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Original Article

Effects of small-dose dexmedetomidine on hyperdynamic responses to electroconvulsive therapy

Xiang Li [a], Fang Tan [a], Chao-Jun Jian [b], Na Guo [a], Zhi-Yong Zhong [c], Zi-Qing Hei [a], Shao-Li Zhou [a,*]

[a] *Department of Anesthesiology, The Third Affiliated Hospital, Sun Yat-sen University, Guangzhou, China*
[b] *Department of Anesthesiology, The University of Hong Kong-Shenzhen Hospital, Shenzhen, China*
[c] *Department of Psychiatry, The Third Affiliated Hospital, Sun Yat-sen University, Guangzhou, China*

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Abstract

Background: Acute hemodynamic responses to electroconvulsive therapy (ECT) may increase the risk of cardiovascular complications in vulnerable patients. The aim of the current study was to assess the effect of small-dose dexmedetomidine on hyperdynamic responses to ECT.
Methods: Seventy-eight patients were enrolled and randomly allocated to receive either 0.2 μg/kg dexmedetomidine (Dex group, n = 39) or saline (Control group, n = 39) prior to ECT. Heart rate (HR) and mean arterial pressure (MAP) were recorded immediately after the administration of dexmedetomidine (T1), and 0, 1, 3, 5 and 10 min after the electrical stimuli ended (T2, T3, T4, T5 and T6). In addition, the peak HR after ECT, seizure duration, recovery time, and incidence rates of post-ECT adverse effects (agitation, headache and nausea) were also recorded.
Results: HR and MAP in the Dex group were significantly lower than those in the Control group from T2 to T5. In addition, peak HR was significantly lower in the Dex group compared with that in the Control group. Seizure length and time to spontaneous breathing, eye opening, and obeying commands in the Dex group were similar to those in the Control group. The incidence rates of post-ECT agitation and headache in the Dex group were significantly lower than that in the Control group.
Conclusion: The administration of 0.2 μg/kg dexmedetomidine to patients receiving ECT leads to a significant reduction in HR, MAP, and peak HR responses to ECT without altering seizure duration or delaying recovery. Furthermore, dexmedetomidine effectively reduced the incidence rates of post-ECT adverse effects such as agitation and headache.

Keywords: Anesthesia; Dexmedetomidine; Electroconvulsive therapy

1. Introduction

Electroconvulsive therapy (ECT) is an effective non-pharmacological intervention for patients with severe and persistent psychiatric illnesses, and is recommended by the American Psychiatric Association as an alternative to ineffective pharmacotherapy.[1–3] However, hyperdynamic responses resulting from parasympathetic discharge coincident with ECT, followed by a sympathetic response, may increase the risk of cardiovascular complications in patients with ischemic heart disease, hypertension and cerebrovascular disease.[4] In recent decades, it has been customary to perform ECT under general anesthesia supplemented with a muscle relaxant. In addition, a number of anesthetic agents such as α-2 adrenergic agonists and β blockers have proven to be effective in reducing the incidence of hyperdynamic responses during ECT.[4,5]

As a full agonist of the α-2 adrenergic receptor, dexmedetomidine has traditionally been applied during ECT at the dose of 0.5 or 1.0 μg/kg, which effectively blunts the acute hemodynamic response to ECT.[4,6–9] However, some side effects

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* Corresponding author. Dr. Shao-Li Zhou, Department of Anesthesiology, The Third Affiliated Hospital, Sun Yat-sen University, Guangzhou, China.
E-mail address: zsl13610272308@163.com (S.-L. Zhou).

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have been reported. For example, delayed recovery was observed when 0.5 μg/kg dexmedetomidine was administrated during ECT.[9] Moreover, the effect of dexmedetomidine on parasympathetic activity may increase the risk of transient bradycardia or even asystole during ECT.[10,11] These side effects of dexmedetomidine may narrow its application for vulnerable patient populations. Recent studies have evaluated the effects of small-dose dexmedetomidine in clinical practice.[11–13] The present study explored whether the use of 0.2 μg/kg dexmedetomidine would effectively reduce the hyperdynamic response to ECT. In addition, the influence of small-dose dexmedetomidine on seizure duration, recovery time and incidence of post-ECT adverse effects were also assessed.

2. Methods

2.1. Participants

The present clinical trial was in accordance with the Declaration of Helsinki, approved by the Research Ethics Committee of the Third Affiliated Hospital of Sun Yat-Sen University (Guangzhou, China) and registered with the Chinese clinical trial registry (ChiCTR-IOR-15006463).

Patients who were scheduled to undergo ECT as a treatment for a psychiatric condition at the Third Affiliated Hospital of Sun Yat-Sen University between June 2015 and March 2016 were recruited for the study. Inclusion criteria were that patients be between 16 and 55 years of age, and have an American Society of Anesthesiologists' (ASA) Physical Status class of I-II. Patients who declined to participate or suffered any of the following symptoms or diseases were excluded from the study: severe hepatic and/or renal insufficiency, brain organic disease, cardiac insufficiency, sick sinus syndrome, bradycardia, atrioventricular block of degree II and III, and any contraindications of dexmedetomidine. Written informed consent was obtained from each participant and their legal guardian.

All participants were randomly allocated to the dexmedetomidine group (Dex group) or the control group (Control group) using a computer-generated random number with a 1:1 allocation. An independent investigator, not involved in the clinical management or data collection, maintained allocation data. The allocation details were stored in a sealed, opaque envelope in order to enable patients and data collectors to remain blinded to group allocation. Prior to ECT, the allocation details were shown to the anesthesiologists who did not participate in data analysis, and the related data were collected during ECT by an independent staff blinded to allocation details. The allocation was not revealed until final statistical analysis was completed.

2.2. Procedures

After routine monitoring of electrocardiogram (ECG), pulse oxygen saturation (SpO_2) and blood pressure (BP), all patients received 0.006 mg/kg atropine in order to counter the initial parasympathetic effects of ECT.[14] After atropine was administered, patients in the Dex group were infused with 0.2 μg/kg dexmedetomidine (diluted to 10 ml with 0.9% saline) at a steady rate of 2 ml/min, while patients in the Control group were infused with 10 ml of 0.9% saline at the same rate. Our choice of 0.2 μg/kg as the dose for dexmedetomidine was consistent with previous studies that added 0.2 μg/kg dexmedetomidine to intravenous anesthetics for attenuating the hemodynamic stress response during surgeries.[15,16] All patients were oxygenated with 100% oxygen before anesthesia induction. After completion of dexmedetomidine (or saline) administration, anesthesia induction was performed with intravenous 1.5 mg/kg propofol. Subsequent to loss of consciousness, 0.7 mg/kg succinylcholine was administered. Assisted ventilation via face mask was performed using 100% oxygen at a flow rate of 4 L/min for all patients during the ECT procedure. A bite block was used to protect the patients' teeth, lips, and tongue from injury caused by the contraction of facial muscles. The ventilation method was in accordance with previous studies.[4,7,8,17]

When neuromuscular response was completely blocked, ECT was performed by Thymatron System IV (Somatics Inc., Lake Bluff, IL, USA) bitemporal electrode stimulation. The dose of electrical charge was titrated to approximately 50% above each individual's seizure threshold and adjusted as needed according to seizure quality throughout the ECT course. There was an interval of two or three days between each ECT sessions.

Age, weight, gender, HR and mean arterial pressure (MAP) were recorded at baseline for all participants. It was reported that the hemodynamic effect is usually due to a sympathetic response occurring within approximately 10 min of electrical stimulation during ECT procedures, and increasing hemodynamic variables during ECT was most significant during the first 5 min post electrical stimulation. We therefore recorded hemodynamics variables at 10 min post electrical stimulation and performed more frequent observations (1–2 min) for the first 5 min.[4,8,18] The HR and MAP were recorded immediately after administration of dexmedetomidine (Dex group) or saline (Control group) (T1) and at 0, 1, 3, 5 and 10 min after the electrical stimulation ended (T2, T3, T4, T5 and T6, respectively). The selection of time points was in line with the previous studies.[4,8] In addition, the values of peak HR immediately after the electrical stimuli were recorded from the ECG.

Recovery time, seizure duration and incidence rates of post-ECT adverse effects (agitation, headache and nausea) were measured. Recovery time was measured as the time from the end of succinylcholine administration until spontaneous breathing, eye opening, and obeying commands were observed. Seizure duration was measured by electroencephalography (EEG) trace. The incidence rate of post-ECT adverse effects was measured as the proportion of ECT sessions that occurred with adverse effects. Agitation score was recorded after full recovery, and conducted according to the following scale: 1 = sleepy, 2 = awake and peaceful, 3 = irritable and noisy, 4 = disconsolate noisy, and 5 = severe blenched or sitting on the bed and shrieking.[19] The incidence rates of bradycardia (HR < 50 bpm) and hypotension (SAP < 90 mmHg) were