

A COMPARISON OF THE EFFECTS OF PROPOFOL AND THIOPENTONE IN MODIFIED ELECTROCONVULSIVE THERAPY

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F.Y.Göğüş, M.D. * / O. Kanbak, M.D. ** / Y.Yazgan, M.D. ***

* *Professor, Department of Anesthesiology and Reanimation, Faculty of Medicine, Marmara University Istanbul, Türkiye.*

** *Specialist, Department of Anesthesiology, Numune Hospital, Ankara, Türkiye.*

*** *Specialist, Department of Psychiatry, Faculty of Medicine, Marmara University, Istanbul, Türkiye.*

SUMMARY

Electroconvulsive therapy has recently been applied under general anesthesia for the treatment of many psychiatric diseases as depression. In our study, we aimed to compare the induction and recovery times, duration of convulsion, arterial blood pressure and heart rate values in 10 psychiatric patients, 5 of whom were female. Electroconvulsive therapies are induced with thiopentone sodium or propofol. The recovery time was found to be 17.45 ± 1.97 minute for propofol and 24.15 ± 2.00 minute for thiopentone sodium. The mean convulsion time for propofol being 20.85 ± 2.5 second was significantly shorter than that for thiopentone sodium being 26.6 ± 2.1 second. A significant increase in arterial blood pressure after electroconvulsive therapy were recorded for both agents, yet the increase was more with thiopentone sodium. Finally, because all the patients included in our study showed a good prognosis, we believe that propofol might be beneficial for those patients who have cardiovascular problems; however concerning the effect of duration of convulsion on therapy, it was concluded that further studies must be started on a broader spectrum of patients.

Key Words: Modified Electroconvulsive Therapy, Propofol, Thiopentone Sodium.

INTRODUCTION

Electroconvulsive therapy (ECT) which is an accepted treatment for depression and other psychiatric disorders since 1938 has become a safe and simple method, called modified ECT after 1960's with the introduction of short acting barbiturates and depolarizing muscle relaxants (1). However, the relation between the seizure duration and the antidepressant effect has not been explained well yet. It is generally believed that the generalized convulsion occurred is essential for the treatment (2). On the other hand the autonomic stimulation caused by the electrical seizure may lead to cardiovascular changes as systemic hypertension. Thiopentone sodium is a short acting barbiturate and propofol is a short acting intravenous anesthetic agent suitable for

induction and maintenance of anesthesia.

In our study we aimed to compare the effects on induction, recovery period and seizure duration of thiopentone sodium and propofol on ECT patients.

METHODS AND MATERIALS

10 Patients (5 female) of ASA grade I-III, aged between 19 and 71 years have been entered into trial. By anesthetising the patients on four occasions a total of 40 ECT administrations have been recorded. The patient's ages, sexes and body weights are shown in table I. No change was made in their psychotropic drug therapy. Between each ECT an interval of two days has been given. In the patients with no premedication the base line arterial pressure and heart rates were recorded following electrocardiographic monitorization. An initial dose of atropine sulphate 0.5 mg was given intravenously followed by the anesthetic agent. Induction was maintained by either propofol or thiopentone sodium titrated at the rate of 2 ml every 5 seconds until the loss of eyelash reflex. After inflating a cuff on the other arm to above systolic pressure in order to isolate the forearm muscles from the effects of muscle relaxant, succinylcholine 0.5 mg/kg intravenously was administered. Ventilation was supported with 100 % of oxygen via a face mask. The ECT stimulus ranging between 29.80 watt/sec and 59.61 watt/sec for 2 or 4 seconds was delivered from an Ectron Duopulse constant current machine using bilateral temporoparietal electrodes following a hyperventilation period for 1 minute. The interval from the beginning of ECT to the termination of all clonic movements were recorded. The patients were taken into the recovery room after verbal communication was succeeded.

Before and after induction and after ECT systolic, diastolic blood pressure, heart rate values of the patients were recorded. Loss of eyelash reflex was accepted as induction time, time to open eyes and to speak was accepted as full recovery time. The data obtained were compared using Student's t test and correlation analyses were performed.

TABLE I. Characteristics of the patients received ECT

SEX		AGE		BODY WEIGHT (kg)	
Male	Female	Range	Mean±SD	Range	Mean±SD
5	5	19-71	42.00±20.00	49-70	59.40±7.01

RESULTS

Mean propofol and thiopentone doses given during induction were 1.99 ± 0.12 mg/kg and 5.59 ± 0.27 mg/kg respectively. Induction and recovery times for both agents were shown in table II. In the thiopentone group time from induction to full recovery was significantly longer than propofol ($p<0.01$). Pre, post induction and post ECT systolic and diastolic blood pressure and heart rate values are shown in table III. The significant reduction in systolic and diastolic blood pressure in the propofol group after induction ($p<0.05$, $p<0.06$) was not seen in thiopentone group. Although after ECT a significant increase in blood pressure values ($p<0.001$ and $p<0.00001$) was observed by both agents, this

increase was more significant with thiopentone. While no rhythm changes were recorded by propofol, two patients in the thiopentone group showed ventricular extrasystoles.

It was recorded that the mean seizure duration by propofol was significantly shorter than by thiopentone ($p<0.05$) (Table IV).

On the other hand while there was no correlation between the dose of propofol and induction and recovery times, there was a moderate positive relation between thiopentone's dose and those times. And after all, a weak negative correlation was found between the seizure duration and induction time for propofol ($p<0.05$).

TABLE II. Induction and recovery times of the patients

	PROPOFOL (n=20)	THIOPENTONE (n=20)
Induction dose (mg/kg)	1.99 ± 0.12	5.59 ± 0.27
Induction time (sec)	86.00 ± 3.41	77.50 ± 5.73
Induction to eyes opening time (min)	$10.80\pm 1.11^*$	16.00 ± 1.32
Induction to spontaneous speaking time (min)	$14.50\pm 1.40^{**}$	20.50 ± 1.69
Induction to full recovery time (min)	$17.45\pm 1.97^{***}$	24.15 ± 2.00
* $p<0.002$		
** $p<0.004$		
*** $p<0.01$		

TABLE III. Alterations in blood pressure and heart rate values of the patients

	PROPOFOL	THIOPENTONE
Systolic blood pressure (mmHg)		
Before induction	123 ± 7	120 ± 3
After induction	$110\pm 6^*$	116 ± 1
After ECT	$145\pm 9^{**}$	$153\pm 6^{***}$
Diastolic blood pressure (mmHg)		
Before induction	80 ± 4	80 ± 3
After induction	$75\pm 3^{****}$	81 ± 3
After ECT	$88\pm 4^{*****}$	$95\pm 3^{*****}$
Heart rate (beat/min)		
Before induction	90 ± 5	91 ± 5
After induction	85 ± 3	92 ± 4
After ECT	$79\pm 3^{***}$	86 ± 4
* $p<0.05$		
** $p<0.001$		
*** $p<0.00001$		
**** $p<0.06$		
***** $p<0.04$		
***** $p<0.005$		

TABLE IV. Convulsion durations of the patients

ECT administration	Convulsion duration (sec)	
	Propofol	Thiopentone
1	23	22
2	12	44
3	14	22
4	29	34
5	25	25
6	48	15
7	24	26
8	12	25
9	20	35
10	23	35
11	12	30
12	14	40
13	30	24
14	25	19
15	30	22
16	22	13
17	10	38
18	38	14
19	6	10
20	0	39
Mean duration	20.85±2.51	26.60±2.10*

*p<0.05

DISCUSSION

In recent years to minimize the possible ECT complications as long bone and vertebrae fractures, tongue, eye and other soft tissue traumas ECT has been applied under general anaesthesia. Most of the ECT candidates receive drug treatment consisting of tricyclic antidepressants or benzodiazepines previously (3).

Therefore, the chosen anesthetic agent has to be compatible with those drugs. Animal studies show no interaction between propofol and tricyclic antidepressants or monoamineoxidase inhibitors (4). It is known that the increased plasma catecholamine level as a result of autonomic nervous system stimulation during ECT may lead to acute increases in arterial pressure and heart rate which can be hazardous for patients having cardiovascular disorders (5-7). Hemodynamic studies of propofol show a reduction in systemic arterial pressure due to a possible decrease in cardiac out-put (8,9). In their studies, Rouse (10) and Rampton (11) have examined the effects of propofol and methohexitone on hemodynamic changes following ECT and found that propofol has caused a mean increase in systolic arterial pressure less than 25 mmHg compared with methohexital. In our study, the increase in mean arterial pressure caused by propofol was less than 10 mmHg compared with thiopentone. On the other hand the lower post ECT heart rate value of propofol may have a favorable

effect on myocardial oxygen demand.

The vast majority of patients undergoing ECT are ambulatory, so the chosen anesthetic method is desired to have a rapid and smooth recovery. Grant et al (12) who examined the effects of propofol and thiopentone as an induction agent on outpatients scheduled for small surgical procedures found the mean recovery time as 5.80 ± 0.45 min and 10.60 ± 0.74 min respectively. In our study the mean recovery times were 17.45 ± 1.97 min for propofol and 24.15 ± 2.00 min for thiopentone sodium. This longer duration of recovery we found could be due to the postictal phase following ECT. Herbert (13) who studied the postoperative mental abilities too, pointed out that the return of performance was more rapid with propofol than with thiopentone.

However the mechanism of treatment of ECT is not known well yet it is generally accepted that the seizure duration is related with the efficiency of therapy (7,14). The duration of seizure is influenced by many factors such as the type of electrical stimulus, blood gas pressures, age, concurrent drug therapy and induction agents (6). In our study, the seizure duration for propofol and thiopentone sodium were found as 20.85 ± 2.51 sec and 26.60 ± 2.10 sec respectively, there was a significant difference between the two durations. This is supported in a 19 years old female patient by observing no convulsions following the first treatment with propofol, while a seizure occurred for 24 sec following the second treatment with thiopentone sodium. In one of the two

studies comparing methohexital and propofol, Rampton (10) reported the mean seizure durations as 30.90 ± 2.80 sec and 17.90 ± 2.50 sec, while Rouse (10) found as 30.8 sec and 18 sec respectively. Dwyer et al (15) measured seizure duration both clinically and with a cerebral function monitor and found that the seizure durations were 25% shorter with propofol than with methohexital. The longer seizure duration with methohexital in these studies may be due to the excitatory effect of the agent on central nervous system (5). But when compared with thiopentone which has a known anticonvulsant effect the significant shortness of seizure duration may indicate some additional anticonvulsant action of propofol. However, in clinical practice there are conflicting reports about the convulsant and anticonvulsant action of propofol (16,17).

In our study, we pointed out that the induction time gets longer, the seizure duration gets shorter with propofol, but we could not show a relationship with thiopentone sodium. Rampton et al (12) also reported that the shorter the induction to ECT interval the shorter the seizure duration and these two findings confirm each other.

In conclusion, since all the patients examined in our study were treated with modified ECT, propofol may have a use in ECT of the patients whose cardiovascular disorders existing as a risk factor but further study is needed to determine whether its anticonvulsant action has any effects on the therapeutic efficiency of ECT with a larger patient group.

REFERENCES

1. Gaines GY III, Rees DI. *Electroconvulsive therapy and anesthetic considerations. Anesth Analg* 1986;65:1345-1356.
2. Ottoson JO. *Seizure characteristics and therapeutic efficiency in electroconvulsive therapy: an analysis of the antidepressive efficiency of grand mal and lidocaine modified seizures. J Nerv Ment Dis* 1962;135:239-351.
3. Mc Cleave DJ, Blakemore WB. *Anesthesia for electroconvulsive therapy. Anesth and Intens Care* 1975; 3: 250-250.
4. Glen JB, Hunter SC, Blackburn TP, Wood P. *Interaction studies and other investigations of the pharmacology of propofol (Diprivan). Postgraduate Medical Journal* 1985;61(Suppl 3) 7-14.
5. Anton AH, Uy DS, Redderson CL. *Autonomic blockade and the cardiovascular and catecholamine response to electroshock. Anesth Analg* 1977;56:48-54.
6. Selvin BL. *Electroconvulsive therapy. Anesthesiology* 1987;67:367-385.
7. Kendell RE. *The present status of electroconvulsive therapy. British Journal of Psychiatry* 1981;39:265-283.
8. Lassen R, Rathgeber J. *Effects of propofol on cardiovascular dynamics and coronary blood flow in geriatric patients. A comparison with etomidate. Anesthesia* 1988;43 (Suppl) 25-31.
9. Coates DP, Prys Roberts J. *Propofol (Diprivan) by intravenous infusion with nitrous oxide: dose requirements and haemodynamic effects. Postgraduate Medical Journal* 1985;61 (Suppl 3): 76-79.
10. Rouse EC. *Propofol for electroconvulsive therapy. A comparison with methohexitane. Anesthesia* 1988;43 (Suppl):61-64.
11. Rampton AJ, Griffin RM. *Comparison of methohexital and propofol for electroconvulsive therapy: Effects on hemodynamic responses and seizure duration. Anesthesiology* 1989;70:412-417.
12. Grant IS, Mackenzie N. *Recovery following propofol (Diprivan) anesthesia-a review of three different anesthetic techniques. Postgraduate Medical Journal* 1985;61 (Suppl 3): 133-137.
13. Herbert M, Matin SW. *Recovery of mental abilities following general anesthesia induced by propofol or thiopentone. Postgraduate Medical Journal* 1985;61 (Suppl 3):132.
14. Robin A, De Tissera S. *A double blind controlled comparison of the therapeutic effects of low and high energy electroconvulsive therapies. Br J Psychiatry* 1982;141:357-366.
15. Dwyer R, Mc Caughey W, Lavery J. *Comparisons of propofol and methohexitane as anesthetic agents for electroconvulsive therapy. Anesthesia* 1988;43:459-462.
16. Hodkinson BP. *Propofol and the electroencephalogram. Lancet* ii. 1987;1518.
17. Wood PR, Brownie QPR. *Propofol infusion for the treatment of status epilepticus (letter) Lancet* iii 1988;480-481.