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Thiopentone Sodium Vs Ketofol: A Comparative Study In Electroconvulsive Therapy

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ABSTRACT-

BACKGROUND & AIM OF STUDY: We aim to compare THIOPENTONE SODIUM (TPS) and KETOFOL (a mixture of KETAMINE+PROPOFOL) as induction agents during ECT regarding their effects on ECT-induced hemodynamic changes, seizure duration and recovery parameters.

METHODS: 60 patients posted for electroconvulsive therapy (ECT) was allocated 30 each in two groups. Patient was premedicated with inj. glycopyrrolate 10 mcg/kg and induced with inj. Thiopentone sodium 3 mg/kg in Group A and Ketofol (Ketamine 0.5mg/kg + Propofol 0.5mg/kg) in Group B along with depolarizing muscle relaxant, inj. Succinyl choline 1mg/kg. Patient seizure duration, time for recovery, Richmond agitation sedation score, Modified Aldrete score, hemodynamics like heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, SPO2 were recorded at baseline and at 0 min, 3 min, 5 min, 10 min, 15 min, 20 min after induction. Parameters average was calculated and were compared by using chi-square test.

RESULTS: Seizure duration was better in Thiopentone sodium group(A) than Ketofol group(B) which was statistically significant with p=0.001.

Recovery time was better in Group B than Group A which was statistically significant with p=0.02. RASS, MAS were comparable between two groups. Hemodynamics such as HR was significantly lesser in 3 min& 5 min with Group B(Ketofol). MAP was significantly lesser during induction& 20th min for group A(TPS). SPO2 was comparable in both groups. SBP at 0 min was lesser in Group A than Group B which was statistically significant. DBP at 10 min was lesser in group A than Group B which was statistically significant. In other minutes it was comparable between two groups.

CONCLUSION: Thiopentone sodium has significantly showed longer duration of seizure time. Ketofol showed significantly shorter duration of recovery time in patient undergone Electroconvulsive therapy.

INTRODUCTION:

Electroconvulsive therapy (ECT) is a well-established and effective treatment for various severe psychiatric conditions, particularly in cases of treatment-resistant depression, schizophrenia, and catatonia. The anaesthetic agent used during ECT plays a critical role in influencing seizure duration, hemodynamic responses, and post-procedural recovery. Thiopentone sodium, a barbiturate, is traditionally used due to its rapid induction and anticonvulsant properties, although it may reduce seizure duration and prolong recovery [1].

In contrast, Ketofol, a combination of ketamine and propofol, has gained interest due to its potential to provide a balanced anaesthetic profile. Ketamine's NMDA antagonism supports seizure activity, while propofol offers rapid onset and smoother emergence. Studies have shown that Ketofol offers better hemodynamic stability and improved recovery scores compared to Thiopentone or Propofol alone [2]. Moreover, its use has been associated with optimal seizure quality and favourable sedation profiles, making it suitable for repeated ECT sessions [3].

This study aims to compare the efficacy of Thiopentone sodium and Ketofol in ECT in terms of seizure duration, recovery time, hemodynamic parameters, and sedation scores Richmond Agitation-Sedation Scale (RASS) and Modified Aldrete Score (MAS), to determine the more effective and safe anaesthetic regimen for ECT.

AIM: We aim to compare THIOPENTONE SODIUM (TPS) and KETOFOL (a mixture of KETAMINE+PROPOFOL) as induction agents during ECT regarding their effects on ECT-induced hemodynamic changes, seizure duration and recovery parameters.

METHODOLOGY:

This prospective, randomized, comparative study was conducted after obtaining institutional ethical committee approval and informed written consent from all participants. The study was conducted in Department of Anaesthesiology, Narayana Medical College and Hospital. This study was conducted from January 2024 to June 2024. A total of 60 adult patients (ASA physical status I–II), aged between 18 and 65 years, scheduled to undergo modified electroconvulsive therapy (ECT) were enrolled. Patients were randomly allocated into two groups (n = 30 in each group) using a computer-generated randomization sequence: Group A (Thiopentone Group): Received Inj. Thiopentone sodium 3 mg/kg IV. Group B (Ketofol Group): Received a combination of Inj. Ketamine 0.5 mg/kg + Inj. Propofol 0.5 mg/kg IV (Ketofol).

Inclusion Criteria: Age 18–65 years, ASA physical status I–II, Scheduled for ECT under general anaesthesia, Provided informed consent Exclusion Criteria: Known allergy to any study drugs, Uncontrolled hypertension or cardiac disease, raised intracranial or intraocular pressure, Pregnancy or lactation, History of seizure disorder not related to psychiatric illness, Patient refusal, ASA III & IV.

Preoperative Preparation All patients were kept nil per oral as per standard fasting guidelines. On arrival in the procedure room, standard monitors were attached including non-invasive blood pressure (NIBP), electrocardiogram (ECG), and pulse oximetry (SpO_2). Baseline vital parameters were recorded. Patients were premedicated with intravenous Glycopyrrolate 10 mcg/kg to reduce secretions and vagal responses.

Anaesthesia and ECT Procedure

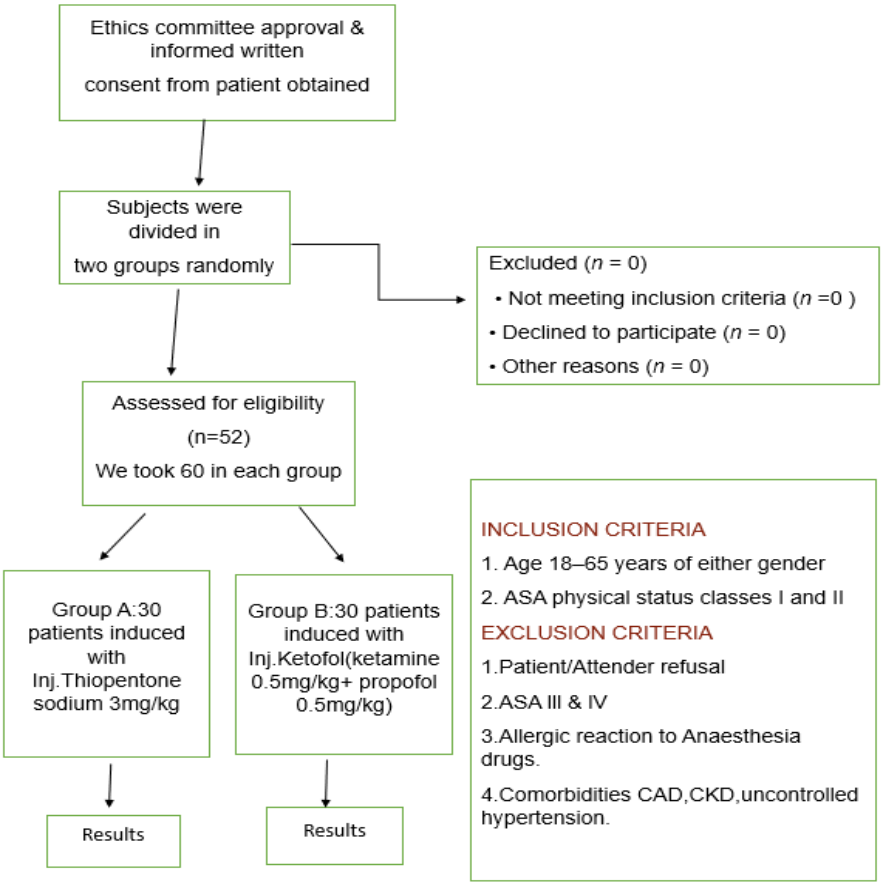
Anaesthesia was induced with the allocated drugs for each group. After confirming loss of consciousness, all patients received intravenous Succinylcholine 1 mg/kg to achieve muscle relaxation. Mask ventilation was maintained with 100% oxygen using a Bain's circuit until spontaneous respiration resumed post-ECT.

The ECT was administered using a brief pulse stimulator with bilateral electrode placement. The stimulus intensity was titrated as per individual seizure threshold.

Outcome Measures

The following parameters were recorded and compared between groups Seizure Duration (motor seizure time in seconds), Time to Recovery (time from end of seizure to eye opening on verbal command), Richmond Agitation-Sedation Scale (RASS): Assessed immediately post-ECT, Modified Aldrete Score(MAS): Assessed until score ≥9, Hemodynamic Parameters such as Heart rate (HR), Systolic blood pressure (SBP), Diastolic blood pressure (DBP), Mean arterial pressure (MAP),SpO_2 were recorded at baseline (pre-induction),0 min (immediately after induction), 3 min, 5 min, 10 min, 15 min, and 20 min after induction.

CONSORT DIAGRAM



Statistical Analysis:
Statistical analysis was performed using SPSS v20 (IBM® SPSS® Statistics V20). Qualitative data were recorded as the number of patients and analysed using the Chi-square test. Quantitative data were recorded, mean ± standard deviation calculated and P value was calculated using a t-test. $P < 0.05$ was considered statistically significant.

RESULTS:

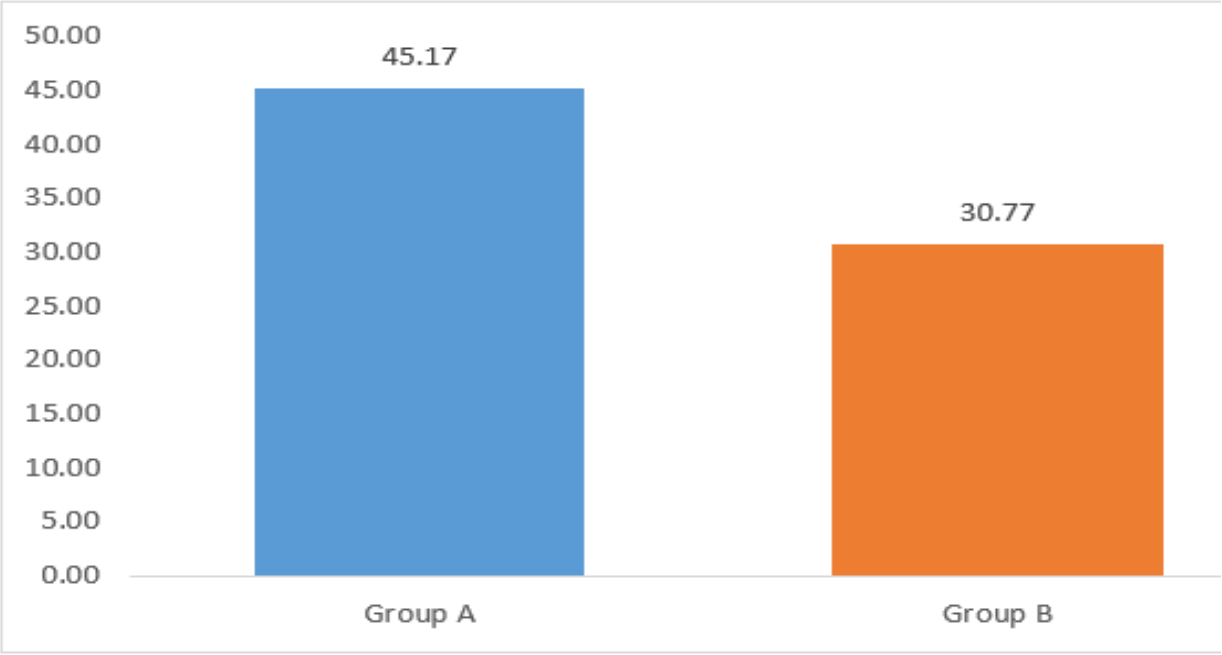
DATA	GROUP A	GROUP B	P-VALUE
AGE (IN YEARS)	39.6±11.2	41.3±10.7	0.480
SEX	M=17; F=13	M=14; F=16	0.441

Table 1: DEMOGRAPHIC RESULTS

The Demography has met the inclusion criteria but didn't show any statistically significant difference.

	Group				P value
	Group A		Group B		
	Mean	Standard Deviation	Mean	Standard Deviation	
Seizure sec	45.17	16.69	30.77	16.08	0.001

Table 2: SEIZURE DURATION (SEC)
Seizure duration was better in Group A than Group B which was statistically significant with p=0.001.

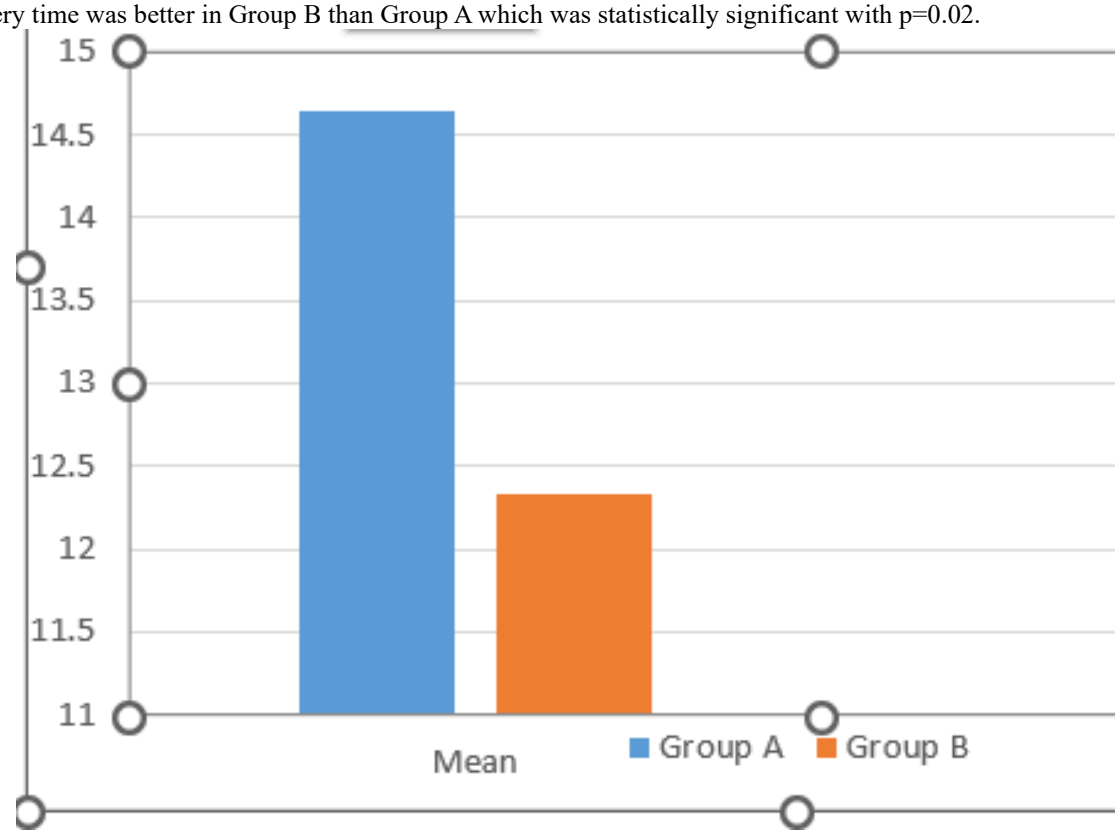


Graph 1:SEIZURE DURATION(SEC)

	Group				P value
	Group A		Group B		
	Mean	Standard Deviation	Mean	Standard Deviation	
Recovery min	14.65	6.97	12.33	3.68	0.02

Table 3:RECOVERY TIME (MIN)

Recovery time was better in Group B than Group A which was statistically significant with p=0.02.

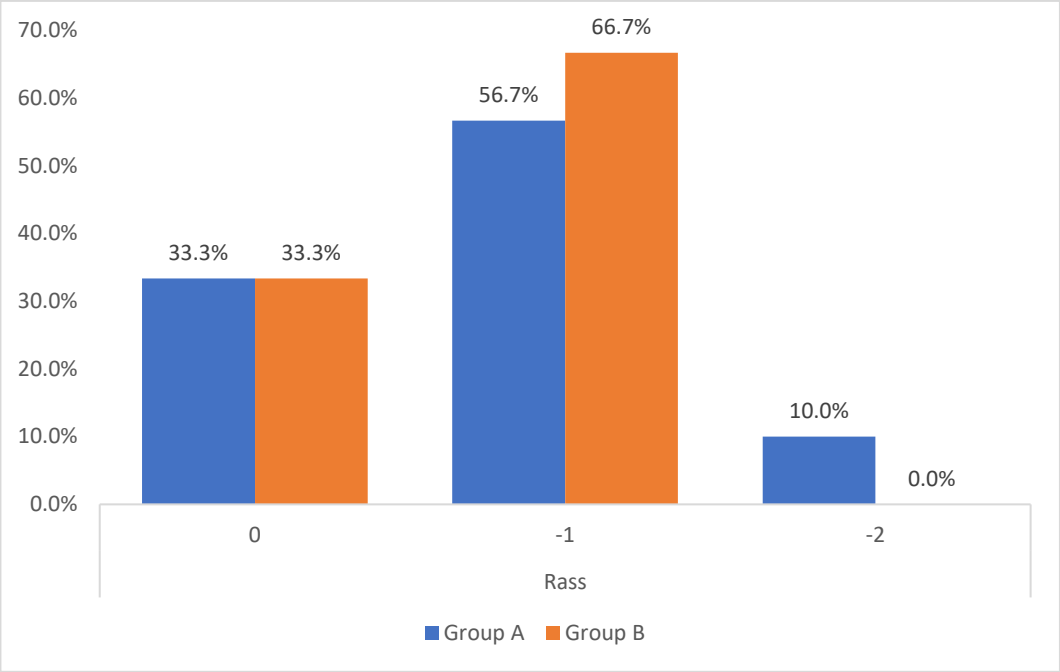


Graph 2: RECOVERY TIME (MIN)

		Group				P value
		Group A		Group B		
		Count	Column N %	Count	Column N %	
RASS	0	20	33.3%	20	33.3%	0.198
	-1	34	56.7%	40	66.7%	
	-2	6	10.0%	0	0.0%	

Table 4: RICHMOND AGITATION SEDATION SCORE

RASS was comparable between Group A and Group B.

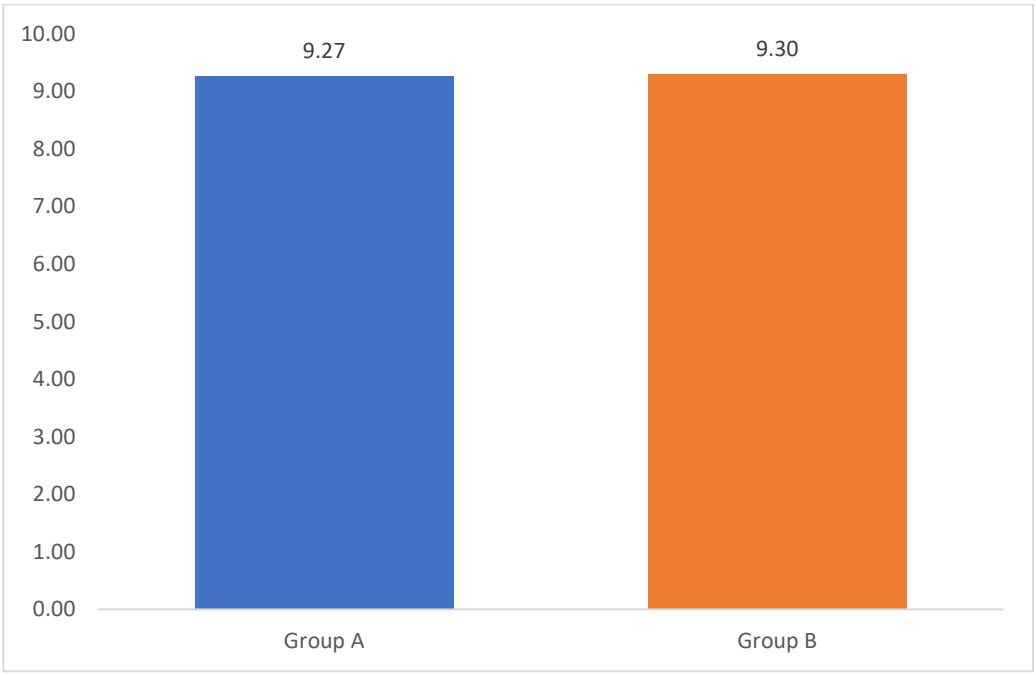


Graph 3: RICHMOND AGITATION SEDATION SCORE

	Group				P value
	Group A		Group B		
	Mean	Standard Deviation	Mean	Standard Deviation	
MAS	9.27	0.45	9.30	0.47	0.779

Table 5:MODIFIED ALDRETE SCORE

MAS was comparably better with Group A than Group B.

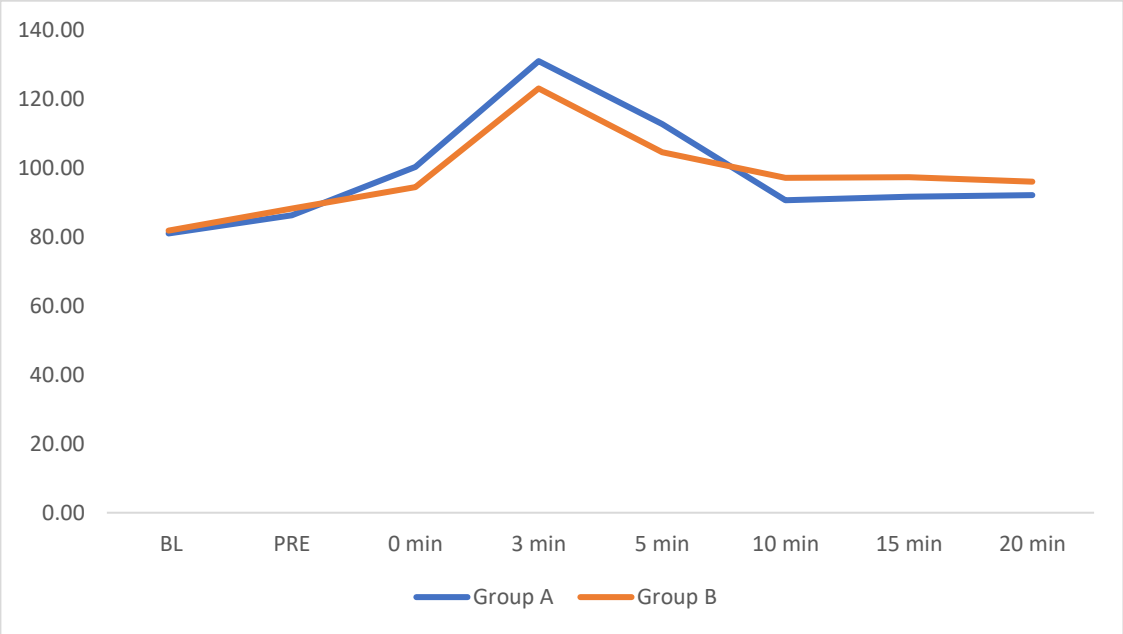


Graph 4: MODIFIED ALDRETE SCORE

HR	Group				P value
	Group A		Group B		
	Mean	Standard Deviation	Mean	Standard Deviation	
BL	81.00	13.15	81.83	9.87	0.782
PRE	86.27	14.57	88.20	13.24	0.593
0 min	100.33	15.73	94.43	13.95	0.13
3 min	131.00	14.54	123.07	15.42	0.045
5 min	112.70	8.31	104.53	13.99	0.008
10 min	90.63	14.94	97.17	13.78	0.084
15 min	91.67	15.49	97.37	16.46	0.173
20 min	92.17	15.91	96.00	13.21	0.314

Table 6: HEART RATE

HR was significantly better with Group B in 3 min, 5 min and comparable between two groups in other intervals.

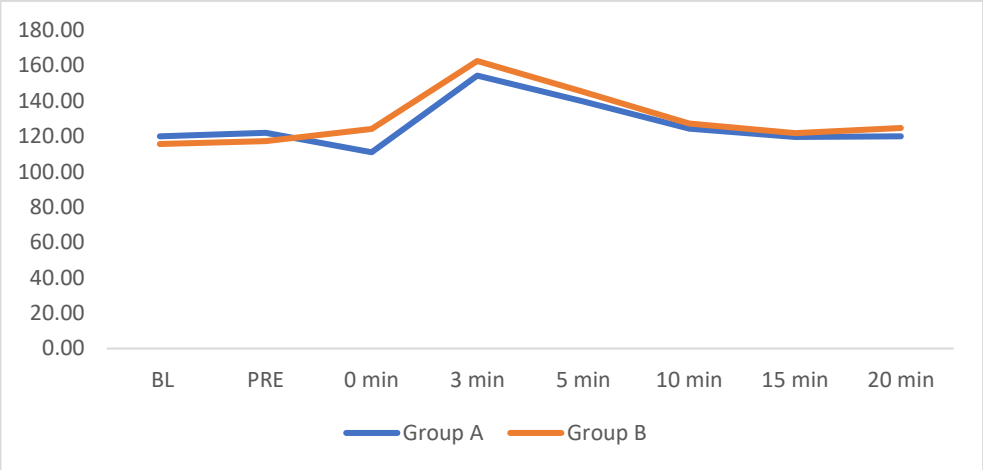


Graph 5: HEART RATE

SBP	Group				P value
	Group A		Group B		
	Mean	Standard Deviation	Mean	Standard Deviation	
BL	120.00	11.74	115.67	10.06	0.13
PRE	122.00	11.57	117.20	9.26	0.081
0 min	111.00	19.18	124.20	11.54	0.002
3 min	154.33	18.70	162.60	21.95	0.122
5 min	139.67	12.17	145.13	11.01	0.073
10 min	124.33	11.65	127.33	12.85	0.347
15 min	119.73	9.51	121.80	11.22	0.445
20 min	120.00	8.71	124.67	11.96	0.089

Table 7: SYSTOLIC BLOOD PRESSURE

SBP was significantly better with Group A during induction and comparable between two groups in other intervals.

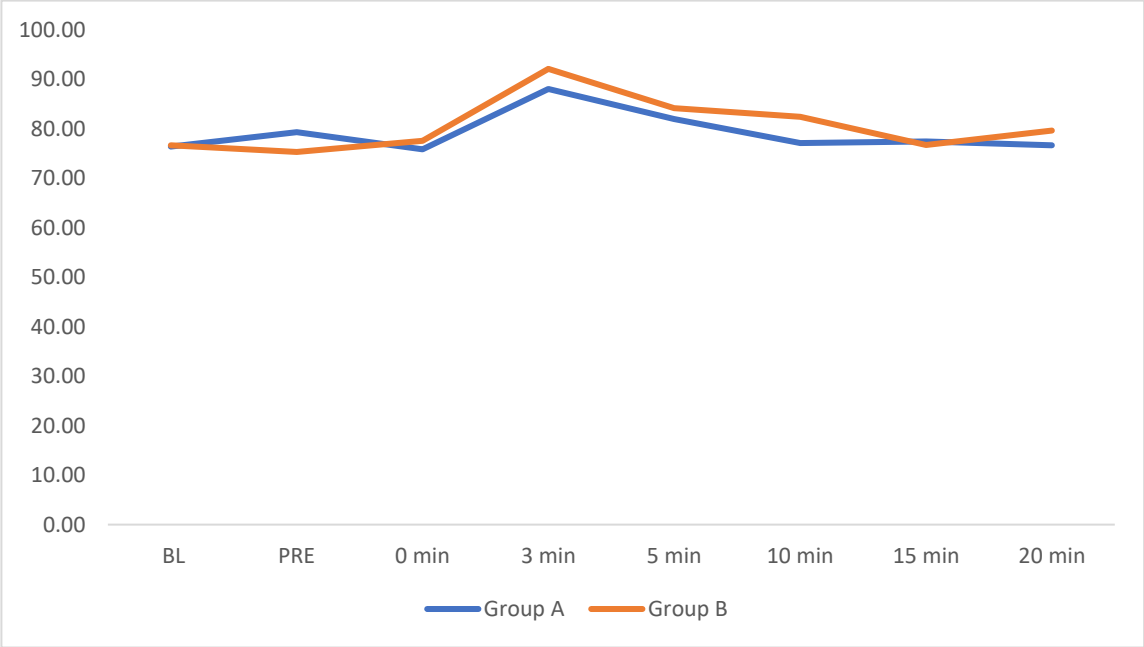


Graph 6: SYSTOLIC BLOOD PRESSURE

DBP	Group				P value
	Group A		Group B		
	Mean	Standard Deviation	Mean	Standard Deviation	
BL	76.40	10.64	76.67	11.55	0.926
PRE	79.33	8.28	75.33	8.60	0.072
0 min	75.87	8.50	77.60	11.54	0.51
3 min	88.07	13.36	92.13	12.02	0.22
5 min	82.00	9.25	84.20	7.99	0.328
10 min	77.13	5.82	82.47	7.98	0.004
15 min	77.47	6.79	76.73	8.33	0.71
20 min	76.67	6.06	79.67	7.18	0.086

Table 8: DIASTOLIC BLOOD PRESSURE

DBP at 10 min was lesser in group A than Group B which was statistically significant and comparable between two groups in other intervals.

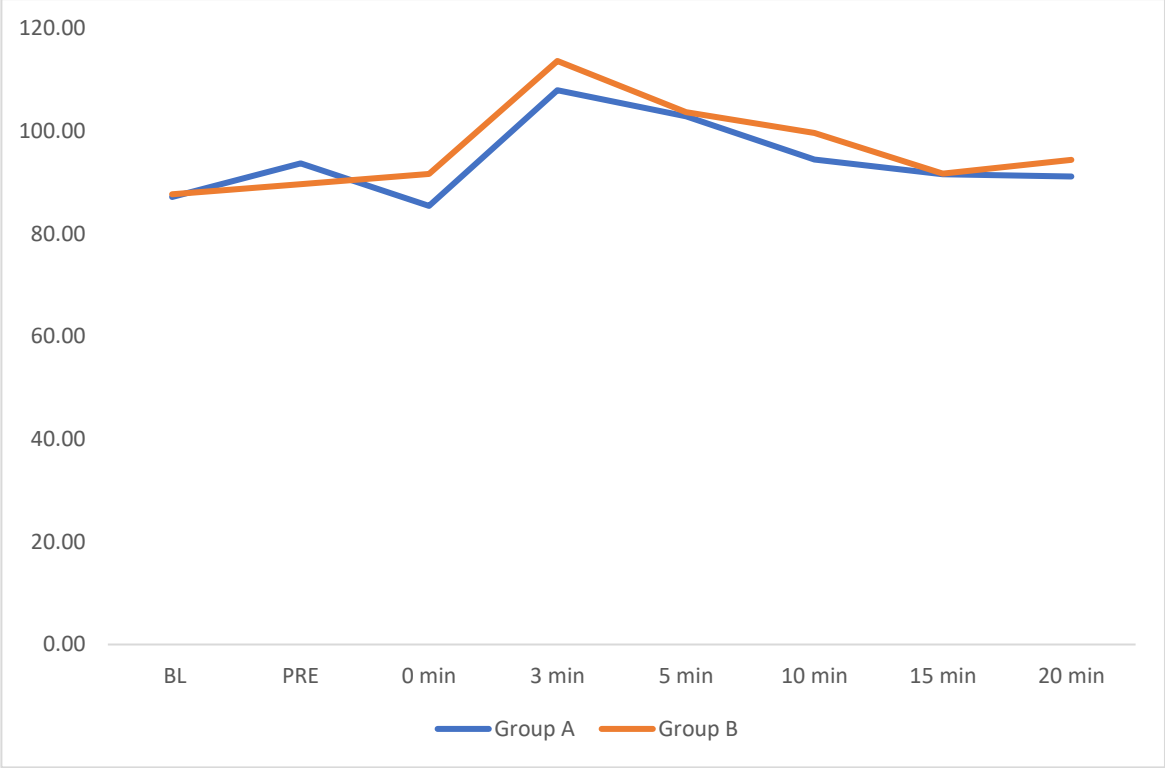


Graph 7: DIASTOLIC BLOOD PRESSURE

MAP	Group				P value
	Group A		Group B		
	Mean	Standard Deviation	Mean	Standard Deviation	
BL	87.17	10.71	87.70	7.74	0.826
PRE	93.73	9.23	89.67	7.82	0.071
0 min	85.43	11.29	91.63	11.50	0.039
3 min	107.97	14.68	113.67	17.55	0.178
5 min	102.87	9.62	103.70	8.20	0.719
10 min	94.47	9.17	99.67	11.81	0.062
15 min	91.57	6.73	91.70	8.47	0.946
20 min	91.13	5.47	94.40	6.82	0.045

Table 9: MEAN ARTERIAL PRESSURE

MAP was significantly lesser during induction& 20th min for group A and comparable between two groups in other intervals.

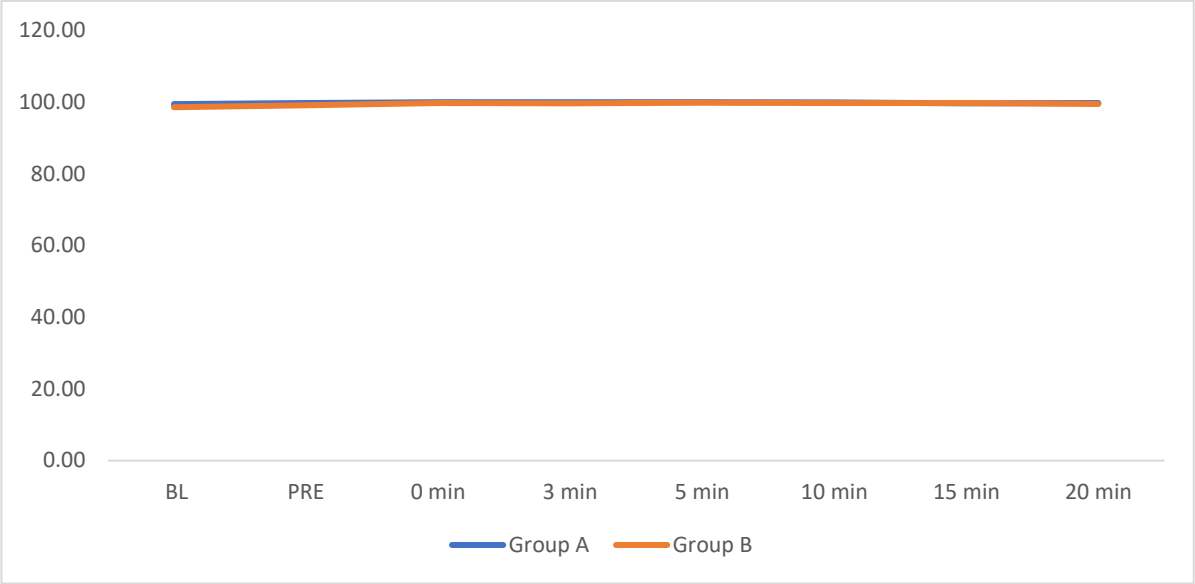


Graph 8: MEAN ARTERIAL PRESSURE

SPO2	Group				P value
	Group A		Group B		
	Mean	Standard Deviation	Mean	Standard Deviation	
BL	99.40	1.10	98.53	2.75	0.115
PRE	99.67	0.61	99.03	2.62	0.202
0 min	99.93	0.25	99.73	0.69	0.142
3 min	99.93	0.25	99.60	1.10	0.112
5 min	99.93	0.25	99.80	0.55	0.233
10 min	99.87	0.51	99.73	1.01	0.522
15 min	99.60	0.72	99.67	0.61	0.7
20 min	99.67	0.61	99.47	0.82	0.287

Table 10: SPO2

SPO2 was comparable in both groups in all intervals.



Graph 9: SPO2

DISCUSSION:

It is important to maintain depth of anaesthesia and adequate seizure duration. Thiopentone sodium and Propofol are commonly used as induction agents for ECT but have deleterious effects on hemodynamics, seizure duration and recovery depending on their pharmacokinetics and pharmacodynamics. Ketofol a mixture of propofol and ketamine balance each other hemodynamic effects and maintain hemodynamic stability. Action of ketamine on seizure duration counteracts anticonvulsant action of propofol thus improving seizure duration when used in combination. In our study, seizure duration was more with Group A when compared to Group B which was statistically significant. Recovery was faster in Group B when compared to Group A which was statistically significant. Hemodynamic parameters, RASS and MAS were comparable in both groups which was statistically not significant.

Seizure duration was significantly longer in the Thiopentone group 45.17 ±16.69 compared to Ketofol 30.77 ±16.08. This finding aligns with the results by Wang et al., [4] who observed shorter seizure durations with Ketofol than with Thiopentone due to the seizure-suppressant effect of Propofol in the combination. Thiopentone, a barbiturate, lacks the NMDA antagonism of Ketamine, which might prolong seizure activity. Hence, seizure adequacy is better supported by Thiopentone. These findings suggest Ketofol may not be ideal where prolonged seizure duration is required.

In contrast, recovery time was significantly shorter in the Ketofol group 12.33±3.68 compared to Thiopentone group 14.65±6.97 in our study. Similar findings were reported by Gupta et al.,[5] where Ketofol showed faster recovery profiles, attributed to the rapid redistribution and metabolism of Propofol and the stimulant properties of Ketamine. Patients in the Ketofol group were observed to attain higher Modified Aldrete Scores earlier. This rapid emergence favors outpatient ECT settings. Hence, Ketofol may improve turnover efficiency in high-volume centers.

Regarding hemodynamic stability, our study found that HR was significantly better at 3 min and 5 min in Ketofol group. Group A was significantly better in SBP at 0 min, DBP at 10 min, and MAP at 0 min and 20th min and was comparable between two groups at other intervals. Whereas with the findings of Poojary et al.,[6] where Ketofol was associated with better control over post-induction hemodynamic responses than Thiopentone. Propofol's vasodilatory effects and Ketamine's sympathomimetic balance resulted in smoother profiles. Thiopentone, in contrast, is linked with transient hypertension and tachycardia post-ECT. This supports Ketofol's advantage in cardiovascular risk patients.

Our study showed better RASS scores with Ketofol, indicating more favourable sedation levels post-procedure. Comparable results were demonstrated by Yalcin et al.,[7] who found more stable RASS levels and less emergence agitation in the Ketofol group. This is due to the dual effect of Propofol's sedation and Ketamine's dissociation. Thiopentone, lacking anxiolytic and analgesic synergy, may lead to more variable sedation post-ECT. Thus, Ketofol provides smoother psychological recovery.

Finally, our Modified Aldrete Scores were higher in the Ketofol group 9.30±0.47, reflecting faster and better-quality recovery than Thiopentone group 9.27±0.45. This observation is in agreement with a study by Kumar et al.,[8] which showed higher Aldrete scores with Ketofol compared to Thiopentone, especially in the early post-anaesthesia period. This enhanced recovery may result from the balanced anaesthesia and minimal residual sedation with Ketofol. This favors its use in repeated ECT procedures. Overall, Ketofol enhances safety and patient throughput.

LIMITATIONS:

Single-Center Study: The study was conducted at a single institution, which may limit the generalizability of the results to other settings or populations.

Small Sample Size: Although the sample size was adequate for primary outcome analysis, a larger sample may be needed to validate secondary outcomes or detect rare adverse events.

Short Observation Period: Post-ECT recovery and agitation were assessed only up to 20 minutes. Long-term outcomes such as cognitive recovery, postictal confusion, or delayed side effects were not evaluated.

Lack of Blinding: Complete blinding was not possible due to the distinctive clinical effects and appearances of the study drugs, which may introduce observer bias in subjective assessments such as RASS and Aldrete scores.

No EEG Monitoring: Seizure duration was assessed clinically through motor activity. EEG monitoring, which is more accurate, was not employed due to logistical limitations.

Fixed Dosing Regimen: Fixed doses were used based on body weight, without titration for individual variability in response or seizure threshold.

Exclusion of High-Risk Patients: Patients with significant comorbidities (ASA III or above) were excluded, thus findings may not be applicable to higher-risk groups undergoing ECT.

CONCLUSION:

Thiopentone sodium showed significantly higher duration of seizure. Ketofol showed significantly lesser time for recovery. Hemodynamics of ketofol has significantly lesser HR in 3 min & 5 min. SBP in 0 min, DBP 10 min; MAP 0 min & 20 min was significantly lesser in Group A than Group B.

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