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Sevoflurane versus propofol for electroconvulsive therapy: effects on seizure parameters, anesthesia recovery, and the bispectral index

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Background/aim: In this prospective randomized cross-over study we compare the effects of sevoflurane versus propofol for electroconvulsive therapy (ECT) anesthesia.

Materials and methods: Twenty four patients (ASA I–III, 18–65 years old) receiving ECT three times per week were included. Anesthesia was induced with either propofol (0.75 mg/kg iv) or 5% sevoflurane in 100% oxygen. Consecutive ECT sessions followed a 2 × 2 crossover design and a 2-day washout period until the 10th ECT. Intravenous succinylcholine (1 mg/kg) was administered while bispectral index (BIS) values were ≤60%.

Results: Electromyogram and electroencephalogram seizure duration, postictal suppression index, BIS values, mean arterial blood pressure (MAP), heart rate, times to start of spontaneous respiration, eye opening, understanding verbal commands, and side effects were compared. No differences were found between the regimens for seizure activity and recovery. At the end of ECT, MAP was higher with sevoflurane. Although BIS values were higher after sevoflurane, no differences between the regimens were found in terms of the need of muscle relaxants and in hypnosis levels.

Conclusion: Sevoflurane (5%) may be an effective alternative to propofol for induction of anesthesia for ECT.

Key words: Electroconvulsive therapy, anesthesia, propofol, sevoflurane, bispectral index

1. Introduction

Electroconvulsive therapy (ECT) is the preferred treatment for affective disorders, and especially major depression, schizophrenia, and other psychotic disorders that do not respond well to psychopharmacological treatment, when pharmacologic treatment is not well tolerated or in situations when a fast clinical response is required. Despite disagreements on the relationship between the effectiveness of ECT treatment and seizure duration, the suggested time for clinical effectiveness is 25–30 s (1,2). Moreover, electroencephalogram (EEG) signs of intense seizure with high amplitude and postictal EEG suppression are reported to be indicative of the efficacy of treatment (1–5). The postictal suppression index (PSI) reports on the degree of EEG flattening at the end of seizure and indicates ECT seizure quality. The PSI is also reported to correlate with clinical efficacy (3,5,6).

Agents used to induce anesthesia for ECT should provide rapid induction and recovery, have few side effects, and not reduce the effectiveness of ECT. However, no anesthetic agent is known to provide all these requirements. A sensitive balance is required between providing optimal seizure duration and achieving a sufficient depth of anesthesia (1). Whereas high-dose anesthetic agents might reduce the effectiveness of ECT, low-dose agents may not provide an adequate depth of anesthesia and may also negatively affect the progress of the psychiatric disease (7,8). Direct measurement of the individual hypnosis level

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is reported to help in determining the anesthetic dosage and ECT application time, as well as reducing awareness (9,10). The bispectral index (BIS) is an EEG-derived scale reflecting the level of hypnosis and an important parameter in anesthesia for ECT (9–11).

Inhalational induction for ECT is preferred for patients with needle phobia, agitation, (known) problems related to inserting an intravenous (IV) line, and for patients with (expected) difficult airway management (12). Sevoflurane is an inhalation anesthetic that provides fast and smooth induction, has a quick elimination, and does not irritate the airways (13). It is also preferred in the last trimester of pregnancy for ECT anesthesia because it reduces uterine contractions (14). Although studies have compared induction with sevoflurane and IV anesthetic agents, there is no consensus on the effects of sevoflurane on seizure duration (12,15–18). It is emphasized that the depth of anesthesia should be determined during induction of anesthesia for ECT (18,19).

In the present study the level of hypnosis was monitored using BIS. The aim was to compare hemodynamic responses, BIS scores, seizure parameters, and recovery profiles for sevoflurane and propofol for induction of anesthesia for ECT.

2. Materials and methods

2.1. Overview

This single-center, single blind, randomized, prospective, crossover exploratory study was performed at Numune Educational and Research Hospital (Ankara). The study was approved by the local Institutional Ethics Committee (2007-08884).

A total of 27 patients (aged between 18 and 65 years) receiving ECT three times a week (to complete an average of 10–12 treatments) were enrolled in the study (between 2007 and 2009). Patients with mask phobia, epilepsy, unstable cardiovascular disease, sinus bradycardia, second or third degree atrioventricular block, cerebrovascular disease, chronic obstructive pulmonary disease, hepatic or renal insufficiency, organic brain disease, current alcohol use or other substance dependence, and patients receiving treatment with beta blockers, anticonvulsants, or benzodiazepines were excluded from the study.

After the patients (or their relatives) were informed about the study and signed consent forms, patients were examined and classified as ASA physical status I–III prior to ECT treatment. During the study, the following patients were also excluded from the analysis: patients who could no longer tolerate mask induction, those who had a very high level of trigger points, patients who had a seizure duration ≥120 s, and patients whose treatments were completed earlier than planned (<8 ECT sessions).

2.2. Study design

Patients were randomized (using the sealed envelope method) to either IV propofol (0.75 mg/kg) or sevoflurane (5%). This study was performed using a 2 × 2 crossover design with a 2-day washout period until the 8th ECT session, at which point the study ended. Electrical stimulus trigger points were determined during the first two ECT sessions; these two sessions were not included in the present analysis, and in these sessions propofol and IV remifentanil were used for induction of anesthesia.

2.3. Procedure and data collection

Patients were brought to the ECT room without any premedication and were administered Ringer's lactate infusion after insertion of an IV line in their forearm. Electrocardiogram (ECG), noninvasive blood pressure, heart rate (HR), peripheral oxygen saturation (SpO_2), end-tidal carbon dioxide (Datex Ohmeda S/5, Bromma), and BIS (BIS XP, Aspect Medical System) were monitored. Hemodynamic data were recorded before induction, after induction, immediately at the end of ECT (ECT-E), and at 1 and 5 min after the end of ECT (ECT + 1 and ECT + 5, respectively). BIS values were recorded before and after induction, before administration of muscle relaxants, immediately before ECT (preictal) and 5 min following ECT (postictal).

ECT was applied bilaterally using the Thymatron TM DGx device (Somatics LLC). EEG electrodes were placed on the frontal and mastoid protuberances by the psychiatrist and the electromyogram (EMG) electrodes were placed on the flexor side of the right forearm. Seizure quality was determined using the PSI, which is calculated by the Thymatron device. All patients were preoxygenated with 4 L/min 100% O_2 for 3 min using a face mask; additionally, 1 min before induction IV remifentanil 1 μg/kg was administered over 30–45 s (in our clinic remifentanil is used routinely to reduce the dosage of the induction agent and to suppress hemodynamic response).

For the first treatment, patients received ECT at 30%–50% of the maximum output stimulus, depending on the attending psychiatrist's decision. After the first ECT session, the same psychiatrist decided on the stimulus amplitudes according to the patient's clinical outcome.

In the propofol regimen, propofol 0.75 mg/kg was administered IV over

30–45 s. Patients were clinically assessed approximately 60 s after propofol administration and additional propofol was given in 10–20 mg increments as needed until loss of eyelash reflex and loss of response to verbal commands. The total propofol dosage was recorded.

In the sevoflurane regimen, the respiratory circuit was primed for 60 s using sevoflurane 5% in 100% O_2 with a 4 L/min gas flow. Patients were asked to breathe through the face mask connected to the system (tidal volume

breathing) and the vaporizer was turned off after induction. Sufficient depth of anesthesia was determined by loss of consciousness, loss of eyelash reflex, and unresponsiveness to the verbal order of "open your eyes"; this was recorded as the induction time. When the BIS value was ≤60, succinylcholine 1 mg/kg (IV bolus) was administered and the times of administration of muscle relaxants were recorded. Ventilation was maintained with a face mask at 4 L/min O_2 and the end-tidal CO_2 was maintained at 32–35 mmHg. Induction of anesthesia was performed with the alternative agent in each consecutive application of ECT (as described above).

Two observers (a psychiatrist and an anesthesiologist) who were not informed about the type of induction were brought into the ECT room before each application of ECT. The EEG and EMG seizure duration, as well as the PSI score, were recorded at the end of the seizure. Times from induction to the beginning of spontaneous respiration, eye opening, and understanding of verbal commands were recorded. Furthermore, adverse side effects such as agitation, nausea, and vomiting, secretion, atrial and ventricular arrhythmias, and bradycardia (HR ≤ 45 beats/min) were recorded. Patients were brought to the recovery room when their modified Aldrete score was ≥9. When the patient was about to leave the recovery room to return to their room, any changes in ECG and/or complications were noted. Then, they were returned to their room when they were able to get off the stretcher and sit on the wheelchair by themselves. During the following 24 h patients were asked about nausea and vomiting, and what they remembered about the ECT sessions.

2.4. Statistical analysis

Baseline characteristics are summarized using number of patients, mean and standard deviation (SD) for continuous data, and number and percentage of patients for categorical data. To investigate any potential imbalance over the two study arms on baseline characteristics, Wilcoxon sum-rank and chi-square tests were used for continuous and categorical subject characteristics, respectively. Additionally, summary statistics were obtained for the outcome parameters at each time point.

One sample Kolmogorov–Smirnov test was used to search the normal distribution of the variables in this study and it was determined that the variables fit the normal distribution.

Outcome variables were analyzed based on the mixed model framework by considering appropriate model structure (fixed effects: sequence, period, and treatment; random effect: intercept and time, with patients nested within sequence). Results are tabulated according to changes from baseline estimates for each study outcome.

The significance level was set at $P \leq 0.05$; all analyses were performed with the open source statistical software package R for Windows. The sample size of the study was determined on the basis of the samples sizes of the previous studies (12,17). The results of this analysis demonstrated a power of 97.9% for two regimens with n = 72 in each group [s (SD):17.84, Δ: –8.58, 2 tailed-α: 0.05].

3. Results

Of the 27 patients enrolled in the study, 17 were diagnosed with depression (of whom 10 had a high risk of suicide and 7 refused oral treatment) and 10 were diagnosed with schizophrenia. All patients were incompatible with oral treatment and had intense psychomotor agitation. Nine of the patients diagnosed with depression had bipolar depressive episodes and the remainder had major depression with psychosis. Of the 27 patients, 2 were withdrawn from the study due to their high threshold values, and 1 patient was withdrawn because he refused the use of a face mask. Therefore, data of 24 patients (with 3–8 ECT sessions) were used for the analyses; the characteristics of these patients are presented in Table 1.

3.1. Seizure parameters

There were no significant differences between the two regimens in terms of induction time, PSI, the energy needed to generate a seizure, and the EMG and EEG seizure duration (Table 2). During a total of 144 ECT applications, mean EMG seizure duration was ≤25 s in 30 patients receiving propofol and in 32 patients receiving sevoflurane ($P > 0.05$).

3.2. BIS variables

Immediately after induction, BIS values were higher in the sevoflurane group compared with the propofol group ($P = 0.000$, Table 2). The times of administration of muscle relaxants were significantly longer with sevoflurane (Table 2). No other significant differences were found between the two regimens in terms of BIS values obtained during administration of muscle relaxants.

3.3. Hemodynamic variables

Data on hemodynamic variables are presented in Table 3. After induction, there was a similar decrease in MAP in both regimens. However, HR was significantly lower with sevoflurane than with propofol treatment ($P = 0.001$). At 1 min after ECT, MAP was significantly higher with sevoflurane than with propofol treatment ($P = 0.048$).

3.4. Recovery parameters

There were no significant differences between the two regimens for the time of starting spontaneous respiration, eye opening, and response to verbal commands (Table 3). There was also no difference between the regimens regarding adverse side effects (Table 4).

Table 1. Characteristics of the study population.

Total number of patients	24
Total number of ECT treatments	144
Mean age in years (range)	38.08 ± 10.06 (20–57)
Mean weight (kg)	69.35 ± 13.38
Gender (male/female)	16/8
Median ASA (range)	2 (1–3)
Indication:	
depression	15
schizophrenia	9
Initial dose of propofol (mg/kg)	0.75
Total dose of propofol (mg/kg)	0.89 ± 0.01
Sevoflurane	5%

Data are presented as means ± standard deviation (SD).

Table 2. Data on seizure outcome and bispectral index (BIS) scores during ECT.

	Propofol Mean ± SD	Sevoflurane Mean ± SD	P-value
Applied energy (mc)	176.92 ± 9.50	178.46 ± 9.01	0.8558
Induction time (s)	69.2 ± 32.3	73.1 ± 40.8	0.480
MRAT (s)	66.1 ± 19.2	89.0 ± 22.7	0.004*
EMG seizure duration (s)	27.5 ± 12.4	29.82 ± 13.6	0.469
EEG seizure duration (s)	41.83 ± 19.8	43.23 ± 32.5	0.832
PSI scores (%)	70.1 ± 23.1	67.5 ± 23.8	0.455
Bispectral index (%)			
Before induction of anesthesia	94.0 ± 3.2	94.78 ± 3.5	0.923
After induction of anesthesia	58.9 ± 15	71.1 ± 17.3	0.000**
Before muscle relaxant	50.0 ± 9.5	55.8 ± 13.1	0.092
Preictal	45.0 ± 10	42.5 ± 13.3	0.129
Postictal	72.5 ± 13.9	69.9 ± 17.5	0.380

Data are presented as means and standard deviation (SD).
MRAT: muscle relaxant administration time; PSI: postictal suppression index.
*: P = 0.004 significantly different from the other regimen. **: P = 0.000 significantly different from the other regimen.

Table 3. Hemodynamic data and recovery times during ECT.

	Propofol Mean ± SD	Sevoflurane Mean ± SD	P-value
Heart rate (bpm)			
before induction of anesthesia	86 ± 13.5	85.4 ± 12.6	0.592
ater induction of anesthesia	80.8 ± 17.3	74.8 ± 16	0.001*
ECT + E	132.2 ± 22.5	131.1 ± 18.4	0.808
ECT + 1	100.1 ± 17.7	96.4 ± 19.2	0.080
ECT + 5	105.1 ± 17.4	105.6 ± 14.8	0.990
Mean arterial pressure (mmHg)			
before induction of anesthesia	87.1 ± 12.1	87.3 ± 13.3	0.901
after induction of anesthesia	72.3 ± 13.3	74.1 ± 13.7	0.353
ECT + E	102.5 ± 21	108.1 ± 23.6	0.051
ECT + 1	99.8 ± 21.0	108.4 ± 23.1	0.048**
ECT + 5	94.1 ± 14.3	93.4 ± 14.8	0.747
Recovery time (min)			
Spontaneous respiration	7.14 ± 1.6	7.31 ± 1.7	0.354
Eye opening	10.43 ± 2.6	11.05 ± 3.29	0.074
Following verbal commands	14.63 ± 5.6	14.80 ± 5.2	0.803

Data are presented as means and standard deviation (SD).
ECT: Electroconvulsive therapy; ECT + E: End of ECT; ECT + 1: 1 min after end of ECT; ECT + 5: 5 min after end of ECT; *: P = 0.001 significantly different from the other regimen. **: P = 0.048 significantly different from the other regimen.

Table 4. Adverse side effects reported after induction with the two regimens.

	Propofol (n=72 ECT applications)	Sevoflurane (n = 72 ECT applications)	P-value
Agitation	13	20	0.108
Nausea/vomiting	3	5	0.414
Secretion	4	2	0.157
Atrial arrhythmia	7	7	1.000
Ventricular arrhythmia	3	4	0.650
Bradycardia	0	1	0.157

4. Discussion

The present study is the first to compare induction with sevoflurane and with propofol for ECT using BIS monitoring to determine the level of hypnosis. No significant differences were found between the two regimens regarding seizure parameters, recovery profiles, and BIS variables. For hemodynamic variables, only MAP at the end of ECT was significantly higher with sevoflurane than with propofol treatment; there were no differences between the two regimens at any other time points. In the present study, mean seizure times were within the suggested range (1,2).

Propofol is preferred for ECT since it provides hemodynamic stability and a comfortable recovery (1,20,21). However, propofol is reported to reduce seizure duration induced by ECT in a dose-dependent manner (19–22). Addition of remifentanil to the induction agent increases seizure time by decreasing the required dose of the anesthetic agent, suppresses the hemodynamic response induced by ECT, and increases the PSI score (22–25). Akcaboy et al. (23) demonstrated that addition of remifentanil (1 μg/kg) to propofol (0.5 mg/kg) increases seizure duration as compared with propofol alone (0.75 mg/kg) (with motor seizure times of 52.2 s and 37.6 s, respectively). For this reason, in our clinic remifentanil is routinely used in ECT induction.

The first study to use sevoflurane as an inhaled anesthetic for ECT anesthesia was performed by Calarge et al. (17); they compared sevoflurane (8% for induction and 2%–4% for anesthesia maintenance) with methohexital in 12 patients (69 treatments) and found that motor seizure time was shorter in sevoflurane treatment. In a total of 56 treatments, Loughnan et al. (12) compared 8% sevoflurane (continued until just before electrical stimuli starts) with 1–1.5 mg/kg propofol and reported that motor seizure time was longer in the sevoflurane group than in the propofol group, but the seizure time was still shorter than the accepted seizure time (the mean time for propofol was 18.5 s vs. 22 s for sevoflurane); while there was no significant difference between the two treatments for EEG seizure times, they reported higher PSI levels in the propofol regimen (with a median of 90.5%) than in the sevoflurane regimen (with a median of 87%). Wajima et al. (18) also compared sevoflurane (induction and maintenance at 5%) to propofol (1.5 mg/kg); they reported that motor seizure time was shorter in the sevoflurane group (with a median of 16 s) than in the propofol group (with a median of 30 s). In another study, Toprak et al. (15) compared 7% sevoflurane (which was ended after induction) with 1.5 mg/kg propofol and found that motor seizure time was significantly higher in the sevoflurane group than in the propofol group (mean time for propofol was 28.91 s, while for sevoflurane it was 43.04 s). Our results for motor and EEG seizure times do not concur with those reported by Loughnan et al. (12) and Toprak et al. (15). Avramov et al. (21) demonstrated that propofol in doses of ≥1 mg/kg caused a 45% decrease in ECT-induced seizure duration. The differences between our results and those of the other groups (i.e. Loughnan et al. (12), Toprak et al. (15), and Wajima et al. (18)) might be due to the amount of propofol used. For example, we used a mean of 0.89 mg/kg propofol, which is lower than the amounts used by the other groups. The reason why Calarge et al. (17) and Wajima et al. (18) found a shorter duration of seizure in the sevoflurane regimen might be because, in their protocols, both groups continued sevoflurane administration after induction. In the study by Wajima et al. (18), the patient population was older (mean age 57 years) than ours, which might imply the need to apply a relatively higher concentration of sevoflurane.

Even after a fixed dose of an anesthetic agent for ECT anesthesia, the hypnosis level is not the same for all patients due to pharmacokinetic differences among individuals (4). However, individual pharmacokinetic differences are dealt with by objective monitoring of the depth of hypnosis. This allows administration of an optimal anesthetic dosage, avoiding an inadequate anesthesia level or too heavy a sedation (4,7).

In the present study, before ECT the BIS values were within a range to prevent awareness and were similar in both groups; however, BIS values obtained after anesthesia induction showed a significant difference between the two regimens. During administration of muscle relaxants, this difference disappeared and a satisfactory depth of anesthesia was achieved. We think that the significant difference in BIS values between the two regimens after induction might be because induction with sevoflurane takes longer compared with induction with propofol. It is reported that changes in seizure duration depend on dosage and on whether sevoflurane is used in the maintenance of anesthesia (12,15,17). In the present study, we think that the differences in mean seizure duration were due to how sevoflurane was administered and the concentration used.

A retrospective study by Porter et al. (25) demonstrated that addition of remifentanil to propofol induction provided higher PSI values. A higher PSI value is reported to be an indicator of more clinical improvement (3,5,6). In contrast to Loughnan et al. (12), who found that the PSI values with sevoflurane were better than with propofol, we found no significant difference in PSI values between the two regimens. Moreover, we found that both anesthetic agents were similar in terms of effectiveness for ECT.

A typical response in ECT after electrical stimulation is an increase in the parasympathetic response (for 10–15 s) followed by a sympathetic response. Hypertension, tachycardia, and ventricular extrasystole are often observed during the sympathetic response (1,18). It is also reported that hemodynamic responses are suppressed when using opioid analgesics in ECT (22,23). Recart et al. (24) evaluated hemodynamic responses and seizure duration in ECT using three doses of remifentanil (25, 50, 100 μg, or saline) in addition to 1 mg/kg methohexital; they found that 100 μg of remifentanil suppressed acute hemodynamic responses without affecting seizure duration and recovery. In our study, immediately after ECT application, MAP increased in both regimens (but

slightly more with sevoflurane than with propofol) and 5 min after ECT cessation the values returned to baseline levels. Although not clinically important, a larger increase in MAP and HR in the sevoflurane group was previously reported (12,15,18). In our study remifentanil was effective in providing hemodynamic stability.

The effectiveness of ECT was not investigated in the current study. Another limitation is the presence of sevoflurane odor after induction; although our observers were blinded to the regimen applied, the odor of this agent lingered on.

Although induction with sevoflurane for ECT is not yet common practice, when indicated, it can be used at a 5% concentration together with BIS monitoring, which provides valuable information about the hypnosis level. We think that sevoflurane can be as effective for seizure outcomes as propofol and that it may be an effective alternative to propofol in ECT practice.

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