Original Article

Comparison of Thiopentone Sodium and Propofol as Anesthetic Agents for Modified Electroconvulsive Therapy

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Abstract

Electroconvulsive therapy is a simple procedure, performed on highly diverse patient population with severe, drug resistant depression and other psychiatric disorders. Due to the occurrence of physical and psychological trauma caused to the conscious patient, has led to the concept of modified electroconvulsuve therapy. Ideal anaesthetic used for electroconvulsive therapy should have characteristics that include rapid induction, shorter duration of action, minimal side effects, rapid recovery and no interference with electroconvulsive therapy efficiency. The present study has compared propofol, which has been increasingly used recently with thiopentone, the drug most widely acceptable even today as anaesthetic agents for electroconvulsive therapy. This study was performed to assess the comparative effects of propofol and thiopentone sodium on recovery profile, hemodynamic stability and seizure duration during and after electroconvulsive therapy.

Key words: Anesthesia, electroconvulsive therapy, propofol, thiopentone sodium

Introduction

The use of electroconvulsive therapy to provoke a generalized seizure was described in 1938 and was performed without anesthesia for almost 30 years. The aim of electroconvulsive therapy is to provide a grand mal seizure; it is a seizure rather than the electrical stimulus, which is responsible for the therapeutic effect. This also causes widespread physiological changes, particularly affecting the cardiovascular system due to activation of the autonomic nervous system.

The cardiovascular changes may be altered by anesthesia and other drugs. The violent muscular contraction during convulsion can be reduced by muscle relaxants. Because of the brief period of unconsciousness require for the therapy, anesthetic agents with rapid recovery profile offer advantages. Therefore, the study was performed to assess the comparative effects of thiopentone sodium and propofol on recovery profile, hemodynamic stability, and seizure duration. It was also done to study whether propofol has a better recovery profile than thiopentone and if it is hemodynamically stable.

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Materials and Methods

- Fifty American Society of Anesthesiologists (ASA) physical status II psychiatric patients who underwent two consecutive electroconvulsive therapies were included in this study. The patients who were anesthetized with propofol or thiopentone sodium randomly for the first electroconvulsive therapy were alternated with the other drug for the subsequent electroconvulsive therapy
- The propofol group received 1.2 mg kg-1 body weight of 1% propofol
- The thiopentone group received 2.5 mg kg-1 body weight of 2.5% thiopentone sodium
- Depolarizing muscle relaxant succinylcholine 0.6 mg kg-1 was given following the induction agent

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- Pulse rate, blood pressure, seizure duration, and recovery time were recorded.

Those with history of recent myocardial infarction (<3 months), recent cerebrovascular accident, angina pectoris, congestive heart failure, major bone fractures, pregnancy, aneurysms of major vessels, porphyria, and patients with shock and hypovolemia were excluded.

Routine investigations such as hemoglobin percentage, random or fasting blood glucose, and urine analysis for albumin and sugar were done. Blood urea and serum creatinine were tested as well as electrocardiogram and chest x-ray were performed as and when required and the weight was recorded.

In this study, concurrent medications were continued as per the psychiatrist's recommendation. Monoamine oxidase (MAO) inhibitors and lithium were discontinued as per the psychiatrist's recommendation prior to anesthesia. Preanesthetic preparation included a period of fasting of at least 6 h, with an empty bladder and bowel. The procedure was generally scheduled early in the morning in a pleasant and safe area, which had a waiting and recovery area.

All patients received injection atropine 0.6 mg intramuscular (IM) half an hour before the procedure and were preoxygenated for 3 min before induction. All the patients had continuous electrocardiography (ECG) monitoring, blood pressure monitoring through noninvasive method recorded every minute throughout the procedure for 10 min or longer if required. The patients were followed up for any complications after electroconvulsive therapy for next 24 h. Seizure response was monitored visually in the isolated left leg; the duration of seizures was recorded. Electroencephalography (EEG) was also recorded during the procedure.

The duration of time for recovery was assessed.

Statistics

The methods used were descriptive study, cross tabs, Chi-square test, independent sample *t*-test, paired sample *t*-test, and repeated measure analysis of variance (ANOVA).

Observation and Results

Data were collected and statistical analysis was performed as explained in the methodology of the study. The results and interpretation are explained below.

The mean age was 28.22 years. The minimum age was 18 years and the maximum age was 52 years.

Graph: Type of psychiatric disorders

Majority of the patients were suffering from depressive disorder [severe depression (24%), recurrent depressive disorder (4%), and schizophrenia (18%) including paranoid schizophrenia] [Figure 1].

There was maximum rise in the heart rate seen in the 1st minute after electroconvulsive therapy in both the groups but the rise was comparatively more in the thiopentone group than in the propofol group. Later, the heart rate reduced to almost the preanaesthesia baseline values after 5 min after electroconvulsive therapy in the propofol group, whereas in the thiopentone group it reduced to the baseline value at about 10–12 min after electroconvulsive therapy [Figure 2 and Table 1].

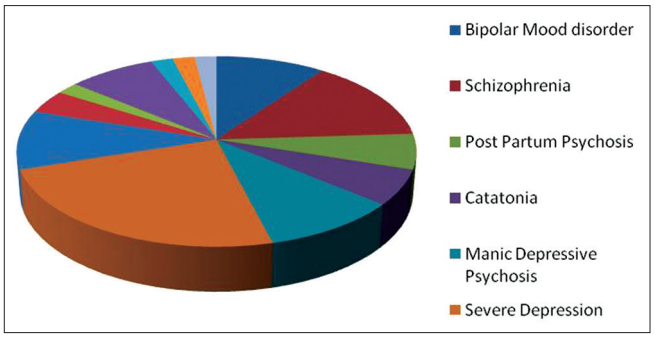

Figure 1: Type of schizophrenia

The maximum rise in systolic blood pressure was seen in the 1st minute after electroconvulsive therapy and the rise was comparatively more in the thiopentone group than in the propofol group. Later, systolic blood pressure decreased in both the groups but the fall was slower in the thiopentone group and systolic blood pressure was seen to be above preanaesthesia values at the end of 5 min and almost reached the preanaesthesia values around 10 min. In the propofol group, the fall in systolic blood pressure was very rapid and systolic blood pressure reached the preanaesthesia values by 5 min after electroconvulsive therapy.

The maximum rise in diastolic blood pressure was seen is the 1st min after electroconvulsive therapy and the rise was more in the thiopentone group than in the propofol group. Diastolic blood pressure decreased after the 1st minute in both the groups but the fall was slower in the thiopentone group and the diastolic blood pressure was more than the preanaesthesia values at end of 5 min returned to preanaesthesia value by 10 min. In the propofol group, the fall in diastolic blood pressure was much more rapid and it returned to the preanaesthesia value by 5 min after electroconvulsive therapy.

The change in mean arterial pressure was observed in a lesser number of patients in the propofol group when compared to the thiopentone group [Figure 3]. Mean arterial pressure of more than 110 mmHg was seen in 24 patients at 1 min, 14 patients at 2 min, 5 patients at 3 min, 1 patient at 5 min, and none at 10 min in the propofol group when compared to 42 patients at 1 min, 32 patients at 2 min, 16 patients at 3 min, 7 patients at 5 min, and 2 patients at 10 min in the thiopentone group [Table 2].

The maximum rise in mean arterial pressure was seen in the 1st minute after electroconvulsive therapy and the rise was more in the thiopentone group than in the propofol group. Mean arterial pressure decreased in both groups after the 1st minute although the decrease was very rapid in propofol group and mean arterial pressure reached preanaesthesia values at 5 min

after electroconvulsive therapy. In the thiopentone group, the fall in the mean arterial pressure after 1st minute was slower and it was still higher than preanaesthesia at 5 min but reached around the preanaesthesia value around 10 min.

Seizure duration in both the groups was above the minimum seizure duration (>30 s) required for therapeutic effect. There was a clinically shorter seizure duration in the propofol group as compared to the thiopentone group but there was no statistically significant change in seizure duration ($P = 0.464$) between the propofol and thiopentone groups [Table 3].

The above table shows that in propofol group 13 patients (26%) recovered in less than or equal to 6 min compared to four patients (8%) in the thiopentone group. Twelve patients (24%) recovered in 6–7 min, 20 patients (40%) in 7–8 min, 4 patients (8%) in 8–9 min in the propofol group compared to 6 patients (12%) who recovered in 6–7 min, 15 patients (30%) in 7–8 min, and 12 patients (24%) in 8–9 min in the thiopentone group. There was only one patient who recovered by 10 min in the propofol group, whereas 11 patients recovered by 10 min and 2 patients recovered by 15 min in the thiopentone group.

Recovery time

There was a statistically significant change in the recovery time. Propofol had recovery time of around 7.36 min and thiopentone around 8.48 min [Figure 4 and Table 3]. Propofol has a better recovery time or very short duration of recovery compared to thiopentone sodium ($P < 0.0001$).

Side effects

There was significantly more discomfort on intravenous injection with propofol. There was no venous thrombosis with either of the drugs. No prolonged apnea was noticed in any of the cases. There were two cases who had delayed recovery in the thiopentone group and they recovered by 15 min after

Table 1: Mean Heart rate

DRUG Mean ± SD	Mean ± SD		P
	Propopol	Thiopentone	
Pre-Anaes	87.64±14.31	85.14±13.32	<0.368
Ind	80.44±15.17	90.42±14.19	<0.179
Post ECT			
1 MIN	107.36±14.89	126.44±16.36	<0.000
2 MIN	101.36±12.29	119.38±16.58	<0.000
3 MIN	96.94±14.39	110.18±15.29	<0.000
5 MIN	88.04±12.17	101.70±15.06	<0.000
10 MIN	83.14±12.26	93.52±13.15	<0.000

Table 2: Mean arterial pressure

DRUG Mean ± SD	Mean ± SD		P
	Propopol	Thiopentone	
Pre-Anaes	95.48±8.61	94.12±8.15	<0.422
IND	90.94±9.26	92.57±8.06	<0.352
Post ECT			
1 MIN	112.44±8.89	122.42±9.81	<0.000
2 MIN	105.84±7.96	116.47±8.71	<0.000
3 MIN	99.86±8.27	108.12±8.45	<0.000
5 MIN	94.62. ±7.48	101.26±8.40	<0.000
10 MIN	90.50±7.92	96.0±8.17	<0.001

Table 3: Mean duration of recovery

Propofol	Thiopentone	P
7.36±1.02	8.48±1.23	<0.0001

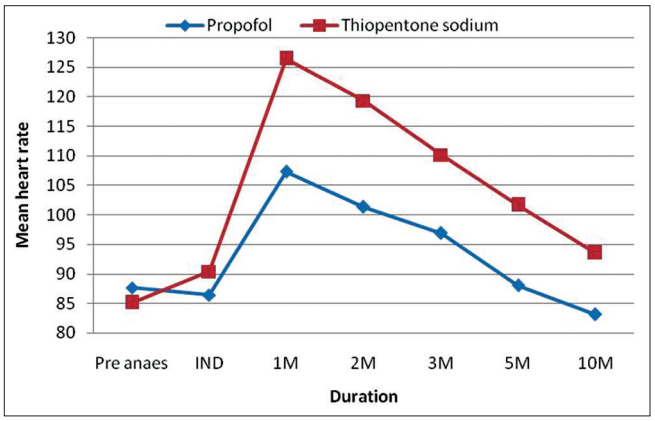

Figure 2: Mean heart rate

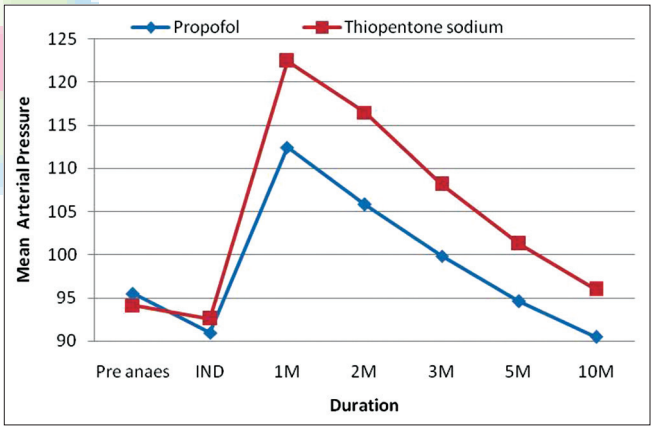

Figure 3: Mean arterial blood pressure

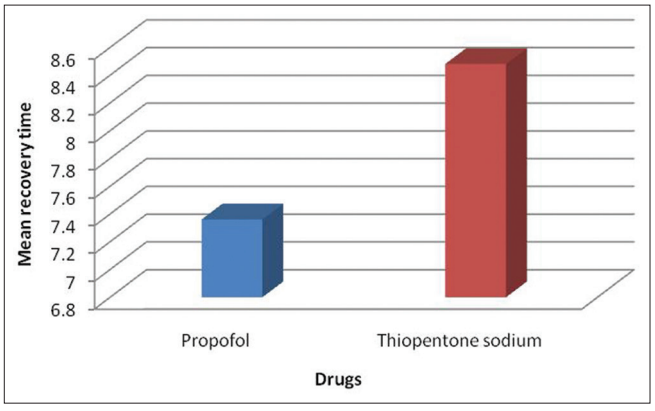

Figure 4: Mean duration of recovery

electroconvulsive therapy. No dysrhythmias were noted in the ECG in any of the cases except sinus tachycardia. Oxygen saturation was maintained between 96% and 99% in all the cases.

Discussion

The ideal anesthetic used for electroconvulsive therapy should have characteristics that include rapid induction, shorter duration of action, minimal side effects, rapid recovery, and no interference with electroconvulsive therapy efficiency.

Because of its rapid induction and recovery profile, propofol was recently introduced for electroconvulsive therapy. The present study has compared propofol, which has been increasingly used recently with thiopentone, the drug most widely acceptable even today as an anesthetic agent for electroconvulsive therapy.

In the present study, the maximum number of patients presented with the diagnosis of severe depression (24%) and schizophrenia (14%). In the study conducted by Boey *et al*. too[1] in 1990, the most common conditions for which patients received electroconvulsive therapy were schizophrenia and severe depression.

The mean induction dose used in the present study for propofol was 1.2 mg kg^{-1} and for thiopentone sodium was 2.5 mg kg^{-1} with a relative potency ratio 2:1. This potency ratio correlated with the relative potency ratios of 2:1 by Leonora *et al*. in1985. The dosage of propofol used was comparable to that used by Simpson *et al*. (1.3 mg kg^{-1}) and Boey *et al*.[1] in 1990 (1.33 mg kg^{-1}). The dosage of propofol used is more than that used by Kadoi *et al*.[2] in 2000 (1 mg kg^{-1}) and Fredman *et al*.[3] (0.75 mg kg^{-1}). In contrast, Mulier *et al*.[4] had reported that propofol at a dose of 1.4 mg kg^{-1} reduced systolic arterial blood pressure mainly through its negative inotrophic properties. The dosage of propofol was more than that used by Gazdag *et al*.[5] (1 mg kg^{-1}) in 2004. The dosage of thiopentone sodium used in present study was comparable to that used by Moacyr A Rosa *et al*.[6] in 2008.

Electroconvulsive therapy itself with a parasympatholytic agent such as atropine causes 25% increase in the heart rate. In the absence of atropine premedication, a marked transient sinus bradycardia occurs immediately following the electroconvulsive therapy stimulus; it may be associated with periods of asystole that last for several seconds (parasympathetic effect). This is rapidly followed by tachycardia.

Saito *et al*.[2] in 2000 studied 40 patients undergoing modified electroconvulsive therapy and administered randomly 1 mg of propofol in comparison with 2 mg of thiopentone and reported that the heart rate in the thiopentone group significantly increased after the application of electrical shock. Maximum heart rate was observed at 1 min after the electrical shock and it was 31% ± 13%. In the propofol group, he reported that the heart rate did not change significantly throughout the electroconvulsive therapy.

Kadoi *et al*.[2] in 2001 reported the heart rate changes using propofol 1 mg kg^{-1} and on comparison, the heart rate changes of the present study is also similar.

In the present study, the heart rate changes following propofol were significantly lower than following thiopentone sodium at all times after electroconvulsive therapy. The mean change in the heart rate after electroconvulsive therapy varied 30–40 beats/min above the baseline values with thiopentone while the mean change in the heart rate observed with propofol was only 10–20 beats/min above the baseline value within the first 3 min followed by a decrease in the heart rate gradually to baseline values in the next 2 min.

Boey *et al*.[1] in 1990 studied 32 patients undergoing electroconvulsive therapy who had administered 1.25 mg kg^{-1} of 1% propofol and 2 mg kg^{-1} of thiopentone sodium and observed that the systolic blood pressure and diastolic blood pressure of both groups increased above the preanaesthesia baseline values significantly after electroconvulsive therapy. The increase in the systolic blood pressure and diastolic blood pressure were greater with thiopentone sodium.

Kadoi *et al*.[2] in 2001 reported the systolic blood pressure and diastolic blood pressure changes using propofol 1 mg kg^{-1}, which were the same as in our study.

Rampton *et al*.,[7] studied the effects of methohexitone (1.08 ± 0.03 mg kg^{-1}) and propofol (1.60 ± 0.04 mg kg^{-1} in 15 patients who underwent electroconvulsive therapy. It was observed that changes in the systolic blood pressure and diastolic blood pressure were less following propofol. The maximum increase over baseline systolic blood pressure was 2.1 ± 2.9 mmHg following propofol ($P < 0.0001$), which was significant.

Geretsegger *et al*.[8] in 2007 also concluded a moderate increase in blood pressure after electroconvulsive therapy with propofol (120.9 ± 50 mg) as compared to methohexitone (83 ± 26.3 mg) in 50 patients.

In the present study, the rise in systolic blood pressure after 1 min of electroconvulsive therapy was 21.28 ± 0.06 mmHg with propofol and 42.27 ± 3.87 mmHg with thiopentone anesthesia. In the propofol group, the fall in systolic blood pressure was very rapid and systolic blood pressure reached the preanaesthesia value by 5 min after electroconvulsive therapy. In the similar way, the diastolic blood pressure increased more in the thiopentone group as compared to the propofol group and reached the baseline value in the propofol group within 5 min after electroconvulsive therapy.

In our study, the change in mean arterial pressure in the 1st minute after electroconvulsive therapy was 16.96 ± 0.28 mmHg in the propofol group as compared to 28.30 ± 1.66 mmHg in the thiopentone group. The fall in mean arterial pressure was rapid in the propofol group and reached the preanaesthesia value within 5 min of postelectroconvulsive therapy, whereas in the thiopentone group mean arterial pressures reached the preanaesthesia value at around 10 min.

Gazdag *et al*.[5] in 2004 compared etomidate (0.2 mg kg^{-1}) and propofol (1 mg kg^{-1}) in 34 patients who underwent electroconvulsive therapy and found similar results for mean arterial pressure. The propofol group had an increase of only 8.1 ± 10.2 mmHg after electroconvulsive therapy.

Shigeru Saito *et al*.[2] in 2000 came to a similar conclusion with mean arterial pressure increase of 39% ± 9% in the thiopentone group after 30 s of electric shock and continued till 5 min; in the thiopentone group, the increase was (17% ± 13%) more than the preanesthesia value.

These studies showed the values of mean arterial pressure after the electroconvulsive therapy were significantly increased in the thiopentone group than with the propofol group, which concurs with the observation drawn in the present study.

Seizure threshold and seizure duration are inversely related. Therefore the electroconvulsive therapy evoked seizure duration may be shortened by anesthetic drugs, which increase the seizure threshold. Furthermore, excessive anesthetic dosage has been cited as a common cause of short or abortive seizures. Since barbiturates increase the seizure threshold and shorten the duration of seizure activity in a dose related manner when used for electroconvulsive therapy, the barbiturate dosage should be minimized.

In the present study the duration of seizure recorded were:

- Propofol group: 35.84 ± 6.21 s
- Thiopentone group: 36.78 ± 6.55 s.

The duration of seizures were compared by using student unpaired '*t*' test and the value ($P = 0.464$) showed no statistically significant change in seizure duration when either propofol or thiopentone were used.

The mean duration of seizure in the propofol group of 35.84 ± 6.21 s correlates with the 37.5 s mean seizure duration recorded by Boey *et al*.[1] in 1990. Gazdag *et al*.[5] in 2004 also reported a seizure duration of 33.6 ± 15.95 with propofol in 34 patients for electroconvulsive therapy. Geretsegger *et al*.[8] 1998 measured seizure duration of 34.1 s with propofol (1 mg kg^{-1}) Fredman *et al*.[3] 1994 also reported the seizure duration with propofol as 34 ± 1.65 s.

Rampton[7] had used propofol (1.6 ± 0.04 mg kg^{-1}) and seizure duration decreased to 17.9 ± 2.5 s.

Gabor *et al*.[9] in 2007 concluded that propofol (1 mg kg^{-1}) has significant seizure shortening properties (32.8 ± 17.6 s) and does not elevate seizure threshold or drop seizure duration under the minimal threshold. Geretsegger *et al*.[8] in 2007 again came out with the results that propofol shortens the duration of seizure and seizure quality, which was not different significantly.

Duration of time to recovery was assessed by a single measure, asking the patient to open his or her eyes at 1-min interval and recording the time from the end of the injection of the initial dose of induction agent until spontaneous movements and response to verbal commands was first obtained. In the present study, mean recovery time were as follows:

- Propofol group: 7.36 ± 1.02 min
- Thiopentone group: 8.48 ± 1.23 min.

40% of patients recovered within 7–8 min in the propofol group, whereas the thiopentone group had 30% patients recovered in that duration of time whereas only 2% of the patients had recovery time ≥9 min in the propofol group in contrast there were 26% of patients had recovery time ≥9 min duration in the thiopentone group. These patients eventually recovered at around 10–12 min.

The values were compared using student unpaired *t*-test and the value ($P < 0.0001$) was found to be statistically significant.

This result concurred with the conclusions found by Moacyr A. Roas *et al*.[6] in 2008 where, the recovery time with propofol (1-1.5 mg kg^{-1}) and thiopentone (2–3 mg kg^{-1}) were 7.4 min and 9.4 min, respectively.

Fredman *et al*.[7] in 1994 concluded that cognitive recovery with propofol was more favourable. Geretsegger *et al*.[8] in 2007 concluded that improved cognitive performance was seen after propofol (120.9 ± 50 mg) anesthesia for electroconvulsive therapy.

In the present study patients in the propofol group recovered faster, had a lesser hemodynamic variation when compared to patients in the thiopentone group. There was no significant decrease in the seizure duration in both the groups.

Conclusion

An effort to avoid or minimize the physiologic sequelae and attendant complications of electroconvulsive therapy, a technique of modified electroconvulsive therapy has evolved gradually, featuring the use of drugs to minimize the detrimental effects of electroconvulsive therapy without the concomitant abolition of the essential beneficial effects.

Based on the present study, we conclude that propofol when compared to thiopentone sodium is a safe anesthetic agent for electroconvulsive therapy with minimal side effects.

The heart rate and mean arterial pressure variability were significantly less in the propofol group as compared to thiopentone sodium. The recovery of patients was faster with propofol compared to thiopentone sodium.

So, propofol is superior to thiopentone sodium in attenuating the physiological response to electroconvulsive therapy with milder hemodynamic change and better recovery profile.

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Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Boey WK, Lai FO. Comparison of propofol and thiopentone as anaesthetic agents for electroconvulsive therapy. Anaesthesia 1990;45:623-8.
2. Saito S, Kadoi Y, Nara T, Sudo M, Obata H, Morita T, *et al*. The comparative effects of propofol versus thiopental on middle cerebral artery blood flow velocity during electroconvulsive therapy. Anesth Analg 2000;91:1531-6.
3. Fredman B, d'Etienne J, Smith I, Husain MM, White PF. Anesthesia for electroconvulsive therapy: Effects of propofol and methohexital on seizure activity and recovery. Anesth Analg 1994;79:75-9.
4. Mulier JP, Wouters PF, Van Aken H, Vermaut G, Vandermeersch E. Cardiodynamic effects of propofol in comparison with thiopental: Assessment with a transesophageal echocardiographic approach. Anesth Analg 1991;72:28-35.
5. Gazdag G, Kocsis N, Tolna J, Iványi Z. Etomidate versus propofol for electroconvulsive therapy in patients with schizophrenia. J ECT 2004;20:225-9.
6. Rosa MA, Rosa MO, Belegarde IM, Bueno CR, Fregni F. Recovery after ECT: Comparison of propofol, etomidate and thiopental. Rev Bras Psiquiatr 2008;30:149-51.
7. Rampton AJ, Griffin RM, Stuart CS, Durcan JJ, Huddy NC, Abbott MA. Comparison of methohexital and propofol for electroconvulsive therapy: Effects on hemodynamic responses and seizures duration. Anesthesiology 1984;70:412-7.
8. Geretsegger C, Nickel M, Judendorfer B, Rochowanski E, Novak E, Aichhorn W. Propofol and methohexital as anesthetic agents for electroconvulsive therapy: A randomized, double-blind comparison of electroconvulsive therapy seizure quality, therapeutic efficacy, and cognitive performance. J ECT 2007;23:239-43.
9. Gábor G, Judit T, Zsolt I. Comparison of propofol and etomidate regarding impact on seizure threshold during electroconvulsive therapy in patients with schizophrenia. Neuropsychopharmacol Hung 2007;9:125-30.