J Anesth (2002) 16:332–335

Journal of
Anesthesia

Clinical reports

Hemodynamic changes during electroconvulsive therapy under propofol anesthesia

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Key words Rate pressure product · Electroconvulsive therapy · Propofol

Introduction

Electroconvulsive therapy (ECT) is effective for serious, drug therapy-resistant depression. Because the therapy can be completed within 10 min, the anesthetics used for ECT should have a short action and a rapid recovery profile. In addition, because the seizure itself is believed to be important for the efficacy of the therapy, the anesthetics used should not interfere with electrical seizure. Recently, propofol at no less than 1 mg·kg^{-1} has been recommended for ECT anesthesia, because many recent studies demonstrated that the hemodynamics during ECT under propofol anesthesia were more stable than those with other anesthetics without propofol [1–4].

The rate pressure product (RPP) is calculated as the product of heart rate (HR) and systolic blood pressure (SBP), and is thought to be an index of the oxygen demand of the heart [5]. However, there has been no report describing RPP change after ECT under propofol anesthesia. In the present study, we continuously measured RPP during ECT under propofol anesthesia. The dose of propofol was 1 mg·kg^{-1}.

It is possible that elderly patients, whose vascular compliance is decreased, show larger hemodynamic alterations during ECT. Also, these patients may have higher risks of cardiovascular complications after ECT. We compared the hemodynamic changes, including RPP, between a low-age group (group 1, age ≦60 years) and a high-age group (group 2, age >60 years).

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Received: November 22, 2001 / Accepted: May 27, 2002

Patients and methods

Informed consent was obtained from each patient or, where necessary, the appropriate relative. The study protocol was approved by a local Clinical Study Committee, which considers the ethics and legal aspects of clinical investigations. ECT was prescribed to 30 patients suffering from endogenous depression. The patients ranged from 15 to 78 years of age and were in good physical heath. No patient had any known cardiovascular or cerebrovascular complication or drug allergy. All patients were treated more than six times (three times per week at 2-day intervals). The data were obtained in the second ECT trial in each case.

To avoid an unfavorable parasympathetic reflex, atropine (0.01 mg·kg^{-1}) was given intramuscularly as premedication. Arterial blood pressure was measured continuously at the right radial artery by a tonometric BP monitor (CBM-7000; Colin, Komaki, Japan). General anesthesia was induced with propofol (1 mg·kg^{-1}). Propofol was administered over 15 s through an intravenous catheter. After loss of consciousness, succinylcholine chloride (1 mg·kg^{-1}) was administered and ventilation was assisted by a face mask with 100% oxygen. One minute after succinylcholine chloride injection, an electrical current was applied bilaterally for 5 s at the minimal stimulus intensity, which had been determined in the first ECT trial by stepwise increasing electrical intensity. The electroshock stimulus was delivered by a trained psychiatrist using an ECT-stimulator (CS-1; Sakai Iryo, Tokyo, Japan). The efficacy of electrical stimulation was determined by the so-called tourniquet technique, which requires observation of convulsive movements of the distal leg, around which an inflated tourniquet was set to block the distribution of muscle relaxant. The end-expiratory CO_2 partial pressure (end-tidal CO_2) at the nostrils and arterial blood oxygen saturation (SpO2) were monitored by a respiration monitor (Capnomac Ultima; Datex, Helsinki, Finland). The

end-tidal CO_2 tension was maintained at 30–35 mmHg, and the SpO_2 value (measured at the left index finger) was maintained above 98% by manual ventilation assistance throughout the study.

For statistical analysis, patients were categorized into a low-age group of ≦60 years (group 1, $n = 17$) and a high-age group of >60 years (group 2, $n = 13$). The data are expressed as means ± SD. The data were compared by analysis of variance for repeated measures with a P value <0.05 considered statistically significant. For comparison of each mean value, two-way analysis of variance was applied, and post hoc testing was performed by Scheffé's method (StatView 5.0, SAS, Cary, NC, USA).

Results

Except for age, there was no significant difference between the demographics of the patients in the two groups (Table 1). The patients had been prescribed multiple psychiatric medications at various doses in their history. However, they were unresponsive to the drug therapy, and the medications were interrupted at least 1 day before the start of ECT therapy. In both groups, HR did not change significantly throughout the ECT trial. Mean arterial blood pressure (MAP) increased until 3 min after the electrical shock in group 1 and until 1 min after the electrical shock in group 2 ($P < 0.05$, Table 2). RPP increased at 0.5 min (158% ± 43% in group 1, 138% ± 23% in group 2) and 1 min (154% ± 33% in group 1, 142% ± 27% in group 2) after the electrical shock compared with preanesthesia values under propofol anesthesia ($P < 0.05$, Fig. 1). The changes in HR, MAP, and RPP did not differ between the two groups.

Discussion

The ideal anesthetics for ECT would have rapid induction, short duration of action, minimal side effects, rapid recovery, and no interference with ECT efficacy. Because of its rapid induction and rapid recovery after anesthesia, propofol was recently introduced for ECT anesthesia. Although propofol anesthesia reduced the duration of seizures as compared with other anesthetics, recent psychiatric reports concluded that the efficacy of ECT under propofol anesthesia did not differ significantly from that under other anesthetics [4,6]. The present study used 1 mg·kg^{-1} propofol for anesthesia induction, as in our previous study. This was because for many patients, less than 1 mg·kg^{-1} propofol was not sufficient to cause them to lose consciousness.

In the present study, HR did not change significantly throughout the ECT trial. MAP increased until 3 min after the electrical shock. This phenomenon and the degree of alterations were comparable with our previous observations [7]. Stable systemic hemodynamics during ECT under propofol anesthesia were described previously in several reports [1–4]. Boey and Lai [3], who compared ECT under thiopental and propofol, found no alteration in either HR or MAP after electrical shock under propofol anesthesia. Several other studies have compared methohexital and propofol and have demonstrated minor hemodynamic changes dur-

Table 1. Patient data (mean ± SD)

Group	No. (M/F)	Age (years)	Height (cm)	Weight (kg)	Voltage (V)	No. % seizures
Total patients	30 (7/23)	50 ± 21	156 ± 6	53 ± 11	102 ± 6	30 ± 8
Group 1 (age ≦60 years)	17 (2/15)	34 ± 13	159 ± 7	55 ± 12	103 ± 7	28 ± 6
Group 2 (age >60 years)	13 (5/8)	70 ± 6	152 ± 6	50 ± 8	100 ± 2	33 ± 9

Table 2. Heart rate (HR) and mean arterial pressure (MAP) change during ECT (mean ± SD)

				Time after ECT (min)					
Measurement	Group	Pre	Ane	0.5	1	2	3	5	10
HR	Group 1	93 ± 24	94 ± 15	105 ± 23	107 ± 24	99 ± 28	100 ± 25	95 ± 33	101 ± 23
	Group 2	92 ± 20	97 ± 24	106 ± 25	105 ± 25	103 ± 23	100 ± 25	97 ± 17	95 ± 21
	All subjects	93 ± 21	95 ± 19	105 ± 23	106 ± 23	101 ± 25	100 ± 25	96 ± 27	98 ± 22
MAP	Group 1	100 ± 9	104 ± 12	139 ± 18*	131 ± 14*	127 ± 15*	120 ± 14*	116 ± 14	107 ± 14
	Group 2	104 ± 9	110 ± 9	126 ± 13*	130 ± 18*	122 ± 16	118 ± 12	113 ± 15	101 ± 11
	All subjects	102 ± 9	106 ± 11	134 ± 17*	130 ± 16*	125 ± 15*	119 ± 13*	115 ± 15	104 ± 13

Pre, Preanesthesia; Ane, immediately after anesthesia
* Significantly different from preanesthesia ($P < 0.05$)

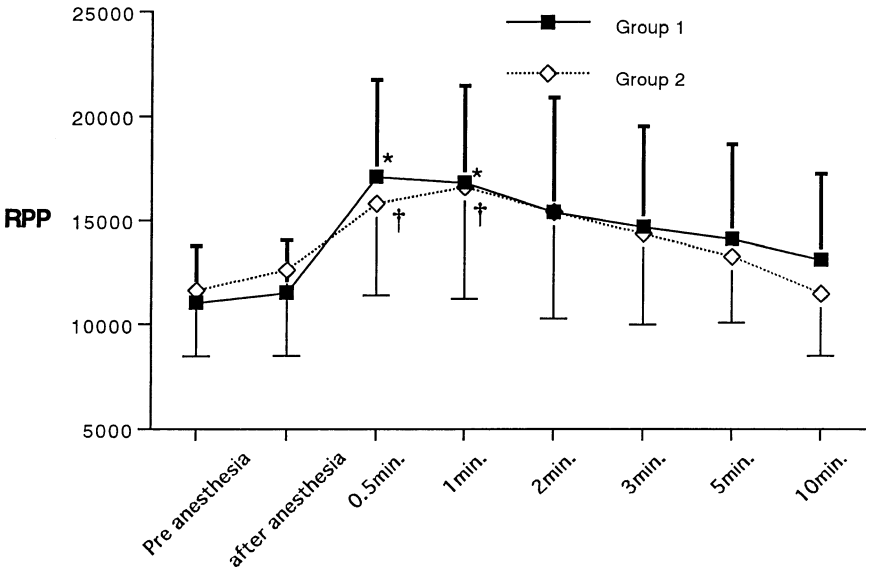

Fig. 1. Rate pressure product during electroconvulsive therapy. The rate pressure product significantly increased after the application of electrical shock, and the increase continued until 1 min after the shock. *† $P < 0.05$. Patients were divided into group 1 (age ≦60 years, $n = 17$) and group 2 (age >60 years, $n = 13$). There was no significant difference between the groups

ing ECT under propofol anesthesia as compared with methohexital anesthesia [1–4]. Fredman et al. [1] reported that 0.75 mg·kg^{-1} propofol, which is smaller than the doses used in other studies, could ensure stable hemodynamics if labetanol was used prior to the electrical shock.

The increase in RPP was observed at 0.5 and 1 min after ECT. Because RPP is thought to be an index of the oxygen demand of the heart [5,8], this continuous increase in RPP suggests an elevated risk of heart ischemia [9]. Kadoi et al. [10] reported that the systolic performance of the left ventricle, estimated by echocardiography, decreased transiently in the period immediately after ECT. These observations indicate that the first several minutes immediately after ECT are critical for patients with cardiac complications, such as angina pectoris or cardiomyopathy. In the present study, we examined the effect of propofol at a single dose because this dose is often used in clinical settings. To compare the pharmacological actions of propofol on cardiac oxygen demand extensively, further examination at other doses will be necessary.

In the present study, we assessed whether aging has a significant impact on hemodynamic changes after ECT. However, the trends in hemodynamic variables were similar in both group 1 (age ≦60 years) and group 2 (age >60 years). There was no significant difference between the two groups regarding MAP and RPP elevation after electrical shock. It has been reported that compliance of blood vessels decreases in the elderly [11]. However, autonomic and endocrine responses are blunted in these patients [11]. The phenomenon we observed in this study might be a net result of these physiological changes in the elderly. Further study with large-scale and multiple physiological measurements will be necessary to clarify the complicated hemodynamic changes after ECT. No study has clearly demonstrated increased cardiac risk among elderly patients with no preexisting disease receiving ECT under general anesthesia [12].

In conclusion, the rate pressure product increased 0.5 and 1 min after electrical shock under propofol anesthesia. There was no significant difference in changes in HR, MAP, or RPP between the low- and high-age groups. Anesthetists need to pay attention to systemic hemodynamic variables during ECT, especially in patients who cannot tolerate abrupt systemic hemodynamic change.

Acknowledgments. The authors thank Dr. Hiroshi Miyota and Dr. Naoya Yuki (Department of Psychiatry, Gunma University) for their cooperation in this study. The authors also thank Dr. Euan Low and Dr. Mayumi Nishihara for editing the manuscript.

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