A Comparative Study between Intravenous Thiopentone Sodium and Propofol on the Cardiovascular Changes after Electroshock in Patients Undergoing Electroconvulsive Therapy

Roshan M. Shende[1], Santosh N. Bodkhe[2], Sonal Agrawal[3], Pankaj R. Bhople[2]

ABSTRACT

Introduction: Electroconvulsive therapy is also known as Electroshock or Shock therapy or Electroplexy. Objectives: To study the cardiovascular changes between intravenous Thiopentone Sodium and Propofol groups after electroshock in patients undergoing ECT.

Material and methods: This study was carried out on psychiatric patients, after clearance from the ethics committee. Each patient underwent a series of prescribed electroconvulsive therapies. In this study, two treatment groups were included: ECT with Thiopentone Sodium (Group A) and ECT with Propofol (Group B), as induction agents. A total of 60 cases were included in this study. Analysis of the quantitative was done by the Unpaired t - test using the statistical software: SPSS.

Result: Increase in then mean heart after induction in both the groups was statistically not significant. After 1 min, the rise in the mean heart rate post electroconvulsive therapy was significant, which then gradually decreased over the study period but never reached the base line. These changes in the mean heart rate were not statistically significant. There was also no significant difference in the systolic blood pressure between two groups, with respect to the baseline values. The increase in systolic blood pressure immediately after induction, i.e., pre-electroconvulsive therapy and after electroconvulsive therapy, was statistically significant in both the groups (p value <0.05). Increase in the systolic blood pressure was more marked in the Thiopentone Sodium group and it remained significant till the end of 45-minutes after induction.

Conclusion: Propofol, as induction agent, is better with respect to heart rate and systolic blood pressure.

Keywords: Propofol, Thiopentone Sodium, Electoconvulsive Therapy, Heart rate, Blood Pressure.

INTRODUCTION

Electroconvulsive therapy is also known as Electroshock or Shock therapy or Electroplexy.[1,2]

It was introduced in clinical practice based on the finding that psychiatric symptoms were improved after seizure in the patient suffering from both schizophrenia and epilepsy. Seizure activity produced by ECT is considered as the therapeutic aspect of this form of treatment, but is accompanied by untoward physiologic consequences, mainly cardiovascular responses and cerebral in nature.[3] During the few seconds following ECT stimulus, there may be a temporary drop in the blood pressure. This may be followed by a marked increase in heart rate, which may then lead to a rise in blood pressure. Upon awakening, a patient may experience a brief period of confusion, headache or muscle stiffness. Since no surgical procedure accompanies ECT, any morbidity or mortality is especially unfortunate, and may be consequent only to treatment or to the anesthetic. Therefore, the practicing anaesthesiologist must be prepared to manage these patients in a fashion that promotes effective seizure activity and simultaneously attenuates the physiologic effects of therapy.[4]

Propofol has been recommended for ECT anesthesia, because it is reported to provide more stable conditions due to its rapid onset of action and better quality of recovery compared with barbiturate anesthesia.[5]

Propofol has been found to have more potent anti-convulsant effects during ECT than other IV anesthetics.[6-8] However, the use of a minimal hypnotic dose of Propofol (0.75 mg/kg) has been associated with a seizure duration that is comparable to the standard hypnotic doses of Methohexital.

When electroconvulsive therapy was introduced in the treatment of psychiatric disorders, it was often conducted just under sedation; without the use of general anesthesia and muscle relaxants. Hence, it was associated with physical and psychological trauma to patient. With the use of general anesthesia and muscle relaxants, though the incidence of physical and psychological trauma was reduced, but still it is frequently associated with cardiovascular instability, cerebrovascular changes, neuroendocrine responses and side effects of general anesthesia. Study objectives were to study the cardiovascular changes between intravenous Thiopentone Sodium and Propofol groups after electroshock in patients undergoing ECT.

MATERIAL AND METHODS

This was a prospective, randomized, single blinded (patient), non-crossover study. It was carried out after obtaining clearance from the Institutional Ethics Committee. This study was carried out on psychiatric patients who attended the OPD and were hospitalized. The patients belonged to ASA grade I or II, and were on medication for the psychiatric disorder. A written valid informed consent was taken from their close relatives, as the patients were mentally subnormal and/or having psychiatric disorder, interfering with their ability to understand the nature and consequences of anesthesia and the procedure. The

[1]Assistant Professor, [2]Associate Professor, [3]Junior Resident – I, Department of Anesthesiology, SVNGMC, Yavatmal, Maharashtra, India

Corresponding author: Dr. Santosh N. Bodkhe, Department of Anaesthesiology, SVNGMC, Yavatmal Pin- 445001, Maharashtra, India.

How to cite this article: Roshan M. Shende, Santosh N. Bodkhe, Sonal Agrawal, Pankaj R. Bhople. A comparative study between intravenous thiopentone sodium and propofol on the cardiovascular changes after electroshock in patients undergoing electroconvulsive therapy. International Journal of Contemporary Medical Research 2017;4(6):1382-1385.

study was conducted on adult patients in the age group of 18 to 45 years. Each patient underwent a series of prescribed electroconvulsive therapies. In this study, the patients were divided by simple randomization, in two study groups: ECT with Thiopentone Sodium (Group A) and ECT with Propofol (Group B), as the induction agents. A total 60 cases were included in this study. The inclusion criteria consisted of: ASA grade I and II (psychiatric patients without any major illness),age group of 18 to 45 years, no history of drug allergies or anaphylaxis while the exclusion criteria were ASA Grade III or IV, Age<18 years and >45 years, pregnancy, any history of allergies and/or anaphylaxis. The anesthetic technique was standardized. All patients' current medications were recorded and kept constant throughout the study.

STATISTICAL ANALYSIS

Analysis of the quantitative data was done by the Unpaired t - test using the statistical software: SPSS software; and the following were considered: P value>0.05-Not significant, P value<0.05-significant, P value<0.01-very significant, P value<0.001-highly significant.

RESULTS

The data in table-1 shows significant differences in the mean heart rate between the two groups with respect to the base line values. Increase in the mean heart after induction, in both the groups, was statistically not significant. Post electroconvulsive therapy, after 1 minute, the rise in the mean heart rate was significant, which then gradually decreased over the study period but never reached the base line. These changes in the mean heart rate were not statistically significant.

The data in table-2 shows that there was no significant difference in the systolic blood pressure between the two groups, with respect to the baseline values. The increase in systolic blood pressure immediately after induction, i.e., pre-electroconvulsive therapy and after electroconvulsive therapy, was statistically significant in both the groups (p value <0.05). Increase in the systolic blood pressure was more marked in the Thiopentone Sodium group and it remained significant till the end of 45-minutes after induction.

DISCUSSION

Electroconvulsive therapy is widely used as treatment modality in psychiatric patients all over the world, in disorders like major depression and schizophrenia. Initially electroconvulsive therapy was done under sedation or without general anesthesia, but nowadays it is conducted under general anesthesia, for the added safety and comfort of patients.

ECT induced cardiovascular changes with a parasympathetic-sympathetic sequence may be hazardous in patients with severe cardiovascular disease. The commonest causes of mortality associated with ECT are the acute changes in heart rate and

Heart rate	Groups				Unpaired T-test applied		
	Group A		Group B		T-value	P-Value	Significance
	Mean	SD	Mean	SD			
Pre-induction	78.73	11.05	74.87	21.47	0.877	0.384	Not Significant
Induction	84.30	11.80	83.20	11.19	0.371	0.712	Not Significant
Pre-ECT	96.70	12.57	92.97	9.44	1.301	0.199	Not Significant
Post-ECT-1	100.97	9.43	97.07	9.27	1.615	0.112	Not Significant
Post-ECT-2	100.7	11.55	94.77	9.11	1.973	0.053	Not Significant
Post-ECT-3	100.20	11.81	94.13	10.22	2.127	0.038	Significant
Post-ECT-4	95.93	11.17	92.70	9.18	1.225	0.226	Not Significant
Post-ECT-5	91-53	9.67	89.60	9.22	0.793	0.431	Not Significant
Post-ECT-10	90.43	9.13	85.57	17.57	1.346	0.183	Not Significant
Post-ECT-15	89.77	9.76	88.07	9.03	0.701	0.486	Not Significant
Post-ECT-30	89.53	8.69	85.40	10.05	1.704	0.094	Not Significant
Post-ECT-45	88.40	8.30	85.53	9.99	1.209	0.231	Not Significant

Table-1: Comparison of the heart rate at various time intervals between Group A and Group B

Systolic BP	Groups				Unpaired T-test applied		
	Group A		Group B				
	Means	SD	Mean	SD	T-value	P-value	Significance
Pre-induction	115.33	9.37	114.67	10.08	0.265	0.792	Not Significant
Induction	112.40	8.36	112.13	9.28	0.117	0.907	Not Significant
Pre-ECT	119.93	6.82	112.13	11.10	3.279	0.002	Significant
Post ECT-1	125-97	23.61	120.27	10.38	1.210	0.231	Not significant
Post ECT-2	127.33	10.63	120.73	11.68	2.289	0.026	Significant
Post ECT-3	125.47	10.56	118.67	10.83	2.463	0.017	Significant
Post ECT-4	122.87	10.43	116.80	11.89	2.100	0.040	Significant
Post ECT-5	121.47	10.74	114.53	10.36	2.545	0.014	Significant
Post ECT-10	120.47	9.35	112.53	9.95	3.049	0.003	Significant
Post ECT-15	119.47	9.57	113.67	8.77	2.447	0.017	Significant
Post ECT-30	118.47	8.64	113.47	7.88	2.342	0.023	Significant
Post ECT-45	120.73	7.42	113.27	8.40	3.650	0.001	Significant

Table-2: Comparison of the systolic blood pressure at various time intervals between group A and group B

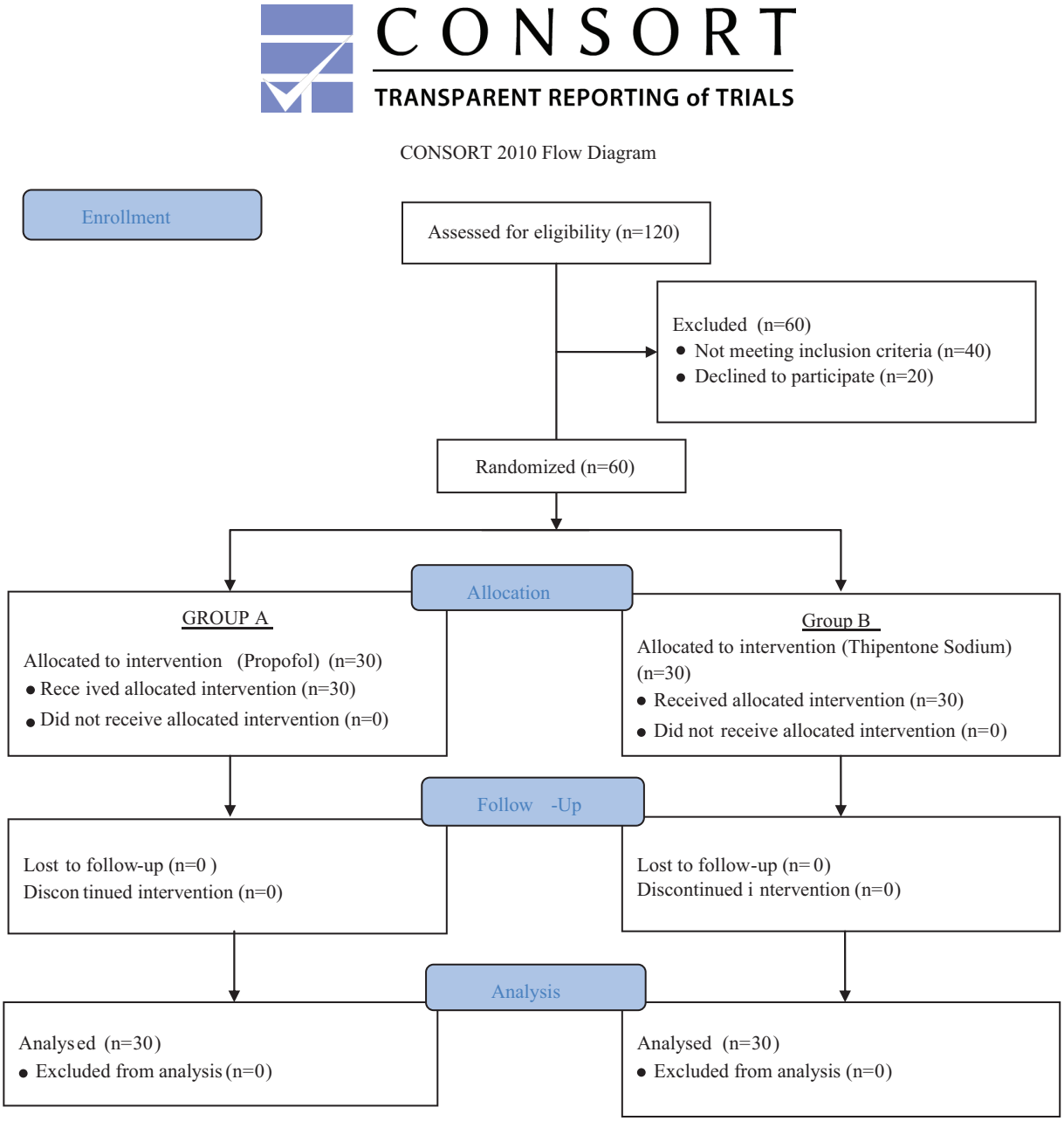

Figure-1: CONSORT 2010 Flow Diagram

blood pressure that follow ECT. In clinical practice, many different strategies have been advocated for the modification of cardiovascular responses to ECT, like the use of beta blockers[9,10], calcium channel blockers[11], fentanyl and lignocaine.[12,13] Use of different induction agents is one such strategy employed to obtund the cardiovascular responses related to ECT. Propofol is a, relatively, new hypnotic agent in our country and there was a need of its comparison with the established ones, in our local population, to facilitate the safe conduct of the procedure.

In this study, it was found that in the Thiopentone Sodium group (Group A) of patients, after induction, the increase in mean heart rate was from 78.73±11.05 beats/min to 96.70±12.57. After giving electroconvulsive therapy, there was a continuous rise in the heart rate. It was maximum (100.97±9.43 beats/min) at 1st minute, then started declining, but did not return to the base line even at the end of 45 min (88.40±8.30).

In the Propofol group (Group B) of patients, after induction, the increase in mean heart rate was from 74.87±21.47 beats/min to 92.97±9.44. After giving electroconvulsive therapy, there was a continuous rise in the heart rate. It was maximum (97.07±9.27 beats /min) at 1stminute, then started declining, but did not reach the basal line even at the end of 45 min (85.53±9.99).

Thus, it was observed that in the Propofol group (Group B) of patients, the rise in the heart rate after electroconvulsive therapy, was less than in the Thiopentone Sodium group (Group A) of patients. However, it was statistically not significant ($p>0.05$).

W.K Body and F.O lai[25] had studied Thiopentone Sodium and Propofol as induction agents for electroconvulsive therapy and found that heart rate in the Propofol group was lower than the control at the fourth minute interval, by 3.1 beats/minute. Propofol causes bradycardia after induction, which was beneficial to blunt the rise in heart rate. However, in this study,

the same was not observed.

In the Thiopentone Sodium group (Group A) of patients, after electroconvulsive therapy, there was a rise in the systolic blood pressure. The rise in systolic blood pressure was more at the end of 1 min after electroshock. At that time, the systolic blood pressure was 125.97±23.61 mm Hg. Thereafter, the systolic blood pressure started decreasing but never reached the base line value till the end of 45 min (120.73±7.42).

In the Propofol group (Group B) of patients, after electroconvulsive therapy, there was a rise in the systolic blood pressure. The rise in the systolic blood pressure was more at the end of 1 min after the electroshock. At that time, the systolic blood pressure was 120.73±10.38. Thereafter, the blood pressure started decreasing and reached near the base line value at the end of 15 min (113.67±8.77).

W.K. body and F.O Lal[15] showed that at one minute after induction, only the systolic blood pressure changed by an average of 3.3±1.3 mm Hg in the Propofol group. The increase in systolic and diastolic blood pressure was significantly greater than the Thiopentone Sodium group.

Villolango A et al[14] studied the cardiovascular responses and anesthetic recovery in the electroconvulsive therapy with Propofol or Thiopentone Sodium. They concluded that the cardiovascular response to electroconvulsive therapy, i.e., the increase in systolic and diastolic blood pressure and heart rate after electroshock, were less marked with Propofol than Thiopentone sodium.

Limitations

Firstly, the results cannot be generalized because the study population was limited to the OPD attendance and hospitalizations in a specific area. So, it is not representative of the whole population. Secondly, due to the limitations of resources, arrhythmias couldn't be monitored during the ECT and in the post-ictal phase. Also the blood enzyme levels for myocardial trauma could not be monitored.

CONCLUSION

Thus, it can be concluded that Propofol causes lesser hemodynamic instability as compared to Thiopentone Sodium during ECT and in the post-ictal phase. Hence, Propofol, as induction agent, is better with respect to heart rate and systolic blood pressure.

REFERENCES

1. Ding Z, White PF. Anesthesia for electroconvulsive therapy. AnesthAnalg. 2002;94:1351–64.
2. Geretsegger C, Rochowanski E, Kartnig C, Unterrainer AF. Propofol and methohexital as anesthetic agents for electroconvulsive therapy (ECT): A comparison of seizure-quality measures and vital signs. J ECT. 1998;14:28–35.
3. Villalonga A, Bernardo M, Gomar C, Fita G, Escobar R, Pacheco M. Cardiovascular response and anesthetic recovery in electroconvulsive therapy with propofol or thiopental. ConvulsTher. 1993;9:108–11.
4. Body WK, Lai FO. Comparison of propofol and thiopentone as anaesthetic agents for electroconvulsive therapy. Anaesthesia. 2007;10:1365–2044.
5. Avramov MN, Husain MM, White PF. The comparative effects of methohexital, propofol and etomidate for electroconvulsive therapy. AnesthAnalg. 1995;81:596–602.
6. Geretsegger C, Rochowanski E, Kartnig C, Unterrainer AF. Propofol and methohexital as anesthetic agents for electroconvulsive therapy (ECT): A comparison of seizure-quality measures and vital signs. J ECT. 1998;14:28–35.
7. Avramov MN, Husain MM, White PF. The comparative effects of methohexital, propofol and etomidate for electroconvulsive therapy. AnesthAnalg. 1995;81:596–602.
8. Fear CF, Littlejohns CS, Rouse E, McQuail P. Propofolanaesthesia in electroconvulsive therapy: Reduced seizure duration may not be relevant. Br J Psychiatry. 1994;165:506–9.
9. Howie MB. Black HA, Zvara I), ci al. Esinolol reduces autononjic hypersensitivity and length of seizures induced by eleetro convulsive therapy. Anesth. Analg. 1990;71:384-88.
10. Knos GB. Sung YF, Stondemire A. et at Usc of labetalol to control cardiovascular respotises to electra convulsive therapy (Abstract), AnesthAnalg., 1990:70:S210.
11. Wells DG, Davies GG. Rosewarnc F, Attenuation at' electro convulsive therapy indttced hypertension with sublingual nifledipineAnaesth.Intens. care. 1989;17:31-33.
12. Wciuger MB. Partridge BL. Hanger R. et at. Prevention ol'tlte cardiovascular attditenroendoeriute response to electra convulsive therapy: 1. Effectiveness of pretreatment regtmctis on HemodvnamicsAueslh. Analg. 1991:73:556-62.
13. Dwyer It, McCaughey W. Laverv J, ci al. Comparison of propofol and metholtexitone as anaestheticagents r electro convulsive therapy. Anaesthesia. 1988:43:459-62.
14. Villalonga A, bernardo M, Gomar C, fita G, Escobar R, Pacheco M: cardicvascular Response and anesthetic recovery in electroconvulsive therapy with propofol of thiopental convuls therapy. 1993;9:108-111.
15. W.k.Body and F.O. Lali comparison of propofol and thiopentone as anaesthetic,agent for electroconvulsive therapy.anaesthesia. 1990;45:623-628.

Source of Support: Nil; **Conflict of Interest:** None

Submitted: 05-06-2017; **Accepted:** 10-07-2017; **Published:** 17-07-2017