# Comparative Effects Of The Haemodynamic Responses Of Modified Versus Unmodified Electroconvulsive Therapy In Nigerians.

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## Abstract

Background: Electroconvulsive therapy (ECT) provokes abrupt changes in systemic haemodynamics. We compared the effects of modified (propofol and thiopentone) versus unmodified ECT on haemodynamic responses of patients scheduled for ECT in two Nigerian tertiary hospitals between September 2008 and March 2009.Methods: In a prospective, randomized study, sixty patients slated for ECT were allocated to unmodified (UG), thiopentone (TG) and propofol (PG) groups with twenty in each group. Anaesthesia was induced with either 1mg/kg propofol (PG) or 5mg/kg thiopentone (TG) and 0.5mg/kg suxamethonium. Anaesthesia was not administered to UG. Blood pressure, systolic (SBP), diastolic (DBP) and heart rate (HR) were recorded before ECT and at 1 and 5 minutes after seizure had ended. The means of the post and pre ictal haemodynamic parameters and increases in means were compared. Results: Mean HR and SBP decreased significantly at 1 min in the modified group and increased significantly at 5 min. The mean HR increased significantly in both modified groups. Increase in MAP was significant in TG (p = 0.028). Though the increase in SBP was not significant in modified groups, it was significantly greater in the TG than PG (p = 0.012) Conclusion: Modified ECT may not be commonly practiced in our environment because of dearth of qualified anaesthetists. This study has shown that modified ECT minimizes increases in haemodynamic response when compared with unmodified ECT. Propofol at 1 mg/kg minimized increases in DPB and MAP more than thiopentone 5 mg/kg. Propofol with rapid recovery profile is suitable for modified ECT in Nigerian patients.

# INTRODUCTION

Electroconvulsive therapy (ECT) employs electrically induced generalized seizures to ameliorate symptoms of psychiatric disorders such as depressive illness, manic and schizophrenic disorders<sup>1</sup>. Electroconvulsive therapy is used as a secondary treatment when a patient has shown insufficient improvement to pharmacotherapy. The American Psychiatric Association (APA), in its 2001 recommendation for treatment, stated that ECT should be considered a primary treatment (or first-line treatment) for persons exhibiting syndromes such as severe major depression, acute mania, mood disorders with psychotic features and catatonia<sup>2</sup>. A decision to use ECT should, however, be based on evaluation of the nature and the severity of acute symptoms.

ECT is a safe and well tolerated procedure; the mortality is low approximating 3:10,000 per patient treated<sup>3</sup>. Though complications are rare, it may include prolonged apnoea, dental injuries, tongue laceration, aspiration, cardiac ischaemia and status epilepticus<sup>4</sup>. These complications may be more common in the unmodified ECT. There are shortlasting, but important, changes in cardiovascular system activities which include tachycardia and transient elevation of blood pressure<sup>5,6,7</sup>. Electroconvulsive therapy induced cardiovascular changes with a parasympathetic-sympathetic sequence may be hazardous in patients with severe cardiovascular disease<sup>8</sup>. The commonest causes of mortality associated with ECT are the acute changes in heart rate and blood pressure that follow ECT<sup>8</sup>.

The early ECTs as introduced by Cerletti and Bini in 1937 were "unmodified"; that is, not implemented under sedation, anaesthesia, neuromuscular blockade nor supplementary oxygenation, and no ventilation administered<sup>9</sup>. There have been serious attendant side effects such as hypoxia, hypercapnoea, cardiac arrhythmia and vertebral compression fracture of the mid thorax. From early 1960s, modified ECT under anaesthesia with neuromuscular blockade has become worldwide standard. The use of an intravenous induction agent, a short acting muscle relaxant with general care of the patient are the hall marks of standard anaesthetic practice for ECT. However, in a few countries, unmodified ECT is still practiced<sup>10,11,12</sup>. A major difficulty in eliminating unmodified ECT in developing countries is a lack of trained anaesthesiologists available to administer the procedure.

#### Several anaesthetics have been used for ECT.

Methohexitone and thiopentone have been commonly used. The advent of propofol added to the number of agents readily available for ECT<sup>4,7</sup>. The effects of these agents on haemodynamic changes post ECT, have been compared by various authors in the America and Europe<sup>2,3,6,13</sup>. Studies of modified ECT in our country are rare. Lim et al<sup>14</sup> compared the haemodynamic effects of thiopentone with methohexitone in Nigeria, while Ogunbiyi et al<sup>15</sup> studied the complications arising from substitution of suxamethonium with mivacurium. Ihezue et al.<sup>16</sup> determined the status and practice of ECT in Enugu. Odejide et al.<sup>17</sup> considered the extent of the use of ECT in Nigeria. No local study has compared the haemodynamic effects of thiopentone with propofol in ECT and both drugs are readily availability in our environment.

This study was therefore carried out to evaluate and compare the haemodynamic effects of modified ECT (using either propofol or thiopentone) with unmodified ECT in Nigerian patients.

## **PATIENTS AND METHODS**

The study was conducted in two hospitals in Nigeria; University of Ilorin Teaching Hospital (UITH) and Federal Neuropsychiatric Hospital (FNPH), Abeokuta. The UITH is located in the North central region of the country while the FNPH is located in the south western part of the country. Each of the hospitals has a bed capacity of over 500.

Following institutional ethics' approval, consecutive consenting patients were randomly assigned into three groups (20 for the unmodified, 20 for thiopentone and 20 for propofol groups) until a calculated sample size of 60 was obtained.

Inclusion criteria were: adult patients above 18 years of age, who were diagnosed with psychotic disorders or were being managed for depression, patients with American Society of Anesthesiologists (ASA) classification not greater than II and patients on admission. Excluded from the study were patients below 18 years of age, patients with intracranial lesion with associated evidence of raised intracranial pressure, previous history of myocardial infarction, porphyria, glaucoma, patients in whom face mask ventilation was anticipated to be difficult (e.g. edentulous patients), family history of malignant hyperthermia, hypertensive patients, patients who refused the procedure and patients with ASA greater than II.

Routine pre-anaesthetic review was carried out the night before administration of ECT. Patients were clinically examined and their age, sex, ASA, height, weight and psychomedication were documented. Informed consent was obtained from the patients' relatives after thorough explanation of the procedure. All patients were made to fast overnight and no premedication was given. Drugs for resuscitation (adrenaline, atropine, sodium bicarbonate etc.) were available in the ECT room, while suction and anaesthesia machines were checked and found to be functional before the administration of electroconvulsive therapy.

In the ECT room, all patients were connected to a "Nellcor Puritan Bennett NPB 4000" multiparameter patient monitor. The baseline heart rate (HR), systolic blood pressure (SBP), mean arterial pressure (MAP) and diastolic blood pressure (DBP) were taken and recorded pre ECT. Arterial oxygen saturation and the electrocardiogram were monitored continuously. Intravenous access was secured with size 18G cannula for drug and fluid administration. All patients in thiopentone and propofol groups had a standard general anaesthesia. After 3 minutes of preoxygenation with 100% oxygen, anaesthesia for the modified groups was induced with either propofol, 1mg/kg or 5 mg/kg of thiopentone. This was followed by intravenous administration of 0.5mg/kg of suxamethonium chloride. Patients were ventilated briefly with 100% oxygen via a face mask using a Bain breathing system as soon as fasciculation subsided. Bite blocks were inserted to protect the tongue and subsequently the patients were electro shocked for 2 seconds using Ectonustion ECT Apparatus series 5B manufactured by Ectron Ltd, Knap Close, Letchwork, England. The 'Ectonustion' is a constant current brief- pulse device with the current set at 800 milliamps. After the ECT, patients were ventilated with 100% oxygen until spontaneous respiration resumed. Patients were thereafter transferred to the recovery room in the left lateral position.

Patients in the unmodified group were preoxygenated with 100% oxygen for 3 minutes through a face mask before ECT. Bite blocks were inserted into the mouth to protect the tongue. Thereafter, patients were electro-shocked while medical assistants gently held the patients down while the seizures took place.

For all the patients, the heart rate (HR), SBP, MAP and DBP were recorded at the first and fifth minutes and thereafter every five minutes.

The mean values for HR, SBP, MAP and DBP at pre ECT (pre ictal) and the mean changes from the pre-ictal values at the first and fifth minute post ECT (post ictal) were calculated. The mean changes at the fifth minute were compared between the 3 groups.

Data were analyzed using Epi-info version 6.2 computer software. Frequency tables were generated and the means compared using the student's t test while the proportions were compared using the chi-square tests. Level of statistical significance was set at p-value <0.05.

# RESULTS

Complete data were obtained from the three groups of sixty (60) psychiatric patients scheduled for ECT. The groups were well matched for their demographic data as shown in Table I. Tables II and III show the comparison between the pre ictal and post ictal mean haemodynamic changes in the three groups at 1 and 5 min respectively. Table IV shows the comparison between unmodified and thiopentone groups, Table V shows that between unmodified and thiopentone groups and Table VI that between thiopentone and propofol groups.

The mean heart rate at 1 min decreased significantly in the unmodified group (91.55 bpm to 83.40bpm, p = 0.004) but increased significantly at 5 min (p = 0.000, Tables II and III). In the modified groups, the mean heart rates increased significantly at 1 and 5 min but they were comparable at 5 min. At 5 minutes the increases in mean HR were significantly greater in the unmodified than the modified groups (Tables IV and V).

Mean systolic blood pressure (SBP) in the unmodified group decreased significantly at 1 min (135.20 to 116.20 mmHg, p = 0.000) but increased significantly at 5 min (135.30 to 144.20 mm Hg, p = 0.002). The mean SBP increases in the thiopentone and propofol groups were not significant. The increase in mean SBP was significantly greater in the

unmodified group than the modified groups (Tables IV and V). The increases in mean SBP between thiopentone and propofol groups at 5 min were comparable (p = 0.988, Table VI).

The mean diastolic blood pressure (DBP) increased in the unmodified group but the increase was only significant at 5 min (p = 0.020). In both thiopentone and propofol groups the increases in mean DBP were not significant. However, the increase was significantly greater in the unmodified than modified groups (Tables IV and V). The increase was also significantly greater in the thiopentone group than in the propofol group at 5 min (p = 0.012, Table VI)

There was a significant increase in MAP in the thiopentone group at 1 min (96.40 to 105.29 mm Hg, p = 0.028). However at 5 min the increases were comparable between the three groups

## Figure 1

Table I: Demographic characteristics of the three groups.

Demography	Groups: n	p- value	
	Unmodified	Thiopentone	
Age (yrs)	28.70± 5.66	29.45 ± 5.96	0.293
Weight (kg)	59.45± 7.39	61.65 ± 8.26	0.060
Height (cm)	$164.79 \pm 7.97$	$169.00 \pm 5.79$	0.130
Sex: M/F	14/6	12/8	
	Unmodified	Propofol	
Age (yrs)	$28.70 \pm 5.66$	29.30 ± 5.35	0.530
Weight (kg)	59.45± 7.39	63.25 ± 6.65	0.060
Height (cm)	164.79 ± 7.97	166.60±7.06	0.069
Sex: M/F	14/6	13/7	
	Thiopentone	Propofol	
Age (yrs)	29.45 ± 5.96	29.30 ± 5.35	0.307
Weight (kg)	61.65 ± 8.26	63.25 ± 6.65	0.072
Height (cm)	$169.00 \pm 5.79$	$166.60 \pm 7.06$	0.190
Sex: M/F	12/8	13/7	

#### Figure 2

Table II: Comparison of pre-ictal and first minute post ictal mean haemodynamic parameters

Variables	Unmodified	Unmodified Thiopentone	
Heart rate			
Pre (beats/min)	91.55 ± 17.46	85.15 ± 14.75	78.85 ± 15.07
Post (beats/min)	83.40 ± 13.50	98.25 ± 23.83	93.40 ± 17.85
Change (beats/min)	8.15 (8.90%)	13.10 (15.38%)	14.55(18.45%)
P value	0.004	0.044	0.007
Systolic Blood Pressure			
Pre(mm Hg)	135.20 ± 2.11	121.10 ± 20.13	118.40 ± 25.31
Post (mm Hg)	116.20±12.92	132.25 ± 23.99	129.20 ± 24.50
Change (mm Hg)	19.10(14.12%)	11.15 (9.20%)	10.80 (9.12%)
P value	0.000	0.188	0.206
Diastolic Blood Pressu	re		
Pre(mm Hg)	76.50 ± 11.71	80.90 ± 13.48	76.70±15.03
Post (mm Hg)	76.50 ± 12.99	89.30 ± 14.72	77.00 ± 4.65
Change (mm Hg)	0.00(0.00%)	8.40(10.38%)	0.30 (0.40%)
P value	1.000	0.073	0.955
Mean Arterial Pressur	P		
Pre(mm Hg)	91.69 ± 10.94	96.40 ± 16.19	89.41 ± 14.44
Post (mm Hg)	$91.29 \pm 1.62$	105.29 ± 20.55	92.54 ± 24.23
Change (mm Hg)	0.40 (0.44%)	8.89 (9.22%)	3.13 (3.54%)
P value	0.067	0.028	0.631

Pre-preictal

Post- post ictal Change- change between pre ictal and post ictal values

### Figure 3

Table III: Comparison of pre-ictal and fifth minute post ictal mean haemodynamic parameters

Variables	Unmodified	Thiopentone	Propofol
Heart rate			
Pre-(beats/min)	91.55 ± 17.46	85.15 ± 14.75	78.85 ± 15.07
Post-(beats/min)	124.25 ± 12.05	97.55 ± 11.27	98.70 ± 15.59
Change (beats/min)	32.70 (35.72%)	12.40 (14.50%)	19.85 (25.17%)
p-value	0.000	0.003	0.000
Systolic Blood pressu	ne		
Pre- (mm Hg)	$135.30 \pm 2.11$	$121.10 \pm 20.13$	$118.40 \pm 25.31$
Post (mm Hg)	144.20 ± 15.58	$124.70 \pm 18.01$	124.85 ± 40.59
Change (mm Hg)	8.90 (6.57%)	3.60 (2.97%)	6.45 (5.44%)
P value	0.002	0.593	0.567
Diastolic Blood Press	sure		
Pre(mm Hg)	76.50 ± 1.71	80.90 ± 13.48	76.70 ± 15.03
Post (mm Hg)	94.60 ± 29.16	87.80 ± 15.22	74.00 ± 19.39
Change (mm Hg)	18.10(23.66%)	6.90 (8.52%)	2.70 (3.52%)
P value	0.020	0.107	0.630
Mean Arterial Press	ure		
Pre(mm Hg)	91.69 ± 10.94	96.40 ± 16.19	89.41 ± 14.44
Post (mm Hg)	101.78 ± 15.22	$101.53 \pm 12.02$	93.68 ± 21.00
Change (mm Hg)	10.08 (10.99%)	5.13 (5.32%)	4.27 (4.78%)
P value	0.110	0.069	0.324

Pre-preictal

Post- post ictal Change- change between pre ictal and post ictal values

#### Figure 4

Table IV: Comparison of mean haemodynamic increases between unmodified and thiopentone groups at fifth minute post ictal

Variables	Unmodified	Thiopentone	Differences	% change	p-value
Mean HR increase (bpm)	32.70	12.40	20.30	165.04	0.000
Mean SBP increase (mm Hg)	8.90	3.60	5.30	147.22	0.003
Mean DBP increase (mm Hg)	18.10	6.90	11.20	162.31	0.004
Mean MAP increase (mm Hg)	9.82	5.53	4.29	77.57	0.956
HR- heart rate					
SBP- systolic blood pressure					
DBP- diastolic blood pressure					
MAP- mean arterial pressure					
bpm- beats per minute					

#### Figure 5

Table V: Comparison of the mean haemodynamic increases between the unmodified group and the propofol group at fifth minute post ictal

Variables	Unmodified	Propofol	Differences	% chang	e p-value
Mean HR increase (bpm)	32.70	19.85	12.85	66.47	0.000
Mean SBP increase (mm Hg)	8.90	6.45	2.45	38.00	0.00
Mean DBP increase (mm Hg)	18.10	2.70	15.40	570	0.005
Mean MAP increase (mm Hg)	9.82	4.27	5.55	129	0.243
HR- heart rate					
SBP- systolic blood pressure					
DBP- diastolic blood pressure					
MAP- mean arterial pressure					

bpm – beats per minute

### Figure 6

Table VI: Comparison of mean haemodynamic increases between thiopentone and propofol group at fifth minute post ictal

Variables	Thiopentone	Propofol	Differences	% change	p-value
Mean HR increase (bpm)	12.40	19.85	7.45	37.53	0.784
Mean SBP increase (mm Hg)	3.60	6.45	2.85	44.19	0.988
Mean DBP increase (mm Hg)	6.90	2.70	4.20	155.5	0.012
Mean MAP increase (mm Hg)	5.53	4.27	1.26	82.95	0.173

HR- heart rate

SBP- systolic blood pressure

DBP- diastolic blood pressure

MAP- mean arterial pressure

bpm – beats per minute

# DISCUSSION

The cardiovascular responses to ECT may be hazardous in patients with severe cardiovascular diseases. Blunting of these responses has been effected with beta blockers<sup>18</sup>, calcium channel blockers<sup>19</sup>, fentanyl and lidocaine<sup>20</sup>. A desirable feature of intravenous anaesthetic agents used for modified ECT is their ability to attenuate these physiological responses. They must also have minimal effect on seizure duration. Since the therapy can be completed within 10minutes, the anaesthetics used for ECT should have a short action and rapid recovery profile.

The three groups of patients showed general increases in the haemodynamic variables between the pre ictal and post ictal periods (1 and 5 minutes). There were significant increases in heart rate in both modified groups. This attending tachycardia has an unfavourable influence on the myocardial oxygen supply and demand ratio. This may lead to myocardial ischaemia in susceptible patients though patients with a previous history of myocardial infarction were excluded from the study.

The typical cardiovascular response to ECT consists of an initial parasympathetic mediated bradycardia, followed immediately by a prominent sympathetic response lasting 5 to 7 minutes.<sup>21</sup> This was seen in the unmodified group of this study at 1 min post ictal as mean heart rate value and systolic blood pressure fell significantly by 8.90% and 14.12% respectively (Table II). This could have been prevented by anticholinergic premedication but the patients were not premedicated based on the Royal Psychiatric association guideline for ECT which recommends the omission of

anticholinergic drugs before ECT administration.<sup>22</sup> Mayur et al.<sup>23</sup> in support of the Royal College of Psychiatrists, recommended that atropine premedication should be avoided during electroconvulsive therapy.

The three groups of patients showed general increases in the haemodynamic variables between the pre ictal and post ictal periods (1 and 5 minutes). The unmodified group had the greatest significant increases in the mean HR (35.72%), SBP (6.57%) and DBP (23.66%) at 5 min post ictal (Table III) while thiopentone and propofol groups had significant increases in mean heart rates only at 1 and 5 min post ictal. The increase in mean SBP and DBP were not significant in the modified groups. Hence both thiopentone and propofol minimized the increase in blood pressure that was seen in the unmodified group.

Propofol at 1 mg/kg resulted in a greater increase in mean HR than thiopentone at 5mg/kg though this was not significant. The dose of propofol used in this study may not be equipotent with the dose of thiopentone. Propofol has been associated with reduction in seizure duration<sup>24,25</sup> which is considered crucial for ECT therapeutic efficacy. Since the dose of propofol and the duration of seizure have an inverse relationship, researchers tend to use smaller doses of propofol<sup>24,26</sup>. For this same reason, we chose the dose of 1 mg/kg of propofol for this study. Recommended doses for both anaesthetic agents in modified ECT range from 0.75-2.5 mg/kg for propofol and 2-5 mg/kg for thiopentone<sup>22</sup>. Other authors have used various dosages for propofol<sup>27,28</sup>. Fredman et al<sup>27</sup>, at a dose of 0.75 mg/kg reported improved haemodynamic stability with a slight increase in post ictal haemodynamic values above the pre-ictal values. Methohexitone (0.75-1.0 mg/kg) is the most commonly used drug for ECT anaesthesia and is considered the 'gold standard'28 and studies have demonstrated no difference in outcome with propofol versus methohexital<sup>29,30,31</sup>. Methohexitone is not readily available in our environment while propofol and thiopentone which have been extensively studied in ECT anaesthesia are readily available and their use in modified ECT in our environment should be encouraged.

The increases in post ictal mean SBP, DBP within each group were not significant indicating that both effectively obtunded these physiological responses to ECT. However, in this study, propofol at 1mg/kg showed advantages over thiopentone 5 mg in 2 of the measured parameters. First, the increase in MAP in thiopentone group was significant at 1 min whereas it was not significant in the propofol group. Secondly, the increase in DBP was significantly higher in the thiopentone group at 5 minutes.

Studies have compared the profiles of intravenous agents for ECT. The results of these studies have been contradictory from which conclusions cannot easily be made<sup>24,32</sup>. Several reasons may account for the discrepancies. First, various authors used different data analysis techniques. Secondly, different drug dosages were employed by different investigators for their studies.

Boey and Lai<sup>24</sup> compared propofol with thiopentone for electroconvulsive therapy and concluded that the increases seen in the systolic and diastolic blood pressure after ECT were higher with thiopentone. Their conclusion is similar to the observation in the present study, where the mean systolic and diastolic pressure increases in thiopentone group were 9.20% and 10.38% respectively as against the mean increases of 9.12% systolic blood pressure and 0.40% diastolic blood pressure in the propofol group in the first minute (Table II). A study that assessed the use of propofol for ECT in comparison with methohexitone concluded that propofol obtunded the hypertensive responses that occur during ECT more effectively than methohexitone.<sup>32</sup> Propofol gave the least increase in the fifth minute post ictal DBP and MAP in the present study.

Conclusion: Modified ECT may not be commonly practiced in our environment because of dearth of qualified anaesthetists. This study has shown that modified ECT minimized the increase in haemodynamic response when compared with the unmodified ECT. Propofol at 1 mg/kg minimized increases in DPB and MAP more than thiopentone 5 mg/kg. Propofol with rapid recovery profile is therefore more suitable than thiopentone for modified ECT in Nigerian patients.

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