---|---|---|
| Baseline | 99.23±21.17 | 95.23±19.37 | >0.05 |
| Start of infusion | 96.63±18.13 | 94.47±20.58 | >0.05 |
| 5 min of infusion | 95.27±18.50 | 87.40±16.20 | <0.05 |
| 10 min of infusion | 93.13±17.47 | 81.50±15.62 | <0.05 |
| After Induction | 104.23±19.65 | 89.37±14.98 | <0.05 |
| 5 min after ECT | 113.30±18.79 | 93.63±14.83 | <0.0001 |
| 10 min after ECT | 111.37±15.58 | 94,57±20.15 | <0.0001 |
| 30 min after ECT | 109.03±13.75 | 90.47±12.87 | <0.0001 |
| 60 min after ECT | 101.07±13.81 | 83.97±11.39 | <0.0001 |

Data are expressed as Mean±SD, p<0.05 is significant using the unpaired t-test

Table 3: Comparison of systolic and diastolic BP

| Parameters | Normal saline | Dexmedetomidine | p-value |
|---|---|---|---|
| Baseline | | | |
| SBP | 124.60±16.85 | 123.27±12.01 | >0.05 |
| DBP | 78.77±10.19 | 78.07±10.17 | >0.05 |
| Start of infusion | | | |
| SBP | 121.63±16.02 | 120.53±14.37 | >0.05 |
| DBP | 76.67±11.13 | 77.97±9.75 | >0.05 |
| 5 min of infusion | | | |
| SBP | 121.93±15.08 | 120.97±16.69 | >0.05 |
| DBP | 95.83±10.82 | 79.60±11.92 | >0.05 |
| 10 min of infusion | | | |
| SBP | 118.37±16.14 | 117.50±18.21 | >0.05 |
| DBP | 75.03±11.69 | 78.33±13.42 | >0.05 |
| After Induction | | | |
| SBP | 119.30±19.24 | 108.13±15.48 | >0.05 |
| DBP | 76.40±12.80 | 70.14±14.05 | >0.05 |
| 5 min after ECT | | | |
| SBP | 138.03±19.32 | 116.53±26.09 | <0.0001 |
| DBP | 82.37±13.28 | 73.77±15.33 | <0.05 |
| 10 min after ECT | | | |
| SBP | 140.10±22.50 | 119.80±22.51 | <0.0001 |
| DBP | 84.27±13.07 | 74.63±14.58 | <0.05 |
| 30 min after ECT | | | |
| SBP | 130.27±20.69 | 110.40±16.87 | <0.0001 |
| DBP | 81.03±16.07 | 69.03±11.27 | <0.05 |
| 60 min after ECT | | | |
| SBP | 118.77±17.69 | 101.53±14.24 | <0.0001 |
| DBP | 80.43±14.39 | 69.97±10.17 | <0.05 |

Data are expressed as Mean±SD, p<0.05 is significant using the unpaired t-test

Table 4: Comparison of mean arterial pressure

| Mean arterial pressure (mmHg) | Normal saline | Dexmedetomidine | p-value |
|---|---|---|---|
| Baseline | 97.20±16.38 | 93.17±10.07 | >0.05 |
| Start of infusion | 95.70±16.25 | 90.70±9.15 | >0.05 |
| 5 min of infusion | 93.70±15.15 | 93.27±13.64 | >0.05 |
| 10 min of infusion | 92.90±16.78 | 90.53±14.65 | >0.05 |
| After Induction | 91.80±18.35 | 82.03±12.14 | >0.05 |
| 5 min after ECT | 101.83±18.13 | 86.63±19.47 | <0.05 |
| 10 min after ECT | 106.07±16.45 | 90.03±17.75 | <0.001 |
| 30 min after ECT | 100.10±17.81 | 82.83±13.88 | <0.0001 |
| 60 min after ECT | 96.10±15.62 | 79.73±12.35 | <0.0001 |

Data are expressed as Mean±SD, p<0.05 is significant using the unpaired t-test

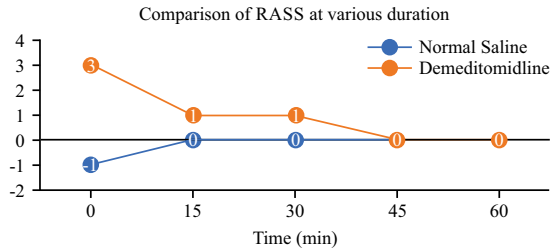


Fig. 1: Comparison of RASS score

Table 5: Comparison of seizure and other parameters

| Study parameter | Normal saline | Dexmedetomidine | p-value |
|---|---|---|---|
| Duration of the seizure (sec) | 27.93±7.91 | 24.77±8.17 | 0.133 |
| Time to spontaneous breathing (sec) | 111.50±56.39 | 135.27±85.60 | >0.05 |
| Time to eye-opening (sec) | 244.43±91.33 | 393.57±192.47 | 0.001 |
| Time to shift to PACU (min) | 5.37±1.75 | 8.73±2.82 | <0.0001 |
| Time to readiness to discharge (min) | 13.00±5.74 | 46.37±19.65 | <0.0001 |

Data are expressed as Mean±SD, p<0.05 is significant using the unpaired t-test

The recovery of patients after the procedure was evaluated using the Richmond Agitation-Sedation Scale (RASS) scores for a duration of 60 min. When comparing these scores between the two groups, there was a significant difference at 0 and 15 min (p<0.05). Based on the values presented in the table, it was evident that patients in group N exhibited more agitation compared to group D (p<0.05) (Fig. 1).

## DISCUSSION

The objective of this study was to compare the impact of premedication with dexmedetomidine (DEX) versus a placebo on hemodynamic responses and seizure duration in patients with psychosis. The findings indicated that DEX yielded the most favorable outcomes, leading to higher patient satisfaction. Consequently, DEX can be considered a desirable option for ECT, as it effectively reduces blood pressure (BP) and heart rate (HR).

Fu and White conducted a study involving six patients to examine the impact of dexmedetomidine at doses ranging from 0.5-1 μg $kg^{-1}$ on the hyperdynamic response during ECT[17]. Their findings revealed that although sedation levels increased with higher doses of dexmedetomidine, it did not lead to a reduction in blood pressure (BP) and heart rate (HR). Consequently, they concluded that dexmedetomidine did not provide any benefit in the context of ECT. In contrast to their study, our results demonstrated a significant decrease in BP and HR in the group receiving dexmedetomidine after the electrical stimulus at all stages (p<0.05). Our findings are consistent with the study conducted by Begec *et al*.[18], where they compared the effects of intravenous dexmedetomidine at a dose of 1 μg $kg^{-1}$ with a placebo[18]. Begec *et al*.[18] concluded that dexmedetomidine was effective in preventing acute responses to ECT.

In this study, Group N's baseline systolic blood pressure was 124.6±16.85 mm of Hg and SBP after 5th min of ECT was 138.03±19.32 mm of Hg. But in Group D, SBP at the 5th min of ECT was 116.53±26.09 mm of Hg as compared to the baseline of 123.27±12.01 mm of Hg. The P Value calculated was significant from the time of induction to 10 min of ECT stimulus. The blood pressure values did not shoot up beyond the baseline values in Group D. We found that in Group N vital parameters returned to baseline values by the end of 10 min of ECT. But in Group D, vital parameters were comparatively still lower than the baseline at the end of the 10th min of ECT, at the same time, patients were hemodynamically stable till discharge. These results suggest that dexmedetomidine is highly effective in suppressing the hyperacute response during ECT. It is important to mention that Fu and White's study administered labetalol in combination with dexmedetomidine[17]. The concurrent use of these medications may have introduced confounding factors that could have influenced their findings. In contrast, our study did not involve such confounding factors, enabling a more accurate assessment of the effects of dexmedetomidine.

In the current study, when comparing the duration of seizures between the two groups, the mean duration in Group N was 27.93±7.91 seconds, while in Group D it was 24.77±8.17 seconds and these values were not significantly different (p = 0.133). These findings are not contradictory to a study conducted by Fu and White, where they observed that both motor and electroencephalographic seizures were relatively prolonged in the group that received pre-treatment with dexmedetomidine compared to the placebo group[17]. However, our study's results differ from the study conducted by Begec *et al*.[18] who found no significant difference in the duration of motor seizures.

The mean time taken for resumption of spontaneous breathing in both the groups was comparable (Group N mean time 111.50±56.39 seconds and in Group D mean time 135.27±85.6 seconds). The p-value calculated was >0.05. However, the time taken for the eye-opening (p<0.01) and time taken to shift from the procedure table to the recovery room were significantly prolonged (p<0.0001) in Group D. Present study results which are comparable to the study done by Fu and White, the mean time to readiness to discharge in Group N was 13.00±5.7 min and in Group D were 46.37±19.67 min (p-value<0.0001)[17]. However, none of the patients required Extended monitoring beyond 60 min post-procedure and all patients were ready to be discharged by the end of 2nd hour of the procedure. None of the patients required hospitalization after the procedure was over.

During the post-ictal period, the recovery of

patients was assessed using the Richmond Agitation Sedation Score (RASS). In Group N, the median RASS score for all 30 patients at baseline was -1. At 15 min, 28 patients achieved a score of 0 and were ready for discharge. By the end of 30 min, the median score reached 0 and only four patients required continued monitoring. In Group D, at 15 min, the median score was 1 and all 30 patients needed further monitoring. At 60 min, 10 patients still required monitoring, with a median score of 0. It was observed that the time taken to achieve a RASS score of 0 for discharge was significantly longer in Group D compared to Group N ($p<0.05$). This finding is consistent with a study conducted by Sannakki *et al*.[19], where they investigated the efficacy of dexmedetomidine at a dose of 1 µg $kg^{-1}$ IV in ECT, focusing on its impact on the hyperdynamic response, seizure duration and sedation. They reported similar results, with a mean RASS score of 0 in Group N at 15 min and 1 in Group D and comparable scores at 30 and 45 min. All patients in Group N were safely discharged at 45 min, while patients in Group D required 90 min for discharge[19]. Our study aligns with a study conducted by Venn *et al*.[20], comparing dexmedetomidine with propofol in patients requiring sedation in intensive care. They concluded that dexmedetomidine is a safe and acceptable sedative agent for those in need of intensive care.

No other complications, such as hypotension, hypertension, tachycardia, bradycardia, dry mouth, nausea, or vomiting, were observed in this study. These findings are consistent with the studies conducted by Fu and White[17] and Begec *et al*.[18], who also did not report any complications in their respective studies.

## CONCLUSION

Dexmedetomidine effectively reduces hemodynamic parameters during ECT without impacting seizure duration. Patients who received premedication with Dexmedetomidine exhibited a calm demeanor (prolonged RASS score) and were sedated in the recovery room. Therefore, based on these observations, we propose that Dexmedetomidine can be considered an ideal choice for patients undergoing ECT.

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