ISSN: 0975-3583, 0976-2833 VOL15, ISSUE 04, 2024

Original Research Article

Autonomic Surge in Modified Electroconvulsive Therapy - A Retrospective Observational Study

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Received: 13-02-2024 / Revised: 19-02-2024 / Accepted: 25-03-2024

ABSTRACT

BACKGROUND

Modified Electroconvulsive therapy has evolved as a safe, pleasant and effective non pharmacological therapy in schizophrenia and bipolar disorders due to anaesthetic management[1]. The goals of anaesthesia are sedation, amnesia, and muscle relaxation during seizures and cardiorespiratory monitoring. Cardiovascular responses to electro convulsive therapy included an initial parasympathetic surge causing bradycardia followed by sympathetic stimulation leading to tachycardia and hypertension[2].

AIMS AND OBJECTIVES

- To analyse pulse and blood pressure changes during MEC
- To observe the effects of glycopyrrolate 0.2mg given along with induction agent on the sympathetic response to ECT.
- To compare the hemodynamics with and without glycopyrrolate in MECT.
- To offer suggestions in patients with cardiac comorbidities regarding use of peripheral anticholinergic drug.

MATERIALS AND METHODS

Study Population

ASA 1 and ASA 2 patients recommended for MECT by psychiatrists.

Study Period

January 2023 to December 2023

Sample Size

50

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Study
Retrospective Observational Study.

METHODS
Following a routine consent, pre anaesthetic check, starvation for 6 hours and withholding of anticonvulsants and benzodiazepines .Patients were counselled to preprocedural area. Intravenous line & Oxygenation were done .Pulse, blood pressure, oxygen saturation were recorded. First 25 cases [Group G1] were given inj. glycopyrrolate 0.2mg IV along with Thiopentone 200mg and succinylcholine 50mg for muscle relaxation. Airway was maintained and bitemporal short ECS applied to elicit seizure. Pulse, BP were noted at 2mins, 6 minutes and 10 minutes after ECT. The same procedure was repeated for next 25 patients without glycopyrrolate (Group. Go).The observations were tabulated and analysed.

RESULTS
The pulse rate increased to more than 0.5 percent [from 80 to 120-140] in group G1 patients especially at 2nd minute after MECT (p=0.5) and group Go patients the pulse and SBP rise was smaller.

CONCLUSION
The primary aims of using an anticholinergic for drying of secretions and manage vagal bradycardia as a routine may be reconsidered due to significant sympathetic response (P=0.5) in the G1 group. This may play a significant role in patients with cardiac comorbidities as it increases myocardial oxygen demand. Kaplan and salock recommend Dr.Andrews D.Krystal's paper on CVS response to ECT. They recommend no peripheral anticholinergic drug if baseline heartrate is 90/min.

KEYWORDS
Modified Electroconvulsive Therapy, Cardiovascular Response, Glycopyrrolate.

INTRODUCTION

An increasing mechanical gadgetry, fast and electronics dependent population in the present era predispose to a variety of mental illnesses. National institute of mental health poses 15 to 100 per one lakh people develop psychosis. Advancement in pharmacotherapy and counselling have no doubt to the management. Yet the costs involved, noncompliant and inconsistent drug usage make the treatment a more challenging scenario. Since it is introduced in 1937 electroconvulsive therapy has offered solution to treat acute stimulations like catatonic schizophrenia acute paranoia, bipolar illness etcetera, especially in refractory and non-compliant patients[1]. Modified ECT is a Boon both for the patients and psychotherapists where a safe and pleasant environment is provided by Anaesthesiologists with induction agent, Muscle relaxant, monitoring, airway and hemodynamic management[3]. An initial parasympathetic followed by sympathetic out surge have been observed during ECT. The study analyses the hemodynamics during various phases of ECT therapy and Discusses the pros and cons of a compulsive need of glycopyrrolate in the modified ECT environment.

AIMS AND OBJECTIVES

- To analyse pulse and blood pressure changes during ECT.
- To observe the effects of glycopyrrolate used along with induction agent on the sympathetic surge immediately after ECT.
- To compare the hemodynamics with and without glycopyrrolate in MECT
- To offer recommendations in patients with cardio vascular and neurological comorbidities.

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MATERIALS AND METHODS

Study population

ASA 1 and ASA 2 Patients recommended for MECT by psychiatrists

Study Period

Jan 2023 to December 2023

Study Place

DSMCH Siruvachur

Sample Size

25+25=50 (arbitrary)

Study

Retrospective analysis and Observational study

Patients on long term medications for more than one month underwent Liver function tests. Pulse, Blood pressure and oxygen saturation were recorded in the preop,During induction ,during shock and after two, six and ten minutes after shock.

25 cases received Glycopyrrolate 0.2 mg intravenously along with Thiopentone sodium 200mg and succinylcholine 50 mg (GROUP G1) and 25 cases received Thiopentone 200 mg and succinycholine 50 mg without Glycopyrrolate (GROUP G0)

The parameter were recorded, tabulated and analysed.

Seizure activity was well established in all cases with an average duration of 35-40 seconds.

The pulse rate variations that were recorded during shock, 2 min and 6minutes showed significant variation in G1 group. There was no significant bradycardia during shock but 2 minutes later the pulse rate increased to a maximum of 120/min from an average of 84/min.

The rate spontaneously started settling to baseline in the 6th minute and returned to baseline in the 10th minute. The BP variation followed the changes in pulse rate.

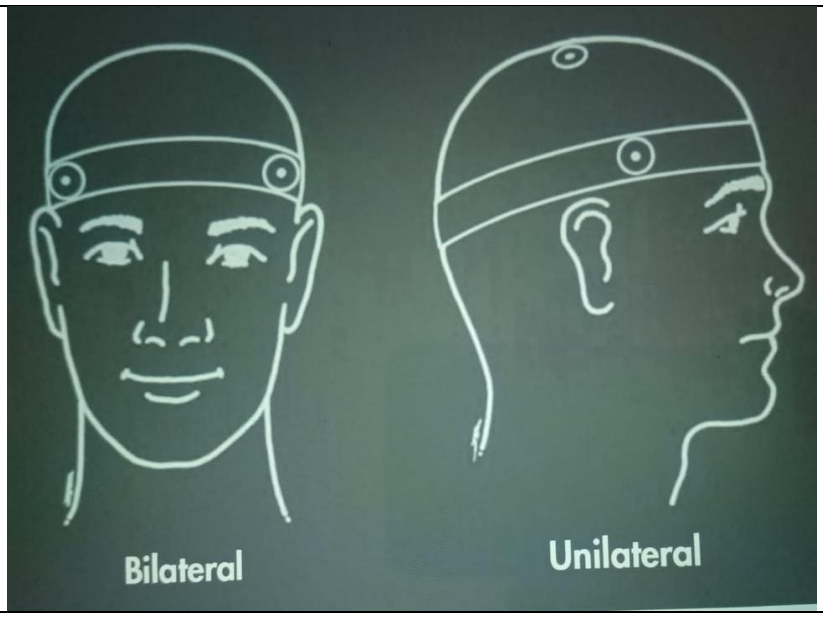

Image 1 : Uni temporal and bitemporal ECT Application

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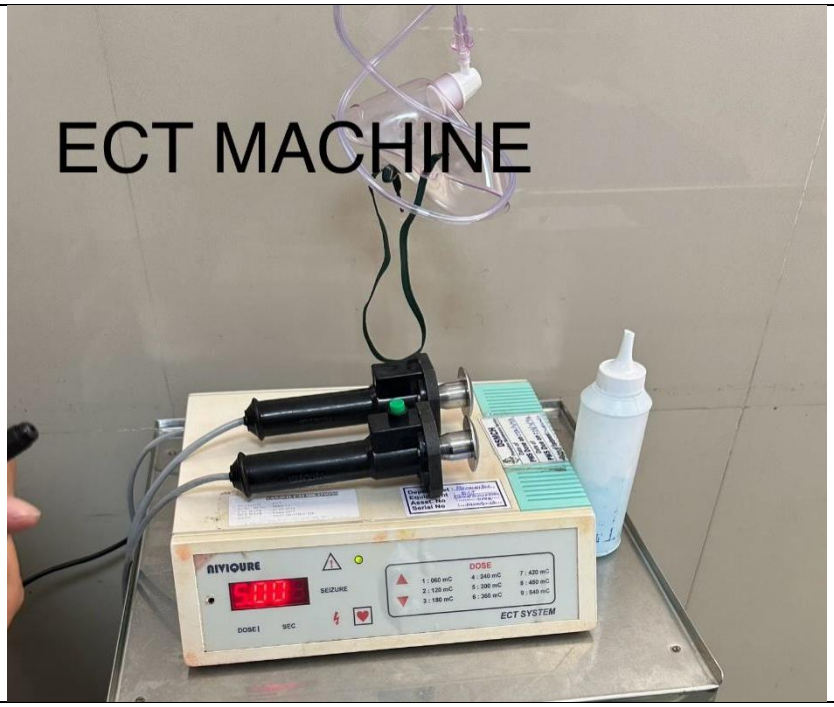

Imaege 2 : ECT Machine

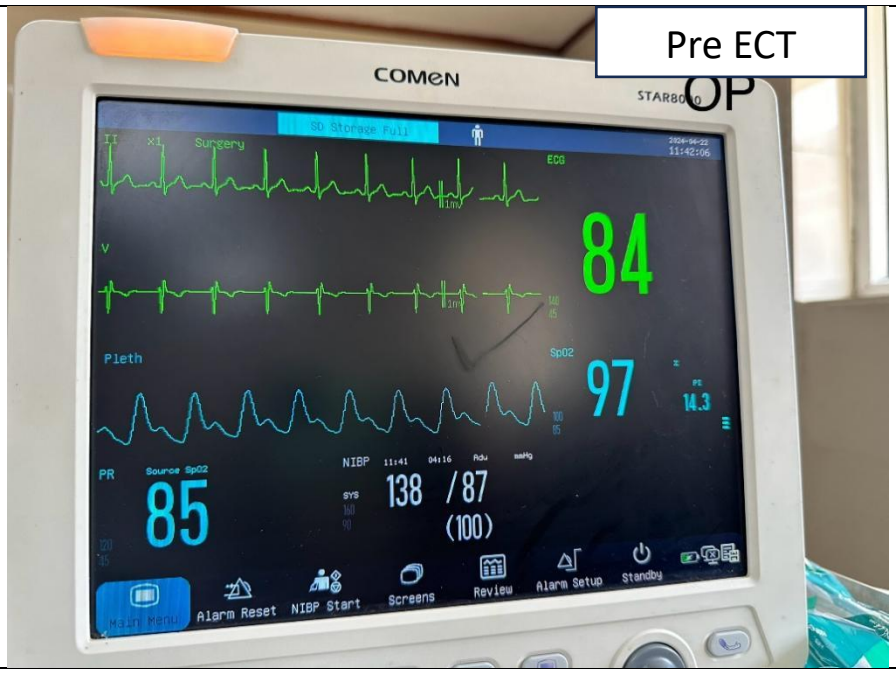

Image 3 - Monitoring -Pre ECT

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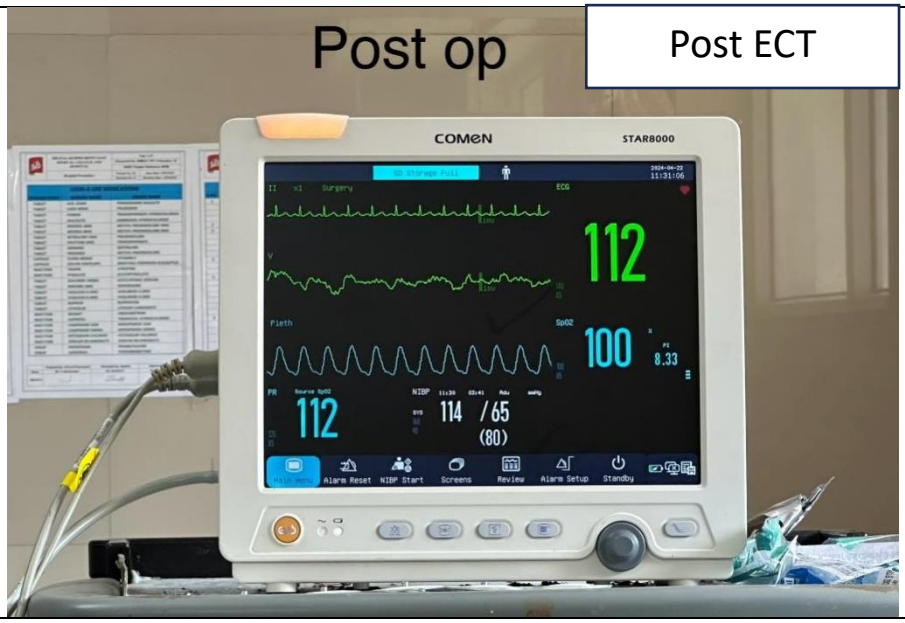


Image 4 - Monitoring -Post ECT

RESULTS

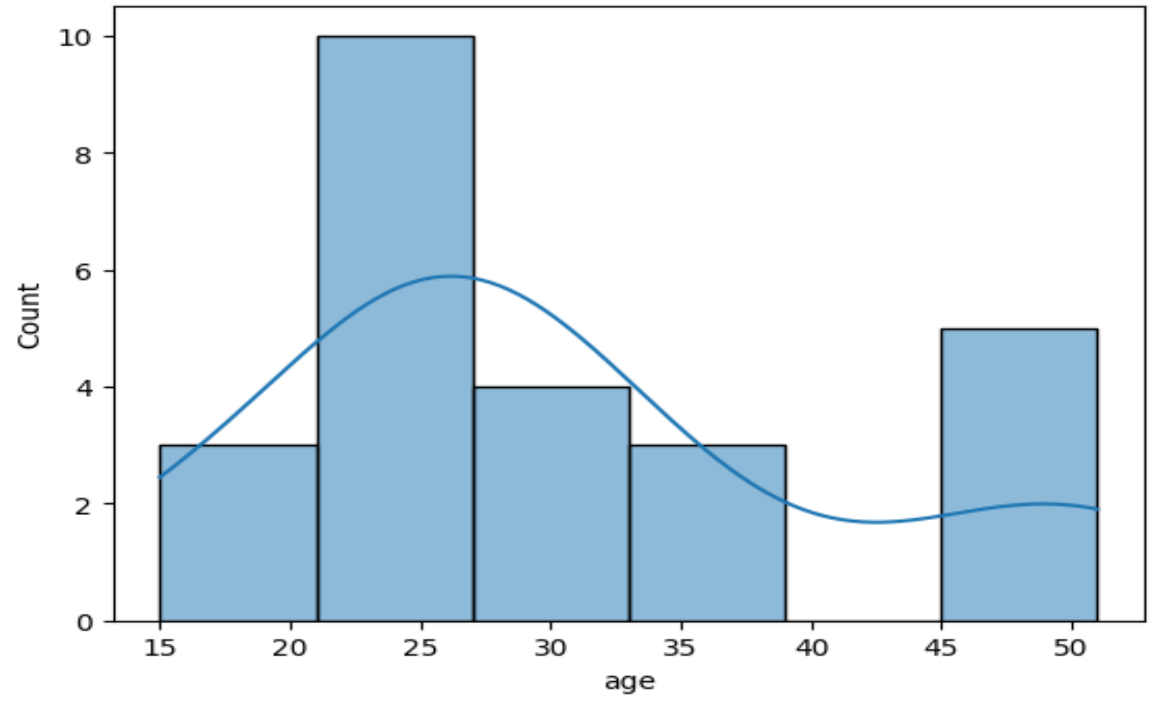
In G0 group without glycopyrrollate the baseline pre op heart rate was around 70-80/min while after ECT the 2 minutes pulse rate was 100-110/ min

In G1 group the 2 minutes after ECT pulse rate was 120-140/ min (p=0.5)

Similarly the post ECT 2minutes systolic blood pressure increased from 150-160 in G0 whlie it was 140-165/min in patients belonging to G1.

Without Drug

Age Distribution



Graph 1 - Age distribution

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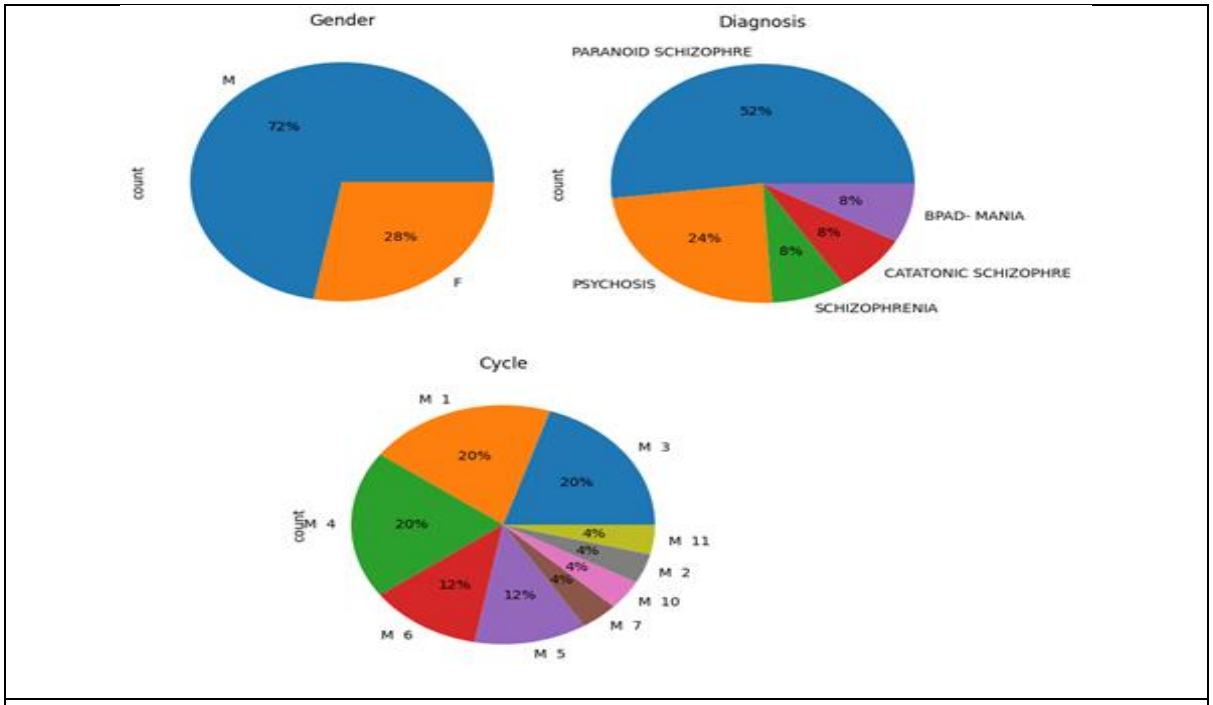
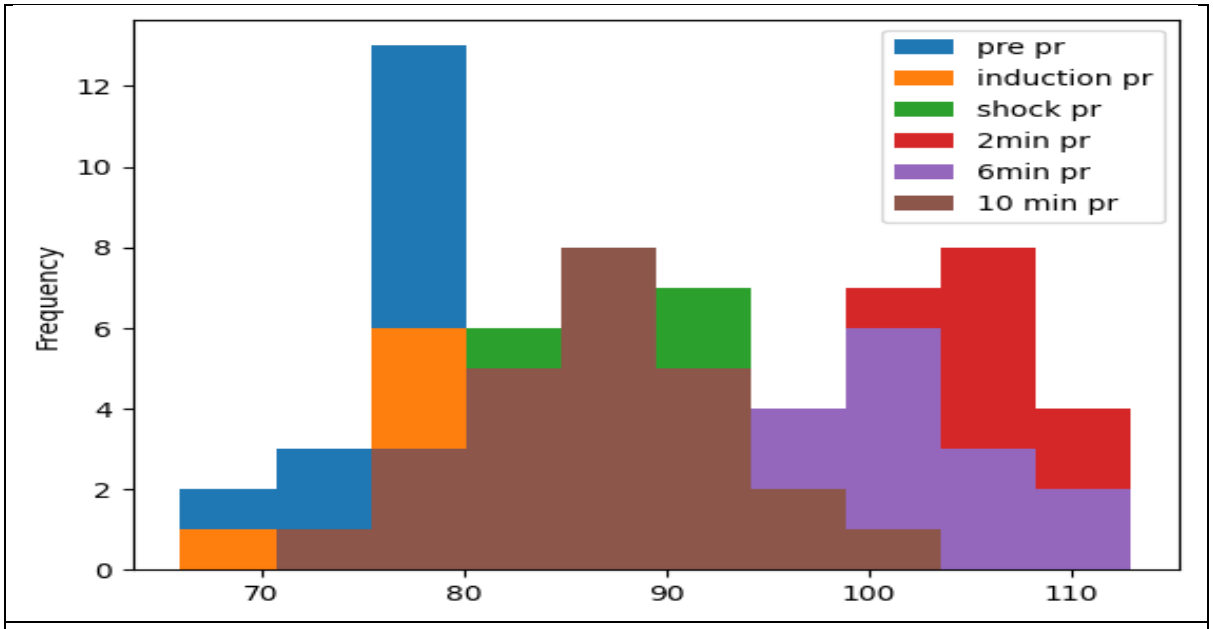


Graph 2 - Distributions



Graph 3 - Pulse rate changes

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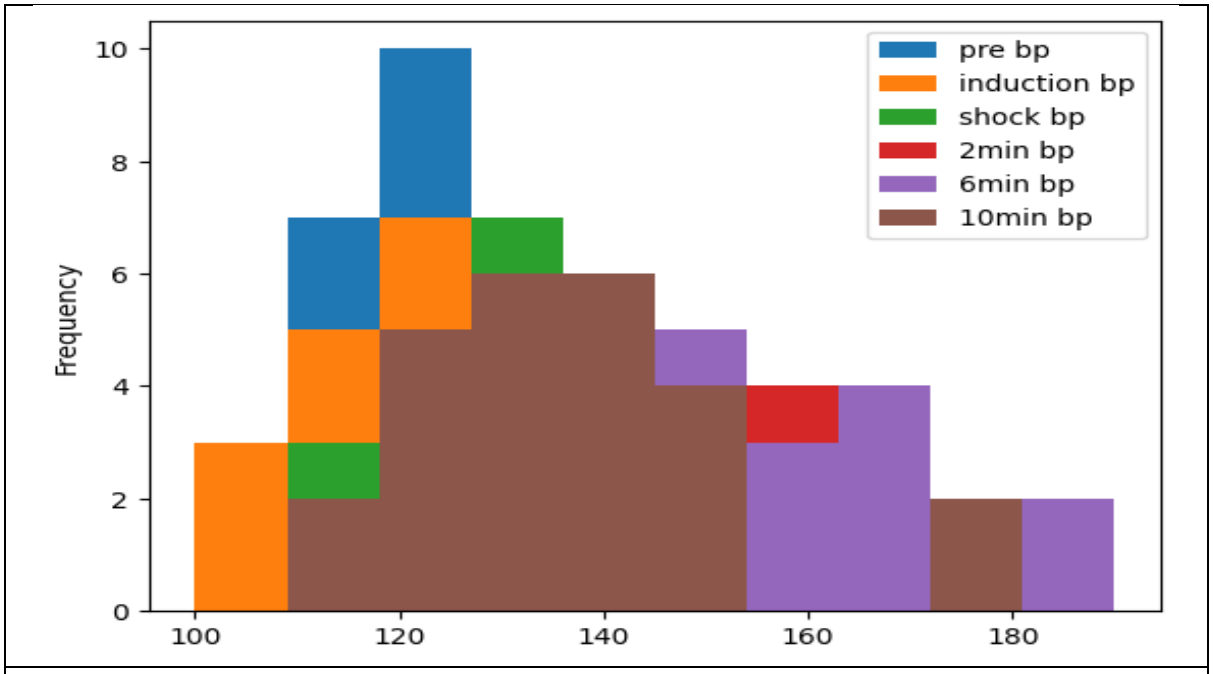
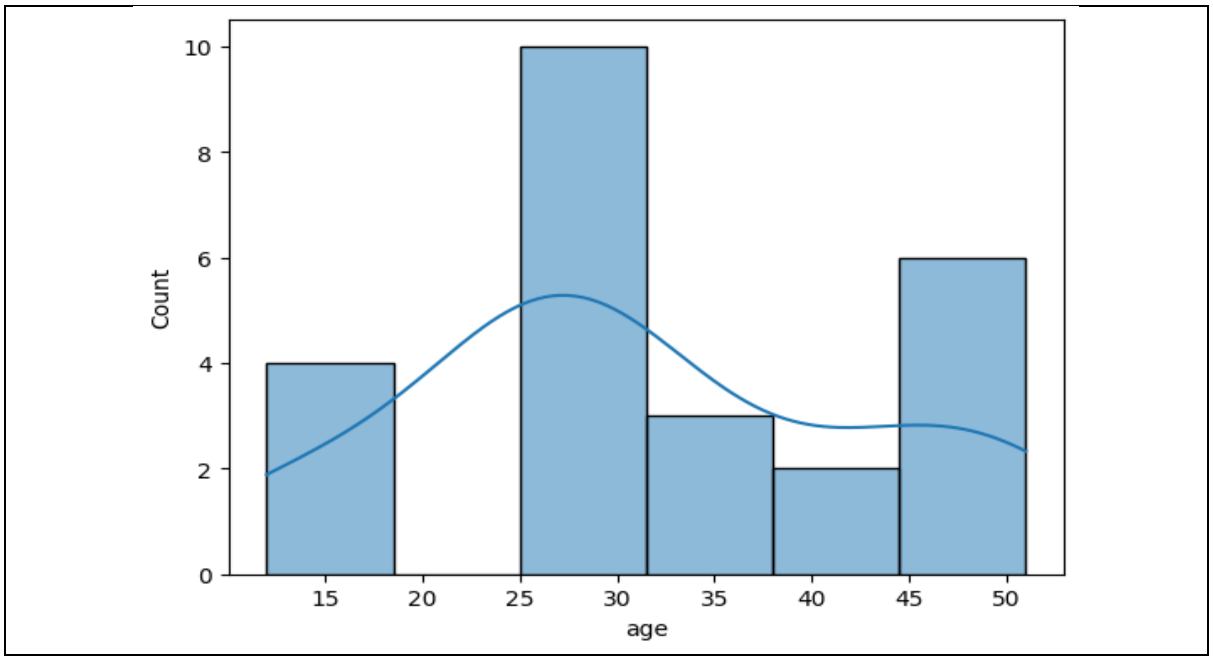


Graph 4 - BP Changes

With Drug

Age Distribution



Graph 5 - Age Distribution

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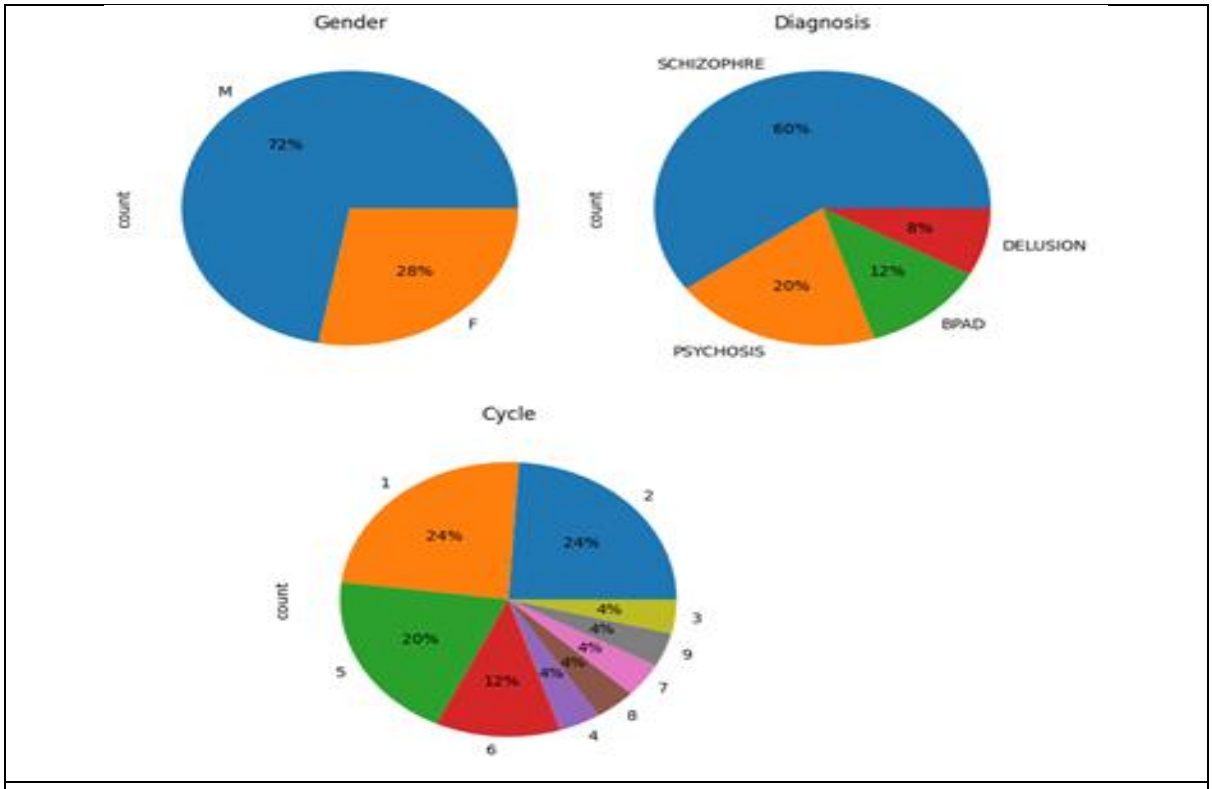
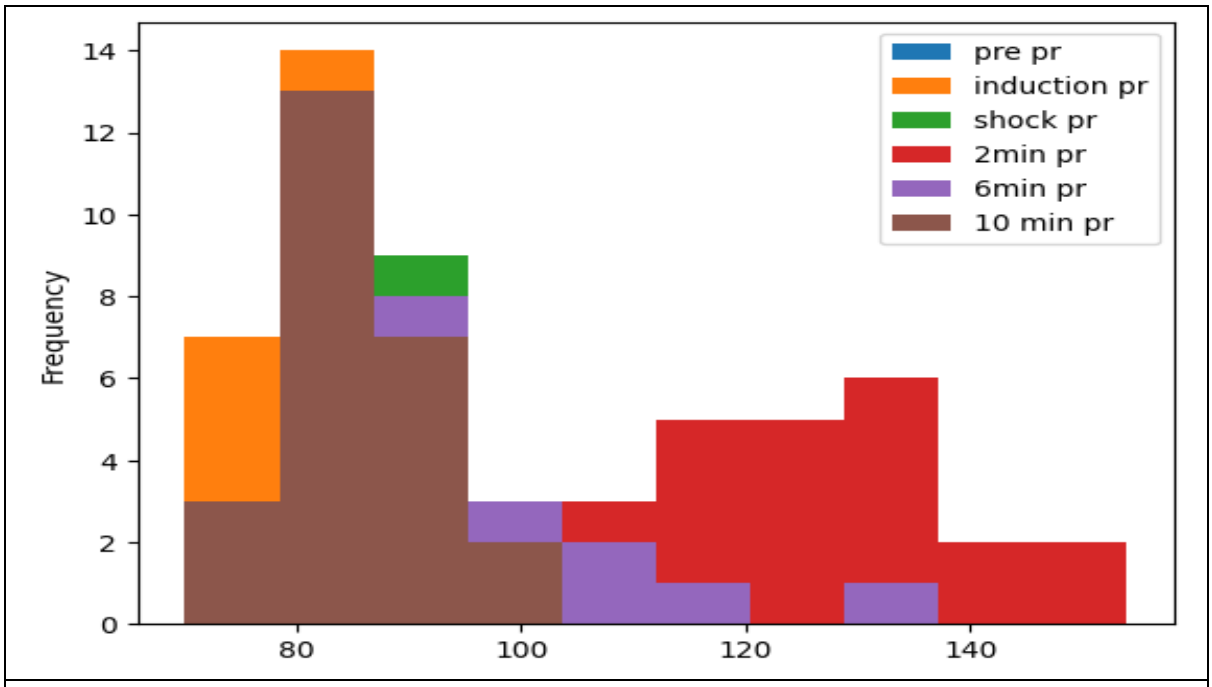


Graph 6 - Distribution

Pulse rate changes



Graph 7 - Pulse Rate Changes

BP Changes

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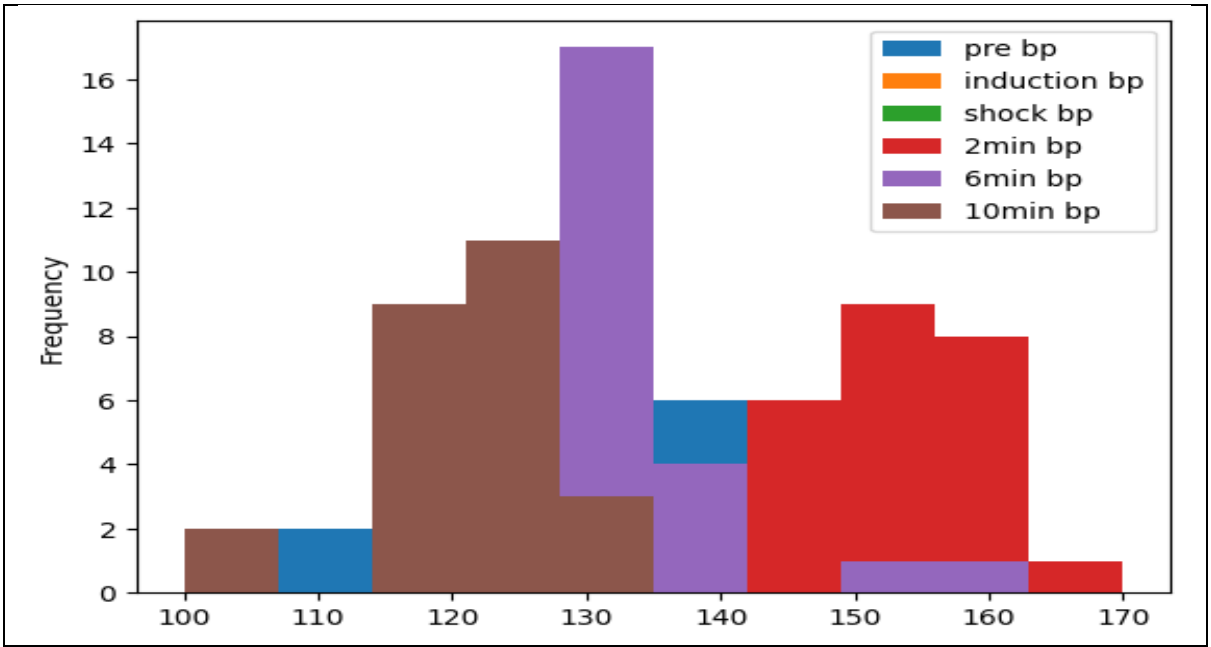
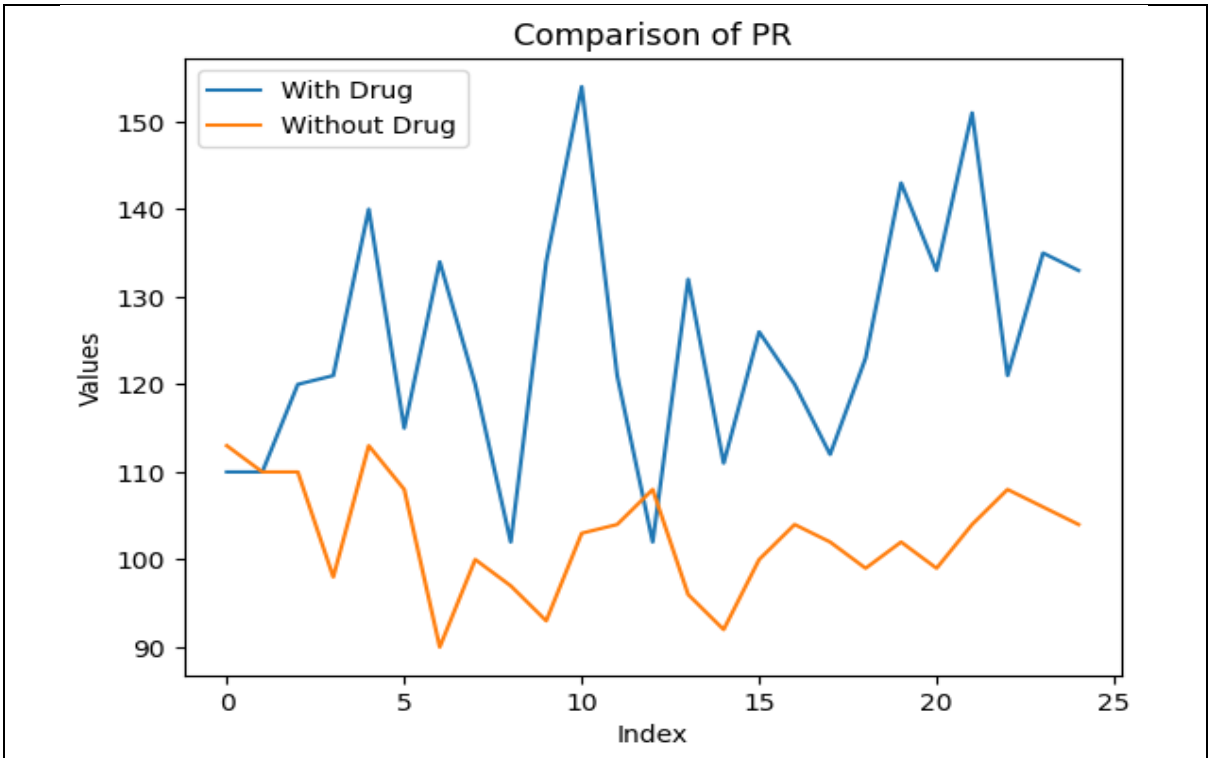


Graph 8 - Blood Pressure Changes



Graph 9 - Comparison of Pulse Rate -with and without Drug

Pearson correlation coefficient (r): -0.11194245052278107
P-value: 0.5942160491164841

DISCUSSION

Electroconvulsive therapy also known as electroshock is defined as electrical induction of seizures in extreme states of psychiatric illness to cause a therapeutic effect. ECT was first administered by injection of camphor in oil in a patient with catatonic schizophrenia by Dr,

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Meduna in 1935. The Schizophrenic symptoms temporarily disappeared after a normal convulsion.

Cerletti and Bini introduced the use of 70-120 volts for a duration at 0.7-1.5 sec to cause grandmal seizure with a tonic phase of 10-15 Seconds and Clonic phase of 30-60 seconds.
The electrodes may be placed temporally or frontally or unilaterally in the nondominant side.
In order to ensure airway protection, muscle relaxation and monitoring of hemodynamics, every patient now only undergo modified electro convulsive therapy (MECT)[4]

ECT works faster than drug and useful in averting acute suicidal tendencies. The mechanism of action of the influence of psychotic thought processes is not clearly kown however several theories have been postulated for mechanism of action namely neurotransmitter theory, neuroendocrine theory, psychological theory and brain damage theory.[5]

The neurotransmitters receive altered substances and the induced seizure are resisted by the brain, the seizure thus dampen the abnormally active brain circuits. Several articles discussing pros and cons of ECT were published during 1990's. Jenkusky et al wrote about a highly negative portrayal of ECT by the media cause of its decline.

Moreover the movie 'One Flew Over the Cuckoo's Nest' elaborated ECT as cruel and inhumane method to achieve behavioural control. The economy over Pharmacotherapy, efficacy in acute situations to improvement in short time especially in catatonic schizophrenia and pleasant and safe experience of modified ECT brought back the role of ECT in Psychotherapy.

Mechanism of action of ECT

ECT causes rapid increased dopamine concentration in frontal cortex and striatum
Dopamine autoreceptor sensitivity is diminished and D1 receptor agonists enhance stimulation of adenylate cyclase.ECT dimnishes muscarinic function in some CNS regions

- Enhanced Dopamine transmission
- Increased GABA A sensitive receptors
- ECS upregulates opioid receptors DADLE (D Alanine DLeucine Encephalin)
- Release of endogenous adenosine periods of electrical activity in CNS

Example Seizure promote extracellular release of adenosine in several receptor types to produce inhibition.

Caffeine is used to increase ECT seizure durations and antagonises adenosine A1 receptors - possible seizure termination mechanisms. Caffeine increases PDE inhibition and calcium release from intracellular stores.
ECT- memory impairment is due to changes in neurotransmitter mechanism.
ECT induced seizures cause a form of NMDA mediated long term potentiation inhibition which has been described in Rodent Hippocampal slices.
ECT leads to increased norepinephrine turnover and reduces presynaptic alpha2 adrenergic receptors.
5HT type 2 receptor inhibition
ECT- monoaminergic transmission
Second messenger system
Gene transcription

Indications for ECT

- Depression, Mania, Catatonia
- NLS, Motor symotoms of parkinson
- Intractable epilepsy
- Diagnostic statistical manual of mental disorder class 4

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Atropine causes tachycardia, memory impairment. Peripherally acting anticholinergic glycopyrollate scores over. Because the anticholinergic induced tachycardia can increase cardiovascular risk of ECT the decision to use these agents must be made on case by case basis. Patient with resting heart rate above 90/min do not need anticholinergics[6]

Several factors affected the seizure threshold while Old age, male gender, benzodiazepines, anticonvulsants and barbiturates, increased seizure threshold, young age, female gender, perylene tetrazol, vasopressor, alcohol withdrawal, Amphetamines, tricyclic antidepressants, phenothiazines, lithium and reserpine lower seizure threshold.

Cardiovascular response to ECT

Transient cardiovascular changes take place in pulse rate and blood pressure with an initial parasympathetic response followed by a sympathetic response with rise in pulse rate and systolic blood pressure which returns to original levels at an average of 6-10 minutes.

Bradycardia due to vagal effects of suxamethonium[7]

Missed or absent seizures are associated with increased likelihood of post stimulus asystole. Atropine may exacerbate total response crosses blood brain barrier.During ECT the sympathetic nervous system is activated with transient surge in BP and HR This can present a physiological challenge to patient with hypertension and Ischemic heart disease[2].

CONCLUSION

Electroconvulsive therapy has been recognised as a non-pharmacological intervention used prophylactically or therapeutically to sustain partial or complete remission of symptoms. Various animal experiments have confirmed neuroendocrine and neurotransmitter modifications and applications have been proved in clinical situations like Parkinsonism where motor disorder improvement occurs due to presumed increase in Dopamine release.

Cardiovascular response to electroconvulsive therapy proved alternating autonomic surges with an initial decrease or stable pulse rate during shock followed by sympathetic surge ,induced tachycardia and moderate hypertension.[8] .Routine use of prophylactic glycopyrrollate, a peripheral anticholinergic and reduce secretions and manage any eventful bradycardia has actually proved significant rise of heart rate from baseline of 80 per minute to 140 per minute in the 2^{nd} minute post MECT($p=0.5$) and may be determental to elderly patients and other patients with cardiac disorders(IHD) due to increasing myocardial oxygen demand.[9]

Moreover use of antipsychotics with anticholinergic properties such as phenothiazine combinations take care of secretions if any due to the induced seizures.[10]

In concurrence with Keith E.I Senberg et al and Perrin GM and Kaplan Saddock themselves it may be inferred the routine use of glycopyrrolate during induction in MECT may be thought of with caution especially in the elderly and those with cardiac compromise if their baseline heart rate is more than 80-90 per minutes, While a very significant rise in BP has not been noted.[11]

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