Comparing Two Types of Anesthetics Regarding Efficacy, Safety, and Hemodynamics, in Using Electroconvulsive Therapy in Depressed Patients

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ABSTRACT

Background: Electroconvulsive therapy (ECT) is an accepted modality of treatment in major depressive disorders. There have been many reports describing cardiac morbidity associated with ECT. Ideal anesthetics used for ECT should have rapid induction, short duration of action, minimal side effects, rapid recovery, no interference with ECT efficacy and haemodynamic changes. **Objective:** to evaluate the safety and efficacy of ECT in depressive disorders using either propofol or midazolam as an anesthetic agent, and to study their effects on haemodynamics during ECT. **Subjects and methods:** 30 patients with major depressive episode criteria were scheduled for modified ECT. Hamilton rating scale, Beck depression inventory and GGIS were used to assess depression and improvement. They were divided into 2 equal groups to receive propofol (1mg/kg) and midazolam (0.1mg/kg) on performing bilateral ECT Cardiac function was examined by transthoracic echocardiographic. Induction time, duration of seizure and recovery time were noted. **Results:** The induction time was least in propofol group (42.16±6.71sec). Duration of seizure was least in midazolam group (20.8±4.33sec) and was (28.86±3.48sec) in propofol group. Propofol had significantly faster clinical recovery than midazolam. In propofol group, no change was observed in heart rate during the study. There was no statistically significant change in afterload, and end-diastolic area (EDA) but there was an increase in end-systolic area (ESA) and decreased fractional area change (FAC) at one minute after the electrical shock compared with the awake condition. In the midazolam group, there was an increase in heart rate from one to three minutes after the electrical shock. An increase in afterload was observed from one to five minutes after the electrical shock. Decrease of FAC was observed from one to three minutes after the electrical shock compared with the awake condition. After ECT course (6 sessions), 53.33% achieved remission (scores on HRSD -17 item <7), 46.66% improved on clinical global impression scale (CGIS). **Conclusion:** Electroconvulsive therapy is a recommended treatment for severe depression. Propofol appears to be a safe anesthetic for ECT. It provides excellent haemodynamic stability, quick induction and early recovery during the procedure. So, propofol can be used safely in cardiac and elderly patients.

Key words: Anesthetics, Hemodynamics, Electroconvulsive therapy, Depression, Propofol, Midazolam

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INTRODUCTION

Electroconvulsive therapy (ECT) has an established and important role in the management of medical resistant depression and life threatening conditions such as depressive stupor, catatonia and suicidal drive[1-4]. ECT seems to increase brain GABA levels as well as GABA β activity and these effects may contribute to its therapeutic mechanisms[5]. There have been many reports describing cardiac morbidity associated with ECT. The cardiac morbidity of ECT is due to arrhythmias and instability of arterial blood pressure resulting in myocardial infarction and cardiac arrest. Therefore it would be important to assess the cardiac function during ECT6.

The typical cardiovascular response to ECT consists of generalized autonomic nervous system stimulation with an initial parasympathetic stimulation induced bradycardia lasting 10-15 seconds, followed by more prominent sympathetic response that results in transient tachycardia and hypertension lasting minutes or longer. The cardiovascular response is associated with release of catecholamines and occasional cardiac arrhythmias[7].

Systolic blood pressure (SBP) is transiently increased by 30-40% and heart rate (HR) is increased by 20% or more resulting in two or four fold increase in the rate pressure product (RPP), an index of myocardial oxygen consumption[8]. The peak HR and SBP values occur 3-5 minutes after the application of electrical stimulus[9].

An echocardiographic automated border detection system has been developed which provide on line, beat-to-beat values of left ventricular cavity area. These measurements can then be used to estimate load-dependent parameters or systolic performance such as fractional area change (FAC) or ejection fraction[6].

Ideal anesthetics used for ECT should have characteristics that include rapid induction, short duration of action, minimal side effects, rapid recovery, and no interference with ECT efficacy[10]. Many anesthesiologists prefer anesthetic agents for brief anesthesia, such as propofol and midazolam[11]. Propofol is an alkyl phenol, introduced for ECT anesthesia because of its rapid induction, rapid recovery, and short elimination half life with high clearance rate[10,12]. It provides haemodynamic stability and allows cognitive function to return sooner[13]. It is known that propofol reduces nausea and vomiting. It is used in patients with preexisting hypertension and tachycardia, who exhibit excessive haemodynamic response[14]. Midazolam is a water soluble, potent and short acting benzodiazepine that has been used for ECT[11]. It has significant anticonvulsive property and decreases arterial blood pressure and heart rate.

Sedation with midazolam or propofol does not alter indices of left ventricular diastolic function in healthy patients and those with preexisting left ventricular filling abnormalities as evaluated by transthoracic echocardiography[15].

The purpose of this study was to evaluate the safety and efficacy of ECT in depressive disorders and haemodynamic changes during ECT under anesthesia with propofol versus Midazolam.

SUBJECTS AND METHODS

This study was conducted in Psychiatry, Anesthiology and Cardiology Departments of Zagazig University Hospitals between January 2008 and July 2008. The study sample was recruited from patients with major depressive episode diagnosed on the basis of a structured clinical interview on the basis for DSM-IV and clinician version (SCID)[16]. Patients were selected by using a randomized controlled trial. Thirty patients, 12 males and 18 females, age range (22-50), mean ±SD (38.13±13.2), 13 of the total number were in their first depressive episode (drug naïve) and 17 of the total number had recurrent depressive episode (with no medical treatment for at least 10 weeks). Informed consent was obtained from the patients or their relatives.

The patients were subjected to general medical examination with careful cardiological examination. Patients with unstable cardiovascular diseases, those receiving β-adrenergic or calcium channel blocking drugs and those with second or third degree atrio-ventricular blockage, arrythmia, hypotension, sinus tachycardia, chronic pulmonary obstructive disease and renal or hepatic failure were excluded.

Participants had no history of schizophrenia or schizoaffective, active substance misuse or nurological illness. Baseline scores on the 17-item Hamilton Rating Scale for Depression (HRSD)[17] were at least 25 or over. Beck depression inventory (BDI)[18] was used for subjective assessment of depression severity. Base line scores were at least 30 or over. The two scales were used to assess severity of depression before and after ECT course (6 sessions). Clinical global impression scale (CGIS)[19], which generates ratings of both illness severity and clinical improvement was used at the end of ECT course. The improvement scale represents an assessment of global change is based on a seven-point scale. The patients were divided into two equal groups, propofol group and Midazolam group (each one included 15 patients).

Procedure:

All patients were kept nil orally for eight hours before ECT session. The patients received atropine sulphate (0.01mg/kg) prior to the procedure to avoid parasympathetic reflexes.

All data of arterial blood pressure (BP), heart rate (HR), arterial oxygen saturation (SPO_2) and echocardiography monitoring were recorded before ECT (awake state), and then throughout the procedure (one minute after induction of anesthetesia then one, two, three, five and ten minutes after electrical shock).

The dose of propofol was 1 mg/kg which was the minimal dose to induce unconciousness[6]. The dose of midazolam was 0.1mg/kg. After the administration of propofol over 15 seconds or midazolam and loss of conciousness, succinyl choline (1mg/kg) was administrated and ventilation assisted by face-mask with 100% oxygen till return of spontaneous respiration. One minute after induction of anesthestia, bilateral electrodes were applied to the patient in the frontotemporal position[20]. The machine used was ECTONUSTIM model ECT apparatus and brief pulse stimulus was given for about 2 msec (Voltage 210-240volt, 200Millicoulombs). Seizure duration was measured using the cuff technique[21]. Induction of a seizure was confirmed by inflating a sphygmomanometer cuff to 200 mmHg just before administration of the muscle relaxant: the convulsion was thus observed in the forearm distal to the cuff.

Transthoracic echocardiographic (Hewlett Packard SONOS 5500R, 3-5 MHz transducer Andover, MA, USA) used in this study, had automated border detection system (Acoustic Quantification® system), which could trace the endocardial border continuously, compute the cross section area of the left ventricular cavity and measure the P-R intervals from the corresponding displayer electrocardiographic tracing[22].

Left ventricular parasternal short axis-images were recorded at the mid-papillary muscle level for measurements. While viewing the real-time images, the investigator activated automated border detection and adjusted the time gain compensation controls of the ultrasonograph to facilitate the automatic tracking of the highlight indicator on the echocardiographic image of the left ventricular endocardial border.

Using the track ball of the ultrasonograph, the investigator outlined a region of interest that contained the left ventricular cavity throughout the cardiac cycle, excluding other cardiac chambers. The areas of left ventricle (LV) were then calculated from each cardiac cycle and displayed in a wave form.

All studies were performed by the same operator to eliminate interobserver variability and improve reproducibility.

The following left ventricular indices were measured for each patient (1) end-systolic area (ESA), (2) end-diastolic area (EDA), (3) fractional area change (FAC); FAC=(EDA-ESA)/EDA multiplied by 100 according to Mulier and colleagues

Statistical analysis:

Data were checked, entered and analyzed by using Epi-Info (2000) software computer package. Unpaired t-test was used for comparison between two groups. Paired t-test was used to measure the change within each group ($P<0.05$) was considered statistically significant.

RESULTS

Participants allocated to bilateral electrode placement were similar and the two groups were not significantly different in age, sex, weight, height, duration of the current episode, number of prior episodes and age at onset of the first depressive episode (table 1).

Prior to ECT course participants were severely depressed as indicated by scores on HRSD (mean±SD, 29.5±5.8), and scores on BDI (35.2±10.4). At the end of ECT course the scores were 6.2±5.9 on HRSD for 16 patients and 15±9.3 for 17 patients on BDI. There is significant difference between scores on both scales prior ECT course and at the end of the course (table 2).

As shown in table (3), 46.67% of patients very much improved, 36.67% much improved and 16.66% minimally improved as measured by CGIC after ECT course.

Table (3): Scores on CGIC after ECT course.

Items	Patients	
	No	%
Very much improved	14	46.67
Much improved	11	36.67
Minimally improved	5	16.66

CGIC: Clinical global impression of change.

The induction time was shortest with propofol (42.16±6.17). It was 76.89±6.71 with Midazolam. The induction was smooth with propofol in comparison to midazolam (table 4).

Table (4): Time for onset of induction

Time (seconds)	Propofol group (N=15)	Midazolam group (N=15)
Mean±SD	42.16±6.17	76.89±6.71*

*P<0.001

Duration of seizure was lesser in midazolam-group (20.8±4.33) than propofol group (29.86±3.48) which is significantly different (table 5).

Table (5): Duration of seizure (seconds).

Time (seconds)	Propofol group (N=15)	Midazolam group (N=15)
Mean±SD	28.86±3.48	20.8±4.33*

**P<0.001

Table (6) showed that, there is significant difference in recovery time ($P<0.05$) between propofol group and Midazolam

group. Recovery time was shorter in propofol group.

Table (6): Recovery time (min).

Parameter	Propofol group (N=15)	Midazolam group (N=15)
Spontaneous respiration	4.5±1.2	4.7±1.0
Eye opening	3.93±0.78	7.9±1.24*
Ability to setup unaided	8.12±1.05	14.23±1.83*
Following commands	3.96±0.87	8.16±1.36*

*P<0.05 values are mean±SD

Incidence of coughing, laryngospasm, gag reflex, movements of head and limbs, nausea, vomiting and postictal confusion and delirium were minimum with propofol.

As regard oxygen saturation, there was a significant decrease in midazolam group in comparison with propofol group only at 3 minutes after the electrical shock. In propofol group, there was a significant decrease at 1 and 2 minutes after the electrical shock compared with the patient status when awake. In midazolam group there was a significant decrease of oxygen saturation at 1 minute, 2 minutes and 3 minutes after the electrical shock compared with the patient status when awake (table 7).

Table (7): Oxygen saturation in two groups during ECT procedure.

Measurement time	**Propofol group**	**Midazolam group**
1	96.7 ± 3.1	97.2 ± 2.0
2	97.1 ± 2.1	97.2 ± 2.1
3	89.7± 8.2	91.4± 7.4*
4	92.7 ± 5.1	91.4 ± 7.4*
5	95.1 ± 2.1	92.1 ± 3.7*
6	96.9 ± 1.0	96.7 ± 1.0
7	96.9 ± 1.0	96.6 ± 1.5

Values are means ± SD *P<0.05 = Compared with period (1) $^{+}$P<0.05 = Compared with propofol group (1) Awake (2) One minute after the propofol or midazolam administration (3) One minute after electrical shock (4) Two minutes after electrical shock (5) Three minutes after electrical shock (6) Five minutes after electrical shock (7) Ten minutes after electrical shock

As regard heart rate, no change was observed in HR in the propofol group during the study. In contrast, in the midazolam group there was an increase in HR at 1, 2 and 3 minutes after the electrical shock. There was a significant increase in HR in midazolam group from 1 to 3 minutes after the electrical shock when compared with propofol group (table 8 and fig. 1).

Table (8): Changes of heart rate in two groups during ECT.

	Propofol group	**Midazolam group**
1	77.3 ± 13.5	72.8 ± 11.5
2	78.9 ± 12.5	73.7 ± 11.6
3	76.5 ± 10.6	89.4 ± 6.3*$^{+}$
4	76.0 ± 7.5	90.0 ± 6.5*$^{+}$
5	76.6 ± 8.5	89.1 ± 10.5*$^{+}$
6	79.5 ± 12.0	76.8 ± 9.6
7	76.6 ± 8.1	73.3 ± 10.6

Values are means ± SD *P<0.05 = Compared with period (1)
$^{+}$P<0.05 = Compared with propofol group

Fig. (1): Changes of HR in two groups during ECT

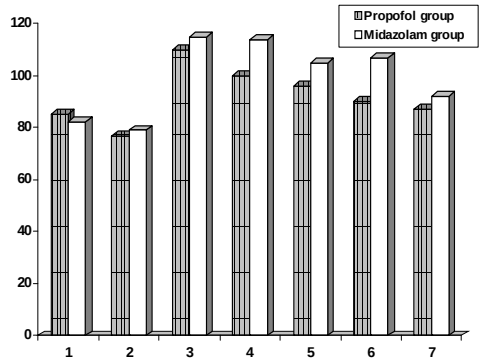
In propofol group, increased MAP was observed from one to two minutes after the electrical shock when compared with the awake status. In midazolam group, increased MAP was observed from 1 to 5 minutes after the electrical shock when compared with the awake status. There was a significant increase of MAP in midazolam group at 2 and 3 minutes after the electrical shock when compared with propofol group (table 9 and fig. 2).

Table (9): Changes of MAP in two groups during ECT.

	Propofol group	Midazolam group
1	85 ± 6	82 ± 7
2	77 ± 7	79 ± 6
3	110 ± 9*	115 ± 11*
4	100 ± 7*	114 ± 10*$^{+}$
5	96 ± 7	105 ± 9*$^{+}$
6	90 ± 8	107 ± 11*
7	87 ± 6	92 ± 8

Values are means ± SD *P<0.05 = Compared with period (1) $^{+}$P<0.05 = Compared with propofol groupMAP = Mean arterial pressure

Fig. (2): Changes of MAP in two groups during ECT



There was no significant change observed in EDA in both groups during the study (table 10).

In propofol group, an increase in ESA and decrease in FAC were observed at 1 minute after the electrical shock when compared with the awake status (table 10).

Table (10): Time course for changes of hemodynamic variables.

		Propofol group	Midazolam group
EDA (Cm^3)	1	8.1 ± 0.49	7.84 ± 0.3
	2	8.17 ± 0.36	7.95 ± 0.39
	3	8.18 ± 0.16	7.96± 0.41
	4	8.2 ± 0.3	7.97 ± 0.31
	5	8.28 ± 0.3	8.17 ± 0.4
	6	8.2 ± 0.32	7.99 ± 0.38
	7	7.98 ± 0.15	7.84 ± 0.39
ESA (Cm^3)	1	3.3 ± 0.47	3.27 ± 0.46
	2	3.5 ± 0.4	3.25 ± 0.49
	3	4.9 ± 0.6*	4.2 ± 0.4*
	4	3.37 ± 0.52	3.4 ± 0.85
	5	3.15 ± 0.23	3.27 ± 0.64
	6	3.2 ± 0.17	3.38 ± 0.64
	7	3.2± 0.15	3.3 ± 0.48
FAC (%)	1	59.1 ± 3.49	57.8 ± 4.5
	2	57.7 ± 4.0	58.8 ± 4.54
	3	57 ± 9.1*	45.8 ± 8.4*
	4	59.1 ± 7.5	48.7 ± 6.4*$^{+}$
	5	57.2 ± 3.95	46.1 ± 5.96*$^{+}$
	6	58.4 ± 4.3	56.1 ± 3.8
	7	59 ± 2.8	57.8 ± 1.92

Values are means ± SD *P<0.05 = Compared with period (1) $^{+}$P<0.05 = Compared with propofol group
EDA = End-diastolic area ESA = End-systolic area
FAC = Fractional area change (%)

In midazolam group, an increase in ESA was observed at 1 minute after electrical shock and decrease in FAC was observed from 1 to 3 minutes after electrical shock when compared with awake status (table 10). There was a significant decrease of FAC in midazolam group from 1 to 3 minutes after the electrical shock when compared with the propofol group (Table 10).

Table (1): Demographic and clinical features of the sample.

		Propofol group (N=15)	Midazolam group (N=15)	P
Age (years)	Range	22-50	22-50	
	Mean±SD	38.9±12.1	37.6±11.7	0.18
Sex	Male	8	4	
	Female	7	11	0.13
Weight (kg)	Range	58-70	60-70	
	Mean±SD	63.8±2.71	66.4±4.2	0.25
Height (cm)	Range	160-180	158-180	
	Mean±SD	168±9.0	167±7.1	0.85
Duration of current MDE (weaks)	Mean±SD	18.0±15.2	16.0±13.7	0.13
Number of previous MDEs	Mean±SD	4.3±2.3	3.9±1.9	0.17
Age of onset of first MDE (years)	Mean±SD	30.2±17.1	31.0±16.8	0.16

MDE, major depressive episode, P>0.05: Non significant, Values as mean±SD, P<0.05: Significant

Table (2): Scores on clinical measures of symptoms.

Measure	Baseline scores (mean±SD)	Patients		Scores at the end of ECT course (mean±SD)	Patients	
		n	%		n	%
HRSD	29.5±15.8*	30	100	6.2±5.9	16	53.33
BDI	35.2±10.4*	30	100	15±9.3	17	56.66

HRSD: Hamilton rating scale for depression (scores from 0-50). BDI: Beck depression inventory (scores from 0-63). *P<0.05

DISCUSSION

Electroconvulsive therapy (ECT) is widely acknowledged as an effective and appropriate acute treatment for major depressive illness[1, 23]. Because the therapy can be completed within 10 minutes, the anesthetics used for ECT should have a short action and a rapid recovery[10]. In addition, because the seizure itself is believed to be important for the efficacy of the therapy, the anesthetics should not interfere with the electrical [seizure22].

In this study we used propofol and midazolam as short acting anesthetics to compare their effects of ECT, regarding time of induction, duration of seizure, recovery time and haemodynamics during ECT session.

The study revealed that, induction time in propofol group was less than midazolam group, and the difference was statistically significant (P<0.05) and this was in accordance with previous study, as the induction was smooth and easy[24].

Duration of seizure in propofol group was greater than with midazolam group and there was significant difference (P<0.05) between the two groups. This is in accordance with previous studies that recommended against using propofol for ECT because of its systematic seizure-shortening effects[25-26], but Fear et al. have shown that reduced seizure duration obtained with propofol is not associated with a reduced therapeutic effect in comparison to that of methohexitone anesthetic agent[21].

Kirkby et al. found that seizure duration was shortened with propofol versus methohexital, but inspite that, the scores on the HRSD were improved to a similar degree[27]. Also, in the present study, the score on clinical measures (HRSD, BDI, CGIS) were improved at the end of ECT course. The significant difference between scores on HRSD and BDI before and after the course of ECT, indicated ECT success in treatment of depressive episodes[2-3, 28-29]. Scores on CGIC after ECT course, indicated that ECT is an effective treatment for severe depression[30-31].

Lesser hemodynamic changes were observed after propofol than after midazolam for ECT. These results were in accordance with the result obtained by Avramov et al., who found that the acute haemodynamic response during ECT procedure is reduced with propofol compared with etomidate, methohexital and thiopental. These results go hand in hand with the results of Yuji et al. [6], who found that a lesser hemodynamic change occurs after propofol anesthesia (1mg/kg) compared with thiopentone anesthesia (2mg/kg) during ECT[32].

These results were also in agree with that the ultrashort acting barbiturate methohexitone is widely used as the anesthetic agent of choice for ECT, propofol is considered an alternative because it is associated with a smaller haemodynamic response during ECT[33].

In this study, propofol was used at a dose of 1mg/kg as a hypnotic drug. Shigeru et al.,

found that the use of a minimally hypnotic dose of propofol (0.75mg/kg) was associated with a seizure duration that was comparable to standard hypnotic doses of methohexital[10]. Also, propofol at a dose of 0.75mg/kg improved haemodynamic stability after ECT[13]. In contrast, Mulier et al. reported that propofol at a dose of 1.4mg/kg reduced systolic arterial blood pressure mainly through its negative inotropic properties[34]. This discrepancy might be attributable to the difference in a propofol dose and in pre-medical drugs such as β-adrenoceptor antagonists or atropine. Recovery time was statistically different between the two groups. Propofol had significantly faster clinical recovery than midazolam with respect to time for the ability to obey commands, orientation, and ability to sit up unaided.

As regard the oxygen saturation, this study showed that there was a decrease in oxygen saturation from 1 to 2 minutes after electrical shock in propofol group and from 1 to 3 minutes after electrical shock in midazolam group. These results were in accordance with the result of Yuji et al. who found that ECT provokes a rise in plasma catecholamiens and induce a considerable increase in myocardial oxygen demand[6].

The study showed no change in HR in the propofol group. This result go hand in hand with the result of Kadoi et al. who found that systematic haemodynamics is stable during ECT using propofol compared with thiopentone[35].

An increase in MAP was observed after ECT in midazolam group from 1 to 5 minutes after electrical shock. In contrast there was a lesser change in MAP from 1 to 2 minutes after electrical shock in propofol group. This result was in accordance with Boey and Lai who proved that the stable systemic hemo-dynamics during ECT was noticed by using propofol[37]. Also these results go hand in hand with Kadoi et al. who found that propofol might cause greater suppression of sympathetic hyperactivity during ECT. This would result in reduced afterload, which might facilitate cardiac output[35].

No change of EDA was observed in either group prior to or after the administration of anesthetic drugs. This might be in part attributable to the dosages used. The doses (propofol 1mg/kg and midazolam 0.1mg/kg) used were not enough to affect preload. This result was in agreement with Gare et al. who found that sedation with midazolam or propofol does not affect indices of LV diastolic performance in healthy patients and those with preexisting diastolic dysfunction[15].

The left ventricular systolic performance assessed by echocardiography was transiently decreased after the electrical shock in both groups. These results were in accordance with the results of Selvin who reported that ECT provokes sympathetic hyper-activity that induces an increase in systemic vascular resistance and afterload and hypertension[37].

This study showed that both groups showed a differential time course in changes to FAC after ECT.

Here we did not examine all segments of LV because we focused on systolic performance during ECT. Although this study realizes that automated border detection might underestimate or overestimate EDA and ESA, echocardiographic automated border detection is a promising non invasive assessment of cardiac performance[38].

CONCLUSION

Electroconvulsive therapy is still highly effective treatment for severe depression and several psychiatric disorders. Propofol appears to be a safe anesthetic for ECT. It provides excellent haemodynamic stability, quick induction and early recovery during procedure and the duration of seizure was shorter with midazolam. So, propofol can be used safely in cardiac and elderly patients.

REFERENCES

1. American Psychiatric Association Committee on Electroconvulsive Therapy The practice of electroconvulsive therapy: Recommendation for treatment, training and privileging. 2001. Washington, DC: APA.
2. Fink M. Electroconvulsive therapy update: recognizing and treating psychotic depression. J Clin Psychiatry 2003;64(3):232-34.
3. Khalid N, Atkins M, Tredget J et al. The effectiveness of ECT in treatment-resistant depression: A naturalistic study. J ECT 2008;24(2):141-45.
4. Thompson JW, Weiner RD, Myers CP Use of ECT in the united states in 1975 and 1986. Am J Psychiatry 1994;151:1657-61.
5. Esel, Ertugurl, Kose, et al. The effects of ECT on GABergic function in major depressive patients. J ECT 2008;24(3):224-28.
6. Yuji K, Shigeru S, Shinjiro S et a. Electroconvulsive therapy impairs systolic performance of the left ventricule. Can J Anaesth 2001;48:405-8.
7. Castelli I, Steiner LA, Kaufmann MA. Comparative effect of esmodal and labetalol to attenuate hyper-dynamic states after electroconvulsive therapy. Anesth Analg 1995;80:557-61.
8. Fu W, Stool LA, White PF et al. Acute hemodynamic responses to electroconvulsive therapy are not related to the duration of seizure activity. J Clin Anesth 1997;9:653-57.
9. Weigner MB, Partridge BL, Hanger R et al. Prevention of the cardio-vascular and neuroendocrine response to electroconvulsive therapy: Effective-ness of pretreatment regimens on hemo-dynamics. Anesth Analg 1991;73:556-62.
10. Shigeru S, Yuji R, Takeshi N et al. The comparative effects of propofol versus thiopental on middle cerebral artery blood flow velocity during electroconvulsive therapy. Anesth Analg 2000;91:1531-36.
11. Auriacombe M, Grabot D, Lincheneau PM et al. Use of midazolam for ECT anesthesia: effect on anti-depressive efficacy and seizure duration preliminary finding. Eur Psychiatry 1995;10(6):312-6.
12. Harti A, Hmamouchi B, Idali H Anesthesia for electroconvulsive therapy: propofol versus thiopental. Encephale 2001;27:217-21.
13. Fredman B, Etienne J, Smith I et al. Anesthesia for electroconvulsive therapy: Effects of propofol and methohexital on seizure activity and recovery. Anesth Analg 1994;79:75-9.
14. Abrahms R. Electroconvulsive therapy. 4th ed New York: Oxford University Press 2002;156.
15. Gare M, Parai LA, Milosavljevic D et al. Conscious sedation with midazolam or propofol does not alter left ventricular diastolic performance in patients with preexisting diastolic dysfunction: a transmitral and tissue Doppler trans-thoracic echocardiography study. Anest Analgesia 2001;93(4):865-71.
16. First M, Spitzer R, Gibbon M et al. Structured Clinical Interview for DSM-IV Axis I Disorder (SCID). 1995 New York, New York State Psychiatric Institute, Biometrics Research.
17. Hamilton M. A rating scale for depression. J Neurol Neurosurg Psychiatry 1960;23:56-62.
18. Beck AT, Steer RA, Garbin MG. Psychometric properties of the Beck Depression Inventory: Twenty five years of evaluation. Clin Psych Rev 1988;8:77-100.
19. Guy W. Clinical Global Impression Scale. ECDEU Assessment Manual for Psychopharmacology, revised national institute of Mental Health Rockville, MD 1976.
20. D'Elia G & Raotma H. Is unilateral ECT less effective than bilateral ECT? B J Psychiatry 1975;126:83-9.
21. Fear CF, Littlejohns CS, Rouse E et al. Propofol anesthesia in electroconvulsive therapy. Reduced seizure duration may not be relevant 1994.
22. Gaines GY, Rees DI. Anesthetic considerations for electroconvulsive therapy. South Med J 1992;85:469-82.
23. UK ECT Review Group Efficacy & safety of electroconvulsive therapy in depressive disorders: a systematic review & meta-analysis. Lancet 2003;362:799-808.
24. Loimer N, Hofmann P, Chaudry HR. Midazolam shortens seizure duration following electroconvulsive therapy. J Psychiatry Res 1992;26:97-101.
25. Rampton AJ, Griffin RM, Stuart CS et al. Comparison of methohexital & propofol for electroconvulsive: effects on hemodynamic responses & seizure duration. Anesthesiology 1989;70:412-17.
26. Simpson KH, Halsal PJ, Carr CME et al. Propofol reduces seizure duration in patients having anesthesia for electroconvulsive therapy. Br J Anaesth 1988;61:343-44.
27. Kirkby KC, Beckett WG, Matters RM. Comparison of propofol and methohexitone in anesthesia for ECT: Effect on seizure duration and outcome. Aust NZ J Psychiatry 1995;29:299-303.
28. Conca A, Germann R et al. Etomidate vs. thiopentone in electroconvulsive therapy. An interdisciplinary challenge for anesthesiology and psychiatry. Pharmacopsychiatry 2003;36(3):94-7.
29. Nguyen TT, Chhibber AK et al. Effect of methohexitone and propofol with or without elfantil on seizure duration & recovery in electroconvulsive therapy. Br J Anaesth 1997;79(6):801-3.

30. Eranti SV & Mcloughlin MD. Electroconvulsive therapy-state of the art. Br J Psychiatry 2003;182:8-9.
31. Keith R. The practice of electroconvulsive therapy: Recommendations for ECT treatment training & privileging (2nd ed). JECT 2002;18(1):58-9.
32. Avramov MN, Husain MM, White PF. The comparative effects of methohexital, propofol and etomidate for electroconvulsive therapy. Anesth Analg 1995;81:596-602
33. Wajima Z, Shiga T, Yoshirawa T et al. Propofol alone, sevofluran alone, and combined propofol sevoflurane anesthesia in electroconvulsive therapy. Anesth Intensive Care 2003;31:396-400.
34. Mulier JP, Wouters PF, Van Aken H et al. Cardio-dynamic effects of propofol in comparison with thiopental assessment with transesophageal echocardiography approach. Anesth Analg 1991;72:28-35.
35. Kadoi Y, Saito S, Ide M, et al. The comparative effects of propofol versus thiopentone on left ventricular function during electroconvulsive therapy. Anesth Intensive Care 2003;31:172-75.
36. Boey WK & Lai FO. Comparison of propofol & thiopentone as anesthetic agents for electroconvulsive therapy. Anesthesia 1990;45:623-28.
37. Selvin BL. Electroconvulsive therapy. Anesthesiology 1987; 67:367-85.
38. Hillel Z & Thys D. Monitoring left ventricular performance. In: Omoto R, Oka Y (eds) Transoesophageal Echocardiography Tokyo: Shindan-to Chiryo Co. Ltd; 2000;57-70.

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