ELECTROPHYSIOLOGICAL CHANGES AFTER ELECTROCONVULSIVE THERAPY (ECT) UNDER PROPOFOL

Mubashar Shah1, Chooni Lal2, Zafar Haider3, Mubeen Ikram3, Muhammad Arif Khan4

ABSTRACT

OBJECTIVE: To find out the electrophysiological changes including pulse, blood pressure, Oxygen saturation and ECG after electroconvulsive therapy (ECT) under Propofol General Anaesthesia (GA).

METHODS: Fifty patients with Depression who underwent ECT were studied. All patients were anesthetized using propofol. They were stratified according to electrophysiological changes including SPAO2, pulse and blood pressure at 60, 90 and 120 seconds after a course of 6 treatments of ECTs. Oxygen saturation was measured using a pulse oximeter.

RESULTS: The age of the patients ranged from 18-60 years and mean age was 45±12.02. The ranges of oxygen saturations recorded before ECT was 96-100%. It was 94-97% at 60 sec, 90-92% at 90 sec, 95-98% at 120 seconds. There was initial decrease in diastolic pressure from pre-ECT (77±07) to 72±05 at 60 sec followed by increase of 73±05 at 90 sec and again coming to 74±05 at 120 sec, whereas in case of systolic pressure initial decrease of 117±06 at 60 sec, 114±04 at 90 sec and 116±07 at 120 sec. There was a decrease in mean pulse from 77±4.64 (pre-ECT) to 73±2.87 at 60 sec, 74±4.02 at 90 sec and maintaining at 74±3.52 at 120 sec after ECT procedure.

Out of 50 patients, 3 (6%) developed bradycardia and 2 (4%) had ST-segment flattening at 60 seconds.

CONCLUSION: ECT under Propofol is very safe modality of treatment for patients with depressive illness and it gives much better and stable hemodynamic changes with rapid recovery from anesthesia.

KEY WORDS: Propofol (MeSH), Electroconvulsive therapy (MeSH), physiological parameters (Non-MeSH), Anesthesia (MeSH).

INTRODUCTION

Electroconvulsive therapy (ECT) has been considered as an effective and safe modality for many psychiatric conditions.1 Swiss physicians Paracelsus used Camphor orally to induce seizures to treat various psychiatric conditions.2

The most common indication for ECT is major depressive disorder, for which ECT is the fastest and most effective available therapy. ECT is a better treatment for those patients who did not respond to medications, have suicidal or homicidal thought or have severe symptoms of agitation or stupor.3

ECT was performed without anesthesia in initial few years. Later on in late 1950s, ECT was considered under general anesthesia.4 Administration of anesthesia for ECT should be given by a highly trained anesthetist.5

Administration of ECT requires general anesthesia and oxygenation.6 The depth of anesthesia should be as light as possible; excessive anesthesia may cause prolonged unconsciousness and cardiovascular complications; anesthesia in low dose may cause problems like autonomic arousal and incomplete unconsciousness.1-2

Thiopentone significantly increased the systolic and diastolic arterial pressures and heart rate after treatment whereas Propofol significantly prolonged the Apnea.7

Beginning with the electrical stimulus, there is an initial parasympathetic discharge lasting 10-15 seconds which may result in bradycardia, hypotension, or even asystole, followed by significant sympathetic changes in which cardiac arrhythmias occasionally occur.8 There was new T-wave inversions in leads V2 and V3. Also flattening of T waves in leads III, V3, V4, V5, and a VF after two courses of ECT with normal pre-ECT electrocardiogram (ECG).9

In Pakistan, most common anesthetics agents used, for induction of anesthesia for ECT, were thiopentone sodium, pentothane and diazepam as propofol is newer hypnotic agent, which has been recently introduced. This study was conducted to observe electrophysiological changes including pulse, blood pressure, Oxygen saturation and ECG after ECT by using newer anesthetic agent propofol in depressed patients that reported to PAF Hospital Mushaf, Sargodha.

METHODS

This descriptive study was carried out in Department of Psychiatry in collaboration with Department of Anesthesiology, Department of Medicine, PAF Hospital Mushaf, Sargodha. Department of Psychiatry, Jinnah Post Graduate Medical Centre (JPMC), Karachi. Department of Anesthesia, PAF Hospital Mushaf, Sargodha. Department of Medicine, PAF Hospital Mushaf, Sargodha. Date Submitted: September 20, 2015 Date Revised: March 22, 2016 Date Accepted: March 28, 2016

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CFD Hospital Mushaf Sargodha from April 2014 to September 2014. A total of fifty patients going through severe depression, to which ECT was advised, were selected by Non-Probability, convenience sampling technique. Electrophysiological changes including Oxygen (O₂) saturation, pulse, blood pressure and ECG changes were recorded. All the patients have given informed consent. Age of patients ranged from 18 to 60 years, with no cardiovascular or cerebrovascular complications or any allergy to drugs. These patients underwent six sessions of ECT (three times per week).

After pre-oxygenation with 100% oxygen, initially a dose of 100-120 mg I/V of propofol and a dose of 0.25-0.5 mg per kg I/V suxamethonium, without atropine, were given to all of these patients. After general anesthesia the patients were ventilated with 100% oxygen through face mask. Electrical current was given on both sides after Succinylcholine chloride injection. Oxygen saturation, pulse, blood pressure and ECG changes were recorded before ECT and then at 60, 90 and 120 sec’s interval. Oxygen saturation was measured using a pulse oximeter, which measures saturations in the range of 75-100%.

Data was analyzed by using SPSS version 20. It includes Descriptive studies for comparison of physiological parameters. Paired t– test was applied for the comparison of PAO₂, pulse and BP before and after ECT. A p value < 0.05 was considered significant.

RESULTS

Out of 50 patients, aged 18 to 60 years, 20 (40%) patients were female and 30 (60%) were male; mean age was 45±12.02 years. The range of oxygen saturations recorded before ECT was 96-100%, 94-97% at 60 sec, 90-92% at 90 sec, 95-98% at 120 sec. There was initial decrease in diastolic pressure at 60 seconds i.e. 73±5.49 (p<0.001) and then slightly increase at 120 seconds 74±5.42 (p<0.000); whereas, in case of systolic pressure there was a decrease i.e. 118±5.68 (p<0.000) at 60 seconds, and reaching to 118±7.71 (p<0.000) at 120 seconds (Table I). There was a decrease in mean pulse from 77±4.64 (p<0.000) (pre-ECT) to 73±2.87 (p<0.000) at 60 seconds to 74±3.52 (p<0.000) at 120 seconds.

The ECG remained normal in most of the patients except few cases. There was ST segment flattening in two patients at 60 seconds which spontaneously reverted at 120 seconds. Out of 50 patients, 3 (6%) developed bradycardia with heart rate less than 60. During the whole procedure no patient developed any active cardiac conditions (Table II).

DISCUSSION

In this study 50 patients were enrolled in which pre-induction heart rate (HR) was between 65-90 beats per minute. HR was continuously monitored with ECG electrodes and monitoring lead II on cardiac monitor. In few patients both bradycardia and tachycardia were noted. HR changes were continuously observed during ECT by cardiac monitor. Initial response was tachycardia due to sympathetic release activation followed by bradycardia due to overdrive of vagus nerve. Most of the changes in HR were abrupt and got normal spontaneously by ventilating the patient with 100 percent oxygen using bag mask ventilation. Measures were taken to handle patients in case tachycardia–bradycardia rhythm fails to revert to normal sinus. Anesthesiologist followed latest algorithm for the management of patients.

In our studies there was an initial decrease in diastolic pressure followed by increase, whereas in case of systolic pressure there was decrease in reading, significant decrease at 90 sec. Electrical current during ECT triggers the autonomic nervous system and aggravates hemodynamic changes in systemic and cerebral circulation. Changes in heart rate, blood pressure, and cardiac functions should be prevented with anticholinergic and antihypertensive agents. Ventilation should be adequately maintained after electrical stimulation.

Mark C. Webb and colleagues de-

<table>
<thead>
<tr>
<th>Parameters (n=50)</th>
<th>0 Sec</th>
<th>60 Sec</th>
<th>90 Sec</th>
<th>120 Sec</th>
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</thead>
<tbody>
<tr>
<td>Pulse (Mean ± SD)</td>
<td>77 ± 4.64</td>
<td>*73 ± 2.87</td>
<td>74 ± 4.02</td>
<td>74 ± 3.52</td>
</tr>
<tr>
<td>BP (Systolic) (Mean ± SD)</td>
<td>122 ± 10.81</td>
<td>118 ± 5.68</td>
<td>*114 ± 4.72</td>
<td>118 ± 7.71</td>
</tr>
<tr>
<td>BP (Diastolic) (Mean ± SD)</td>
<td>76 ± 6.24</td>
<td>*73 ± 5.49</td>
<td>*73 ± 5.36</td>
<td>74 ± 5.42</td>
</tr>
<tr>
<td>O₂ Saturation (Mean ± SD)</td>
<td>98 ± 0.96</td>
<td>96 ± 0.75</td>
<td>*92 ± 0.61</td>
<td>97 ± 0.78</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Total patients</th>
<th>Number of patients in which changes noticed</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bradycardia</td>
<td>50</td>
<td>3</td>
<td>Heart rate less than 60. Out of these three patients two patients have to be given injection atropine as there heart rate decreased below 50 per minute.</td>
</tr>
<tr>
<td>ST Segment Flattening</td>
<td>50</td>
<td>2</td>
<td>at 60 sec which spontaneously reverted at 120 sec</td>
</tr>
<tr>
<td>T-Wave Inversion</td>
<td>50</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>ST Segment Depression</td>
<td>50</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Cardiac Arrhythmias</td>
<td>50</td>
<td>0</td>
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</tr>
</tbody>
</table>
scribed the effects of pulse on cardiac workload and systolic blood pressure on 30 patients, which were suffering from major depression. \(^{11}\) Whereas in our study there was a decrease in mean pulse after ECT procedure i.e. after 60 seconds pulse decreased from 77 to 73; then after 90 seconds it increased from 73 to 74 and 120 seconds later it remained maintained at 73.

The ECT brings hemodynamic changes which can produce myocardial ischemia and infarction, as well as other neurological deficits. Other effects of ECT like short memory loss and serious cognitive dysfunctions have been described in literature. \(^{12}\) The T-wave changes was observed in one of the study, which resulted from increased sympathethic activity during ECT. \(^{13}\) We did not studied EEG findings and seizure activity, we only noted cardiovascular response after ECT as mentioned in results.

Kumar A. et al showed that there was more stable haemodynamic changes i.e. systolic and diastolic pressure during the procedure and heart rates were more pronounced with thiopentone compared to propofol 1 minute after ECT; diastolic blood pressures were also significant at 2 and 3 minutes interval. \(^{14}\) Whereas in case of our study, there was initial decrease in diastolic pressure at 60 seconds followed by decrease at 90 seconds and then slightly increase at 120 seconds. Whereas in case of systolic pressure, there was a decline at 60 seconds and 90 seconds and finally reaching to the base line at 120 seconds. Similarly, in our study, heart rate changes were more pronounced at 2 and 3 minutes intervals following ECT. There was decrease in mean pulse at 60 and 90 seconds and reaching the baseline at 120 seconds. Propofol being a new hypnotic agent in our country, there is a need of its comparison with other agents in our local population for the safe conduct of procedure.

**CONCLUSION**

ECT under propofol is very safe modality of treatment for patients with depressive illness. It gives much better and more stable hemodynamic changes including pulse, blood pressure, Oxygen saturation and ECG and rapid recovery from anesthesia.

**RECOMMENDATIONS**

ECT under general anesthesia with propofol keeps hemodynamic changes within safe limits, therefore; as compared to other general anesthetic agents propofol is recommended to be the preferred agent for ECTs. In our region only few studies have been conducted on physiological changes after ECT under anesthesia, therefore further studies are required to strengthen the confidence in the drug as well as the ECT as a reliable therapeutic tool.

**REFERENCES**


**CONFLICT OF INTEREST**

Authors declared no conflict of interest

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NIL

**AUTHOR’S CONTRIBUTION**

Following authors have made substantial contributions to the manuscript as under:

**MS:** Concept & study design, acquisition analysis and interpretation of data; drafting the manuscript, final approval of the version to be published

**CL & ZH:** Drafting the manuscript, critical revision, final approval of the version to be published

**MI:** Acquisition of data; final approval of the version to be published

**MAK:** Drafting the manuscript, final approval of the version to be published

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.