# “EVALUATION OF EFFECTIVENESS OF DEXMEDETOMIDINE ON ACUTE HAEMODYNAMIC RESPONSE IN PATIENTS UNDERGOING MODIFIED ELECTROCONVULSIVE THERAPY – A RANDOMISED CONTROL TRIAL”

**By**

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**KLE UNIVERSITY, BELAGAVI, KARNATAKA**

# ENDORSEMENT

This is to certify that the dissertation entitled **"EVALUATION OF EFFECTIVENESS OF DEXMEDETOMIDINE ON ACUTE HAEMODYNAMIC RESPONSE IN PATIENTS UNDERGOING MODIFIED ELECTROCONVULSIVE THERAPY – A RANDOMISED CONTROL TRIAL"** is a bonafide research work done by **REG NO. BA0113003.**


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# LIST OF ABBREVIATIONS USED

| | |
|---|---|
| ASA | American society of Anaesthesiologists |
| BMI | Body mass index |
| Bpm | Beats per minute |
| C group | Control group |
| CNS | Central nervous system |
| CVS | Cardiovascular system |
| D group | Dexmedetomidine group |
| DBP | Diastolic blood pressure |
| ECG | Electrocardiogram |
| ECT | Electroconvulsive therapy |
| F | Female |
| GIT | Gastrointestinal tract |
| Hb | Haemoglobin |
| HR | Heart Rate |
| Inj | Injection |
| IV | Intravenous |
| IP / OP No | In Patient / Out Patient Number |
| IBP | Invasive blood pressure |
| Kgs | Kilograms |
| M | Male |
| MAP | Mean arterial pressure |
| Mcg | Micrograms |
| Mg | Milligrams |
| Ml | Millilitre |

| | |
|---|---|
| Min | Minute |
| P | Probability |
| PR | Pulse rate |
| RBS | Random blood sugar |
| RPP | Rate pressure product |
| RR | Respiratory rate |
| S.D. | Standard Deviation |
| SBP | Systolic blood pressure |
| SD | Seizure duration |
| $SPO_2$ | Saturation percentage of oxygen |
| Sl. No | Serial Number |
| Wt | Weight in kilograms |
| α | Alpha |
| β | Beta |

# ABSTRACT

## Background

With administration of electroconvulsive therapy, blood pressure & heart rate increase secondary to rise in plasma adrenaline and nor adrenaline levels. Deaths reported to have occurred with electroconvulsive therapy are most often due to acute hemodynamic response to electroconvulsive therapy. Various drugs are being used to attenuate these responses. This study was designed to know the effectiveness of dexmedetomidine on acute haemodynamic response in patients undergoing modified electroconvulsive therapy.

## Objectives

To know the effectiveness of dexmedetomidine on acute haemodynamic response in patients undergoing modified electroconvulsive therapy.

## Methodology

This study was conducted in the Department of Anaesthesiology, KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Nehru Nagar, Belagavi, on 60 patients, undergoing modified electroconvulsive therapy between January 2014 to December 2014. It was conducted after the approval by the ethical committee and written informed consent was taken from the participant. ASA I and II patients in the age group of 18 and 50 years were included in the study. They were then randomly divided into two groups of 30 patients each: Group C (Control) and Group D (Dexmedetomidine) using a computer generated table.

Patients in Group C received normal saline infusion over 10 minutes, while patients in Group D received dexmedetomidine (1 microgram per kg) infusion over 10 minutes prior to electroconvulsive therapy.

**Results**

The baseline heart rates were similar in the two groups. After ECT the heart rate increased in both groups. The peak heart rate in the control group after ECT was 137.53 while it was 93.30 in the study group.

Though there was increase in heart rates in both the groups but the rise in study group was less as compared to control group which was statistically as well as clinically significant.

**Conclusion**

Pre-treatment with Dexmedetomidine significantly attenuates sympathetically mediated hemodynamic responses to Electro Convulsive Therapy.

**Keywords**

*Electroconvulsive therapy, Dexmedetomidine , haemodynamic responses.*

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## INTRODUCTION

Electroconvulsive therapy (ECT), formerly known as electroshock therapy and often referred to as shock treatment, is a standard psychiatric treatment in which seizures are electrically induced in anesthetized patients to provide relief from psychiatric illnesses.[1]

Electroconvulsive therapy is often used as a last line of intervention[2] for major depressive disorder, schizophrenia, mania and catatonia.[3] Electroconvulsive therapy is now also found to be effective in treatment of secondary psychiatric illness associated with various other diseases.[4]

For the safe conduct of electroconvulsive therapy, an effort to avoid or minimize the physiological sequelae and the associated complications of electroconvulsive therapy, a technique of modified electroconvulsive therapy has evolved gradually, with use of muscle relaxants and induction agents without the concomitant abolition of the beneficial effects. [5]

A usual course of electroconvulsive therapy involves multiple administrations, typically given two or three times per week until the patient is no longer suffering from symptoms. Electroconvulsive therapy is administered under anesthesia with a smaller dosage of induction agent and depolarizing muscle relaxant, succinylcholine.

Electroconvulsive therapy can differ in its application in three ways: electrode placement, frequency of treatments, and the electrical waveform of the stimulus.

These three forms of application have significant differences in both adverse side effects and symptom remission. After treatment, drug therapy is usually continued, and some patients receive maintenance electroconvulsive therapy[6].

Electroconvulsive therapy appears to work in the short term via an anticonvulsant effect mostly in the frontal lobes and longer term via neurotrophic effects primarily in the medial temporal lobe.[7]

Following application of the electrical stimulus during electroconvulsive therapy, there is a vagally mediated short lived bradycardia which is followed by a sympathetically mediated tachycardia and rise in blood pressure[8,9] as there is a sharp rise in the plasma catecholamine levels[10]. This produces a short lived sharp increase in myocardial workload which may pose significant risk for patients with coronary artery disease (CAD), pheochromocytoma, hypertension, cerebrovascular diseases[11]. Hence use of agents which would attenuate this transient adverse physiological consequence would be prudent.

Dexmedetomidine, an alpha 2 adrenoceptor agonist with a distribution half‑life of approximately 6 minutes has been successfully used for attenuating the stress response to laryngoscopy. Currently dexmedetomidine is indicated for intensive care unit sedation in mechanically ventilated patients and for perioperative and procedural sedation.

Sympathetically mediated change in the heart rate (Peak heart rate) is the simplest, noninvasive, time sparing and reliable indicator of magnitude of sympathetic response to electroconvulsive therapy.

We designed this study to determine if dexmedetomidine can serve as an effective alternative to the commonly used agents for blunting the hemodynamic response to electroconvulsive therapy. The aim of this study was to evaluate the effect of dexmedetomidine pre-treatment on hemodynamic response to electroconvulsive therapy.

# OBJECTIVE

To measure heart rate variability and peak heart rate following the modified electroconvulsive therapy.

# REVIEW OF LITERATURE

During 16th century, Paracelsus induced seizures by administering camphor by mouth to treat psychiatric illness. Until late 1930s, Metrazol was the agent used worldwide to induce convulsions.[12] Convulsive therapy was introduced in 1934 by Hungarian neuropsychiatrist Ladislas J Meduna who, believing that schizophrenia and epilepsy were antagonistic disorders, induced seizures in patients first with camphor and then cardiazol.[13] It was an Italian neuropsychiatrist Ugo Cerletti, who had been using electric shock to produce seizures in animal experiments, and his colleague Lucio Bini developed the idea of use electricity in convulsive therapy.

In the 1940s and early 1950s ECT was usually given without muscle relaxants and the seizure resulted in a full-scale convulsion. An anaesthetic was used by a few psychiatrists but most considered it unnecessary as the electric shock produced instant unconsciousness.[14]

A rare but serious complication of ECT without muscle relaxant was fracture or dislocation of the long bones, caused by the violent muscular contractions during the convulsion. In the 1940s psychiatrists began to experiment with curare, the muscle-paralysing South American poison, in order to modify the convulsions. The introduction in 1951 of succinylcholine, a safer synthetic alternative to curare, led to the more widespread use of modified ECT. A short-acting anaesthetic was usually given in addition to the muscle relaxant in order to spare patients from the terrifying feeling of suffocation that can be experienced with muscle relaxants.[14]

By early 1960s, anaesthesia for ECT composed of use of induction agent, muscle relaxant, oxygenation and ventilation.

Several clinical observational studies indicated that Electroconvulsive therapy was associated with adverse physiological alterations especially with respect to cardiovascular system, which at times even resulted in death. Brown, in 1952, showed that there was initial bradycardia following application of electric shock which was due to transient stimulation of cardio-inhibitory centre or nerves.[15] Following the brief bradycardia, Brown reported a prolonged tachycardia, where he observed cardiac arrhythmias and ascribed it to competitive influences of vagal and sympathetic activities.

Tewik and Wells reported that, out of 90 cases of death from electroconvulsive therapy 66 cases were due to cardiovascular complications. Kendell had also observed that death, when it occurs in conjunction to electroconvulsive therapy, was usually due to either myocardial infarction or ventricular arrhythmias.[16]

As the medical care and anaesthesia for electroconvulsive therapy was in the phase of evolution there were evidences to the fact that whenever deaths occurred in relation to electroconvulsive therapy [17], Bodley showed that it was the cardiovascular system which failed.[18]

In 1965, Gravenstein showed that, following administration of electric current there was a sharp increase in the heart rate and blood pressure with a corresponding increase in the plasma catecholamine levels.[19]

Since then various induction agents have been used with different benefits and drawbacks viz benzodiazepines, ketamine, barbiturates, beta blockers, clonidine and

dexmedetomidine.

A retrospective analysis has revealed that during post ECT, more than quarter of patients had systolic blood pressure exceding 220mm Hg and heart rate was between 120 to 140 in 20% of patients and 140 to 160 in 15% of patients. Such profound hyperdynamic state may be explained by enhanced sympathetic outflow following electroconvulsive therapy (ECT)[20]

In 1997 Europe research group studied on perioperative sympatholysis, i.e. beneficial effect of alpha2 agonist on hemodynamic stability and myocardial ischemia. They proved that alpha2 adrenergic agonists provide hemodynamic stability by blunting sympathetic response to nociceptive and surgical stimulation[21.]

In 1997 a double- blind, randomized and placebo controlled study was done to know the effect of clonidine and dexmedetomidine premedication on perioperative oxygen consumption and haemodynamic state, and found that during operation the maximum reduction in heart rate was 18% in the Dexmedetomidine and Clonidine groups compared to the placebo group. After operation the maximum decrease in systolic arterial pressure was 11%, diastolic arterial pressure was 15% and oxygen consumption was 17% in Dexmedetomidine and Clonidine group compared with placebo[22].

In 2009 a study was designed to compare the effects of premedication with dexmedetomidine and midazolam on post-electroconvulsive therapy (ECT) agitation and was found that premedication before ECT may be useful in managing treatment-resistant agitation after ECT, without adverse effects[23].

In 2011 a comparative study to know the efficacy and clinical profile of two α-2 adrenergic agonists, dexmedetomidine and clonidine, in epidural anaesthesia was done. They concluded that dexmedetomidine is a better adjuvant than clonidine in epidural anaesthesia as far as patient comfort and stable cardio-respiratory parameters are concerned[24].

In 2011 a randomised controlled study was formulated to know effect of Dexmedetomidine during tracheal intubation and perioperative period, they found that it significantly attenuated sympathoadrenal response to tracheal intubation and reduced perioperative anaesthetic requirement[25].

In 2012 a randomised study was designed to compare the effect of dexmedetomidine and remifentanil on hemodynamic change after direct laryngoscopy and tracheal intubation and found that dexmedetomidine was more effective in attenuating pressor response to instrumentation[26]

In 2012 a study with entropy analysis was done to know the effect of intravenous infusion of dexmedetomidine on perioperative haemodynamic changes and postoperative recovery and found that Dexmedetomidine, when administered as a pre-anaesthetic medication and intraoperative infusion, attenuated stress response to various noxious stimuli and maintained haemodynamic stability[27].

In 2012 Sukhminder Jit Singh Bajwa, Jasbir Kaur, Amarjit Singh etal in his study concluded Dexmedetomidine is an excellent drug as it not only decreased the magnitude of haemodynamic response to intubation, surgery and extubation but also decreased the dose of opioids and isoflurane in achieving adequate analgesia and anaesthesia, respectively[28].

In 2013 a retrospective comparison on hemodynamic effects of dexmedetomidine and esmolol in electroconvulsive therapy showed that Dexmedetomidine administration before anesthesia induction reduced the acute hemodynamic response compared with esmolol administration in the early period of ECT. Therefore, dexmedetomidine may be effective in preventing acute hemodynamic responses to ECT[29].

In 2013 a double blind, randomised, controlled study was designed to know the effect of dexmedetomidine on hemodynamic and recovery responses during tracheal extubation and found that Dexmedetomidine increased the incidence of bradycardia and hypotension, but did not cause side effects like respiratory depression, laryngospasm, bronchospasm, undue sedation and desaturation[30].

In 2014 a study by Gourishankar Reddy Manne, et al. concluded that low dose dexmedetomidine infusion in the dose of 0.4 mcg/kg/h effectively attenuated haemodynamic stress response during laparoscopic surgery with reduction in post-operative analgesic requirements[31].

# DEXMEDETOMIDINE

Dexmedetomidine is a highly selective, specific, and potent alpha adrenergic agonist (1,620:1 alpha2 to alpha1) This drug is the dextro-isomer and pharmacologically active component of medetomidine, which has been used for many years in veterinary practice for its hypnotic, sedative, and analgesic effects. Compared with clonidine, dexmedetomidine is seven to ten times more selective for alpha2 receptors and has a shorter duration of action than clonidine. Atipamezole is a specific and selective alpha2 receptor antagonist that rapidly and effectively reverses the sedative and cardiovascular effects of IV dexmedetomidine.

**STRUCTURE :**

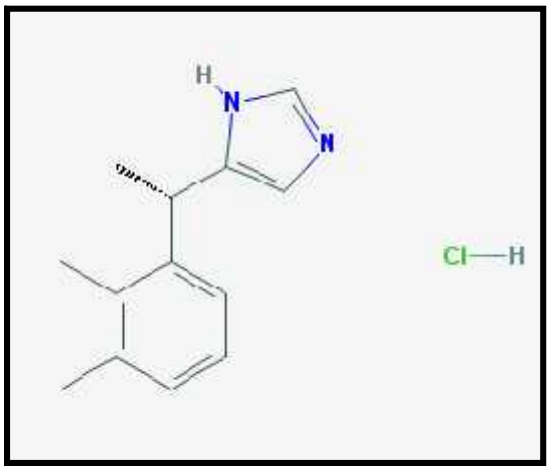



**FIGURE 1: Chemical Structure of Dexmedetomidine**

| | |
|---|---|
| **Chemical Names:** | Dexmedetomidine hydrochloride; Dexmedetomidine HCL |
| **Molecular Formula:** | $C_{13}H_{17}ClN_2$ |
| **Molecular Weight:** | 236.74048 g/mol |

**PRESENTATION:** It is a clear, colourless, isotonic solution containing 100micrograms / ml of dexmedetomidine base and 9mg /ml of sodium chloride in water. The solution is preservative free and contains no additives.

**Availability:**

Available as 1ml and 2ml ampoules containing 50µg in each ml

**Uses:**

- Anxiolysis
- Premedication in children
- ICU and Procedural sedation
- Adjuvant in caudal anaesthesia

**ROUTE OF ADMINISTRATION:** Intravenous, intramuscular, intranasal, caudal, and transdermal.

**DOSAGE:** Intravenous infusion of dexmedetomidine is commonly initiated with a 1 microgram per kg loading dose, administered over 10 minutes, followed by a maintenance infusion of 0.2 – 0.7 microgram per kg per hour.

**MECHANISM OF ACTION:** Dexmedetomidine, an imidazole compound, is the pharmacologically active dextro-isomer of medetomidine. It is a specific alpha-2 adrenoceptor agonist which acts via post-synaptic alpha-2 receptors to increase conductance through potassium ion channels.

Activation of the receptors in the brain and spinal cord inhibits neuronal firing, causing hypotension, bradycardia, sedation, and analgesia.

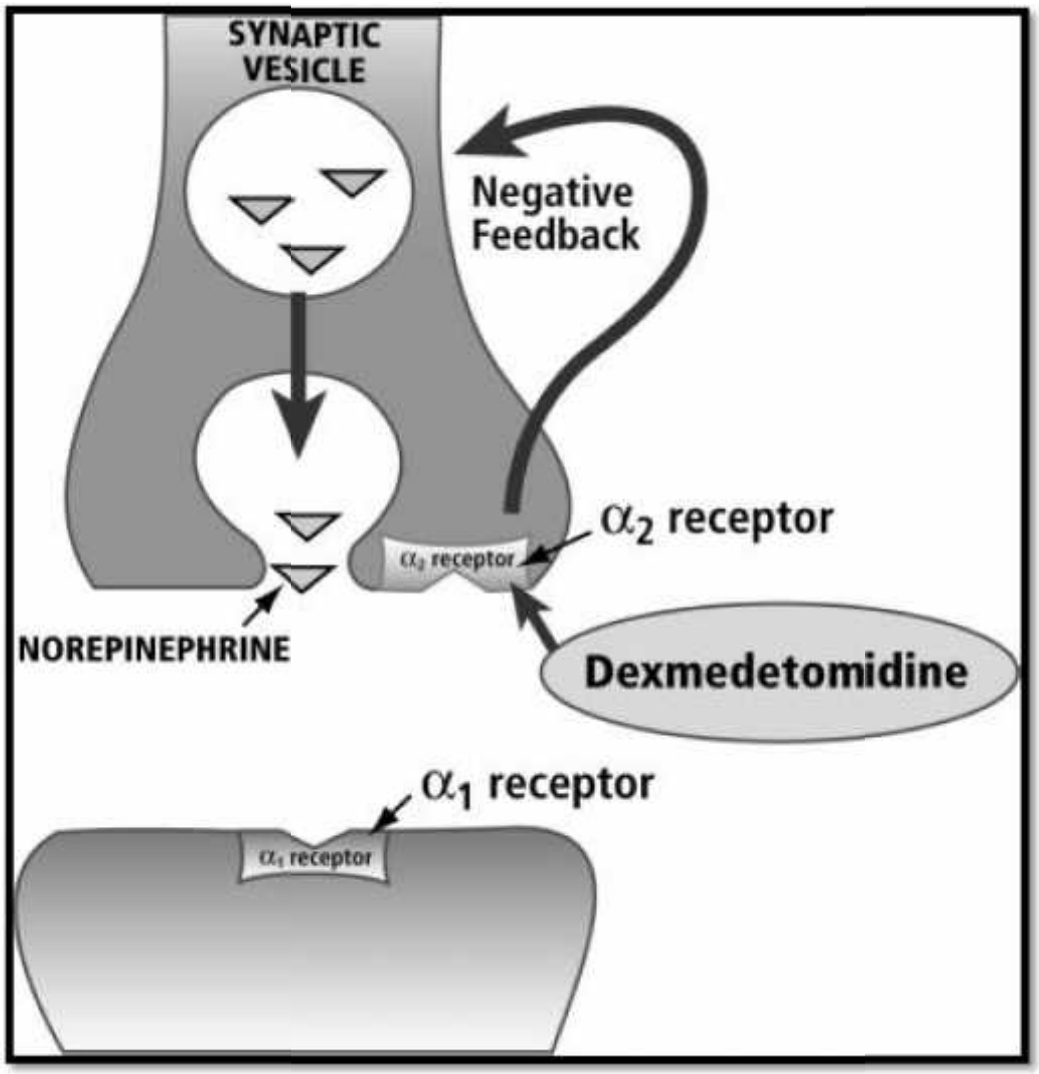



**FIGURE 2: PHYSIOLOGY OF THE $\alpha_2$-ADRENOCEPTOR AGONISTS RECEPTOR.**

**PHARMACODYNAMICS AND PHARMACOKINETICS**

**PHARMACODYNAMICS:**

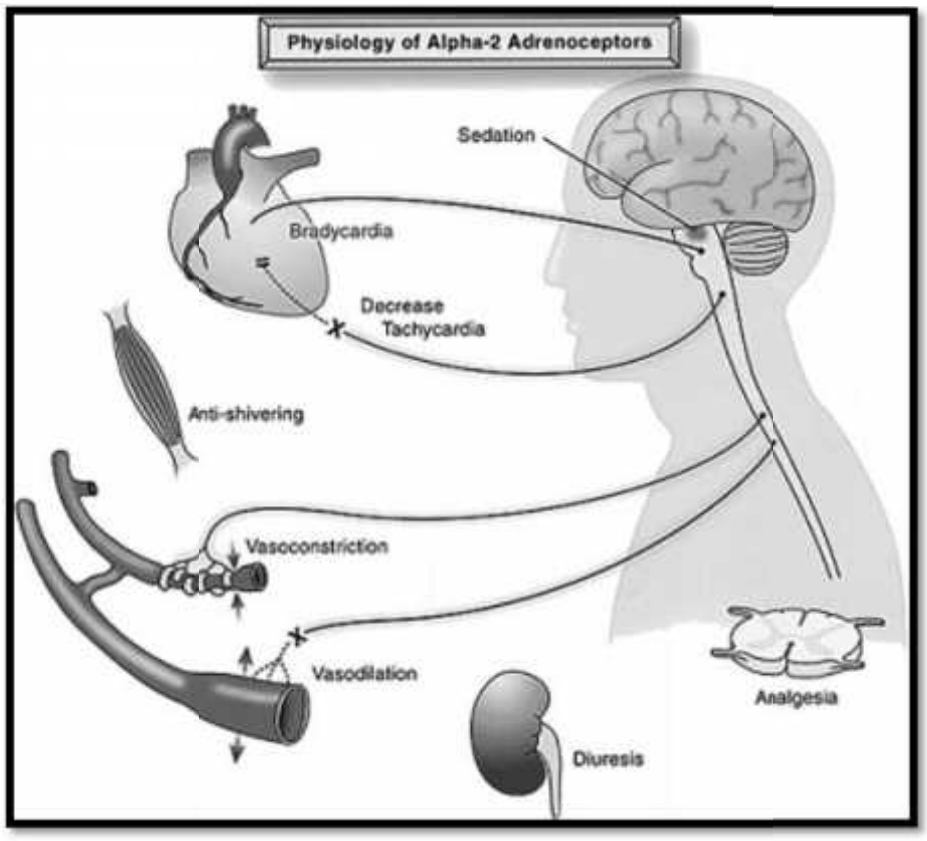



**FIGURE 3: RESPONSES THAT CAN BE MEDIATED BY $\alpha_2$-ADRENERGIC RECEPTORS**

CVS : The drug causes a predictable decrease in mean arterial pressure and heart rate.

RS : Dexmedetomidine causes a slight increase in $PaCO_2$ and a decrease in minute ventilation with minimal change in respiratory rate – these effects are not clinically significant.

CNS: The drug is sedative and anxiolytic. Ventilated patients remain easily arousable and cooperative during treatment. Reversible memory impairment is an additional feature.

GIT: The responses to activation of the receptors in other areas include decreased salivation, decreased secretion, and decreased bowel motility in the gastrointestinal tract;

RENAL: Juxtaglomerular cells in the kidneys participate in the control and release of renin. Renin release is stimulated by β-adrenoceptor mechanisms, whereas $\alpha_2$-adrenoceptor agonists directly inhibit renin release[32] and cause increased glomerular filtration, and increased secretion of sodium and water in the kidney.

OCULAR: Decreased intraocular pressure;

ENDOCRINE: Decreased insulin release from the pancreas[33].

METABOLIC: Dexmedetomidine causes a decrease in plasma adrenaline and noradrenaline concentrations. It does not impair adrenal steroidogenesis when used in the short term.

**PHARMACOKINETICS:**

DISTRIBUTION: Dexmedetomidine is 94% protein – bound in the plasma.

Volume of distribution is 1.33L/kg

The distribution half-life is 6 minutes[34].

METABOLISM: The drug undergoes extensive hepatic metabolism to methyl and glucuronide conjugates.

EXCRETION: 95% of the metabolites are excreted in the urine.

The elimination half life is 2 hours

The clearance is 39L/hour.

The drug shows a pharmacodynamic interaction with volatile agents and analgesic agents. The clearance is decreased in hepatic impairment although renal impairment does not significantly alter its pharmacokinetics.

TOXICITY:

The adverse effects of dexmedetomidine include hypotension, hypertension, nausea, bradycardia, atrial fibrillation, and hypoxia[35,36]. Overdose may cause first-degree or second-degree atrioventricular block. Most of the adverse events associated with dexmedetomidine use occur during or briefly after loading of the drug. By omitting or reducing the loading dose, adverse effects can be reduced. No study has described the long-term use of dexmedetomidine, but adaptive changes and withdrawal syndrome like those seen with the use of clonidine can be expected from dexmedetomidine.

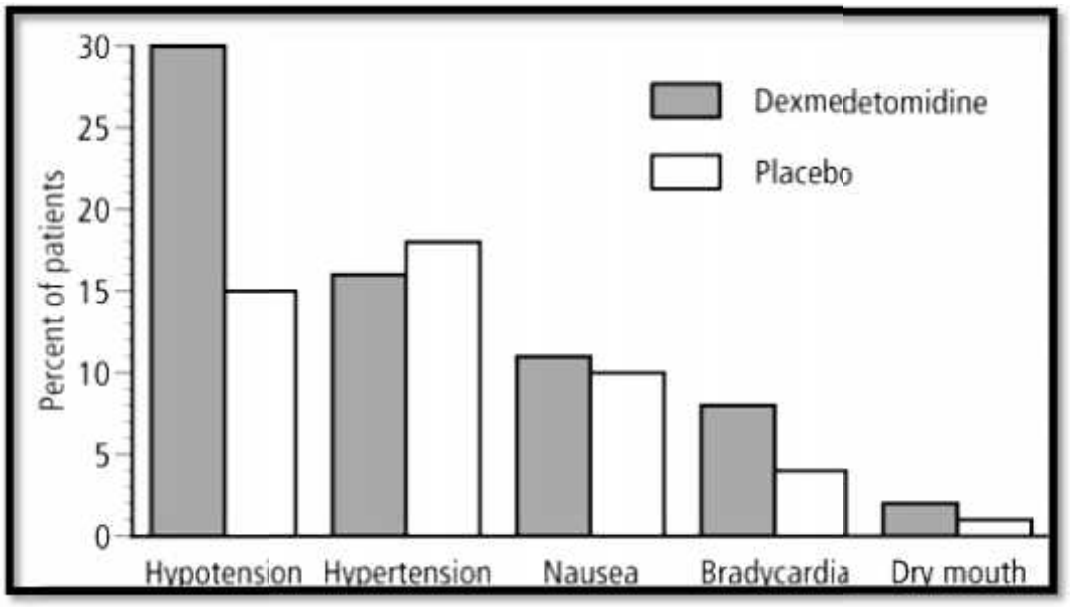



**FIGURE 4: Side Effects Of Dexmedetomidine**

# ELECTROCONVULSIVE THERAPY

## DEFINITION

Electroconvulsive therapy (ECT) is a procedure in which electric currents are passed through the brain (bitemporal or unilateral non dominant fronto temporal area) intentionally triggering a brief seizure.

Much of the stigma attached to ECT is based on early treatments in which high doses of electricity were administered without anesthesia, leading to memory loss, fractured bones and other serious side effects. ECT is much safer today and is given to people while they are under general anesthesia. The current is given with an aim to achieve a seizure of greater than 25-30 seconds duration by behavioral or electrophysiological criteria.

**Techniques used for electroconvulsive therapy administration are of two types -**

1. **Direct -** Electroconvulsive therapy is given in the absence of muscular relaxation and general anaesthesia.
2. **Modified -** Electroconvulsive therapy is modified by drug induced muscular relaxation and general anaesthesia

**TYPES OF ECT:**

1. Unilateral ECT
2. Bilateral ECT

**In unilateral ECT**, the current is just passed across one side. Both of them cause a seizure in whole of the brain. It is now used less. It had been thought to cause less memory loss, but recent research has shown that it is necessary to use larger doses of electricity to make it as effective as bilateral ECT. If the dose of electricity is increased to make it equally effective, the risks of memory loss are as great as with bilateral ECT.

**Bilateral ECT** the electrical current is passed across the whole brain. It seems to work more quickly and effectively; however, there has been concern that it may cause more side-effects.

**TECHNIQUE:**

ECT requires the informed consent of the patient[37]. Whether psychiatric medications are terminated prior to treatment or maintained, varies. However, drugs that are known to cause toxicity in combination with ECT, such as lithium, are discontinued, and benzodiazepines, which increase seizure thresholds, are either discontinued or a benzodiazepine antagonist is administered at each ECT session, or the ECT treatment is adjusted accordingly[37].

The placement of electrodes, as well as the dose and duration of the stimulation is determined on a per-patient basis.

In unilateral ECT, both electrodes are placed on the same side of the patient's head. Unilateral ECT may be used first to minimize side effects (memory loss). When electrodes are placed on both sides of the head, this is known as bilateral ECT.

In bifrontal ECT, an uncommon variation, the electrode position is somewhere between bilateral and unilateral. Unilateral is thought to cause fewer cognitive effects

than bilateral but is considered to be less effective if the dose administered is close to the seizure threshold.

The electrodes deliver an electrical stimulus. The stimulus levels recommended for ECT are in excess of an individual's seizure threshold about one and a half times seizure threshold for bilateral ECT and up to 12 times for unilateral ECT. Below these levels treatment may not be effective in spite of a seizure, while doses massively above threshold level, especially with bilateral ECT, expose patients to the risk of more severe cognitive impairment without additional therapeutic gains[38]. Seizure threshold is determined by trial and error ("dose titration"). Some psychiatrists use dose titration, some still use "fixed dose" (that is, all patients are given the same dose) and others compromise by roughly estimating a patient's threshold according to age and sex[39]. Older men tend to have higher thresholds than younger women, but it is not a hard and fast rule. Other factors, for example drugs, affect seizure threshold.

**DEVICES**

**FIGURE 5: ECT DEVICE**

Most modern ECT devices deliver a brief-pulse current, which is thought to cause fewer cognitive effects than the sine-wave currents which were originally used

in ECT. Typically, the electrical stimulus used in ECT is about 800 milliamps and has up to several hundred watts, and the current flows for between one and 6 seconds[38].

**MECHANISM OF ACTION**

Generalized electrically induced seizures of the central nervous system are responsible for the therapeutic effects of electroconvulsive therapy.

The psychobiological mechanisms remain largely unknown. Biochemical changes at the regional and sub cellular levels currently offer possible explanations. Neurophysiological changes include alterations in permeability of blood brain barrier, regional cerebral blood flow, cerebral microcirculation, neurometabolic activity and brain electrical activity[40,41]

Neuroendocrinal changes include release of adrenocorticotropin hormone, prolactin and hypothalamic peptides. Neurochemical changes include release of brain neurotransmitters and biogenic amines.

**PHASES OF ELECTROCONVULSIVE THERAPY [42] :**

The electroconvulsive therapy has been divided into six phases on the clinical basis as follows :

1. Preparational phase - before electrical dose
2. Stimulatory phase - immediately and after the stimulus dose
3. Kinetic phase-between stimulus and tonus state

a. Atonic -following stimulus

b. Tonic - similar to decerebrate posture

c. Clearly recognizable movements

4. Tonus phase - Generalized rigidity

5. Clonus phase - starts from eyes and proceeds down the feet

6. Recovery phase - includes transient atony, brief decerebrate state, normal respiration

**SEIZURE TIME AND THRESHOLD**

Preceded by a latent period of 2 - 3 seconds, a bilateral grand mal convulsion ensues a tonic phase of 10 -12 seconds followed by clonic phase of 30 - 50seconds. The seizure pattern of the individual patient varies only slightly regardless of stimulus characteristics.

Because seizure is the therapeutic agent, the duration of seizure is a significant variable of therapeutic efficacy. But it has certain limitations, since it only gives an incomplete description of the amount of seizure activity, and it does not reliably correspond to the therapeutic outcome.

Electrical stimulation in excess of what is needed can cause greater post - ictal confusion and memory loss without any therapeutic advantage. The seizure duration does not vary with sex of the patient, is inversely related to age and is reported to increase slightly as number of treatments increase. Increased oxygenation just prior to and during seizure, hypocapnia, drugs like Ketamine increase the duration of seizure, whereas hypoxic condition, hypercapnia, barbiturates decrease the duration of seizure.

For maximal effectiveness, the electrical stimulus must be of sufficient magnitude (approximately 70 - 150 volts for 0.3 to 1.0 seconds) to suppress the patient's variable seizure threshold and head resistance (200 to several thousand ohms) Seizure threshold varies with age, sex, drugs and physiologic condition of the patient.

It is higher in females, old patients. It can rise with coma, acute excitement, dehydration, previous seizure, and in cold dry days. Drugs like barbiturates, benzodiazepines, and local anaesthetics increase the seizure threshold in the dose related manner.

Threshold is lower in males and younger patients. It declines with water retention, vasospasm and hypoglycemia.

**PHYSIOLOGICAL EFFECTS**

Electroconvulsive therapy activates non-adrenergic system, enhances dopamine receptor sensitivity and reduces serotonin uptake. Electroconvulsive therapy activates peripheral autonomic nervous system and causes release of secretions from many endocrine glands. Neuroendocrine responses to electroconvulsive therapy include

- Immediate release (Peak Plasma level at 2 - 5 mins) of Adrenocorticotropic hormone, which returns to normal by 45 minutes.
- Increase in plasma epinephrine concentration to 15 times the baseline by 1 minute, which return to normal in 10 minutes.

- Increase in plasma norepinephrine to three times the baseline at 1 minute which returns to normal by 20 minutes.

The marked increase in levels of circulating catecholamines occasioned by electroconvulsive therapy is the result of their release from the adrenal medulla and to a lesser extent from sympathetic nerve endings. These increased levels are responsible for the hypertensive response.' The post seizure hypertension, tachycardia and cardiac dysrhythmias decline in parallel with the falling plasma concentration of catecholamines.

Transient increase in release of glucagon and inhibition of glucose mediated insulin secretion leads to hyperglycemia.

**Cardiovascular Changes: [43]**

Immediate changes are due to parasympathetic stimulation which are manifested as bradycardia and hypotension.

Later (After 1 min) changes are due to sympathetic stimulation which are manifested as tachycardia, hypertension and arrhythmias like asystole, bradycardia or tachycardia, ventricular premature complexes and ventricular ectopics. Because of these changes, cardiac output and myocardial oxygen consumption are increased.

**Cerebral Changes :[44]**

There is an increase in cerebral oxygen consumption and cerebral blood flow and this leads to increase in intracranial pressure.

**Miscellaneous**

Increase in intragastric pressure.

Increase in intraoccular pressure

**INDICATIONS:**

- Severe depression,
- Treatment-resistant depression,
- Severe mania,
- Catatonia,
- Schizophrenia
- Agitation and aggression in people with dementia,

ECT may be a good treatment option when medications aren't tolerated or other forms of therapy haven't worked. In some other cases where ECT is used:

- During pregnancy, when medications can't be taken because they might harm the developing fetus
- In older adults who can't tolerate drug side effects
- In people who prefer ECT treatments over taking medications
- When ECT has been successful in the past

**CONTRAINDICATIONS**

**Absolute:**

Pheochromocytoma, recent Myocardial Infarction, recent cerebrovascular accident, Intracranial surgery, Intracranial mass lesion

**Relative:**

Angina, congestive cardiac failure, cardiac pacemaker, severe pulmonary disease, severe osteoporosis, major bone fractures, glaucoma, retinal detachment, pregnancy.

**COMPLICATIONS**

- Damage to teeth, tongue, eyes, cutaneous structures.
- Muscle aches, headaches.
- Memory disturbances - complete retrograde amnesia.
- Fractures of long bones and vertebrae were the complications of unmodified electroconvulsive therapy, but have not been reported in past 10 years.

**MORTALITY**

Mortality attributable to electroconvulsive therapy is very low[45]. But in the past, deaths were reported due to pulmonary embolism and cardiac arrest.

# MATERIALS AND METHODS

The study was conducted after approval from institutional ethical committee at KLE'S Prabhakar kore Hospital & MRC, Belagavi during the period of January 2014 to December 2014.

**Study Design**

One year double blinded randomized controlled trial

**Source of Data:**

Adult patients undergoing modified electroconvulsive therapy procedure under short general anaesthesia at K.L.E.S. Hospital and M.R.C., Belagavi.

**Sample Size:**

Total sample size= 60 Patients (30 in each group).

- **Group C**

Control Group (normal saline) – 30

- **Group D**

Study Group (dexmedetomidine )– 30

Randomization was done by computer generated randomization chart.

**Sample Size calculation:**

Total sample size= 60 Patients

(30 in each group).

Using the formula

$$n = \frac{(Z\alpha + Z\beta)^2 \times 2 \times s^2}{(\bar{x}_1 - \bar{x}_2)^2}$$

type I error rate $\alpha = 0.05$ and

type II error rate $\beta = 0.02$ with a power of 80%

n = number of samples

Taking the level of significance at 5%($\alpha$=0.05) and power of the test as 80%($\beta$=0.2), we get $Z\alpha = 1.96$ and $Z\beta = 0.84$

**Inclusion Criteria**:

- ASA physical status I and II.
- Age between 18 and 50 years.
- Both male and female patients.

**Exclusion Criteria**:

- Pregnant patients.
- Hypertensive patients.
- Heart rate less than 60 per minute.
- History of allergy to dexmeditomedine.
- Pheochromocytoma

**Duration of Study:**

January 2014 to December 2014

**PRE ELECTROCONVULSIVE THERAPY WORK UP:**

Each patient was evaluated for medical and surgical illness in the past and previous anaesthetic exposure and experience.

Following investigations were carried out for all patients.

**INVESTIGATIONS:**

Hb %:

Urine routine: (Sugar, Albumin, Micro.)

Blood sugar

Serum creatinine

ECG

Chest X-RAY

**Methodology:**

After obtaining the written informed consent, a total of 60 patients confirming to the inclusion and exclusion criteria were included in the study.

Patients were randomly divided into two groups, Group D who were administered dexmedetomidine and Group C who were administered 0.9 % normal saline, by using computer generated table. An investigator who was not otherwise involved in the study prepared syringes containing saline or dexmedetomidine.

On the day of electroconvulsive therapy, each patient`s over night fasting was confirmed.

On arrival of the patient in the electroconvulsive therapy room, ECG, pulse oxymeter and non invasive blood pressure monitors were attached and baseline heart rate, systolic, diastolic and mean arterial pressures were recorded using a non invasive blood pressure monitor. An intravenous line was secured using a 20 G or 18 G intravenous cannula.

Ten minutes prior to the electroconvulsive therapy procedure, all patients received premedication of Inj. Glycopyrrolate 0.2 mg i.v. and infusion of normal

saline for control group or Dexmeditomedine for study group as per the following table.

| GROUP | Group C | Group D |
|---|---|---|
| | 50 ml Normal saline | 50ml Normal saline with Dexmedetomidine (1 mcg/kg) |

Following preoxygenation, patients were induced using thiopental sodium (3mg/kg) and muscle relaxation was achieved by Injection suxamethonium (0.5 mg / kg) and patients were ventilated with 100 % $O_2$ until fasciculations subsided. A mouth prop was inserted and a bitemporal ECT was administered by the psychiatrist. The mouth prop was changed to Guedels airway after the seizure and ventilation was assisted with the face mask and 100% oxygen until return of spontaneous respiration. The patients were observed for 10 minutes in the ECT room and later monitored in the recovery room for an hour.

**Readings were recorded in the following manner:**

| | GROUP C | GROUP D |
|---|---|---|
| Baseline Heart Rate | | |
| Heart Rate before ECT | | |
| Peak Heart Rate Following ECT within 5 min | | |

**MONITORING**:

1. Baseline Heart was noted before premedication.
2. Heart rate was recorded just before Modified electroconvulsive therapy.
3. Heart rate was recorded at every 15 seconds following modified electroconvulsive therapy for 5 minutes and the peak heart rate change was noted.

**STATISTICAL ANALYSIS:**

Data are presented as mean and standard deviation Statistical analysis was done by using the unpaired Student's't' test for quantitative data. Comparison of proportions (percentage) of the two groups was done using data analysis and $p<0.05$ would be considered significant.

# OBSERVATIONS AND RESULTS

This study was conducted to evaluate the effectiveness of dexmedetomidine on acute haemodynamic response in patients undergoing modified electroconvulsive therapy.

Sixty patients aged between 15-50 years, of both sexes belonging to ASA I and ASA II scheduled to undergo electroconvulsive therapy were included in the study.

**TABLE 1: Randomization of patients**

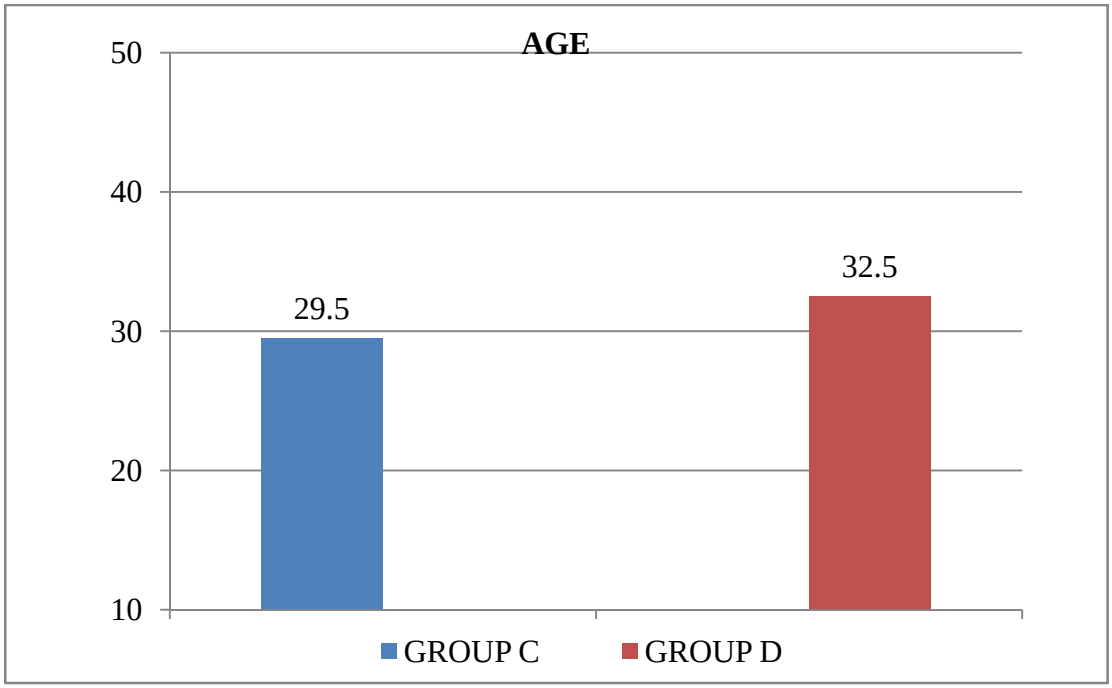
| GROUP | Received drug | Number of patients (n) |
|---|---|---|
| **GROUP C** | 50ml of Normal saline | 30 |
| **GROUP D** | 50ml Normal saline with Dexmedetomidine (1 mcg/kg) | 30 |

Table I: Shows the groups of patients studied and drug received.

**TABLE 2: Age Distribution**

| | **Group C** (Mean ± SD) | **Group D** (Mean ± SD) | **P VALUE** | **Statistical significance** |
|---|---|---|---|---|
| Age (years) | 29.5 ± 7.82 | 32.5 ± 8.37 | 0.157 | Not significant |

**GRAPH 1: Age Distribution**

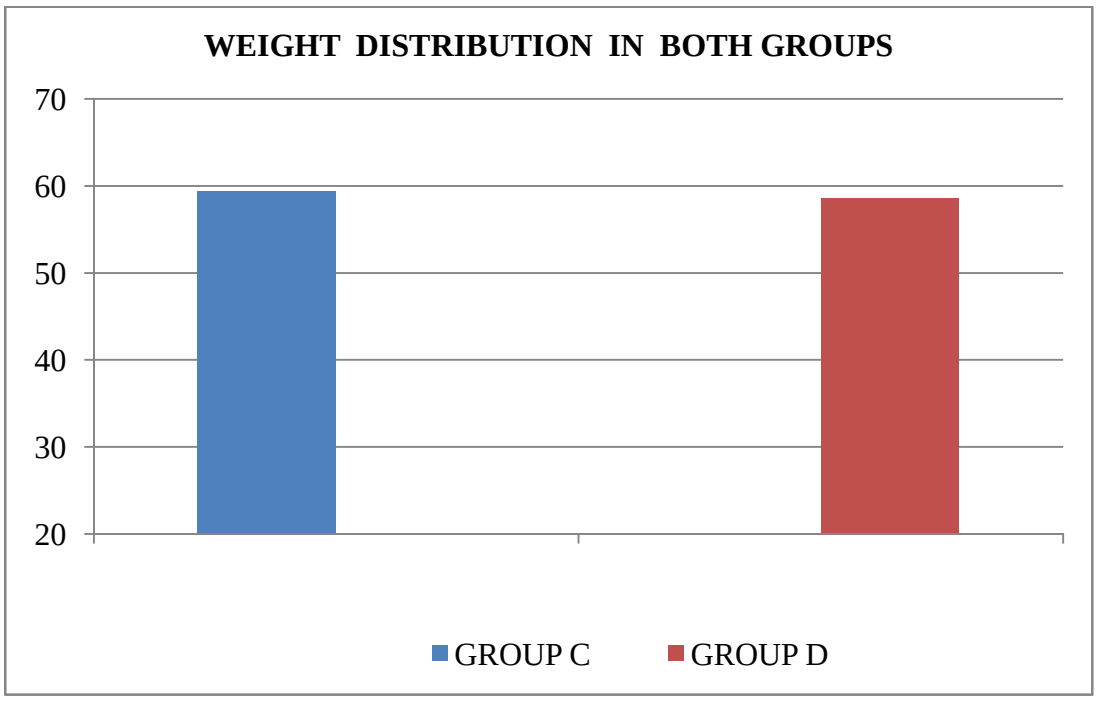



The Age of the patients in both the groups is presented in the table II. The two groups were comparable and there was no significant difference between the two groups

**TABLE 3: Weight Distribution**

| | **Group C** (Mean ± SD) | **Group D** (Mean ± SD) | **P VALUE** | **Statistical significance** |
|---|---|---|---|---|
| Weight(kg) | 59.4 ± 5.33 | 58.6 ± 4.57 | 0.519 | Not significant |

**GRAPH 2: Weight Distribution**

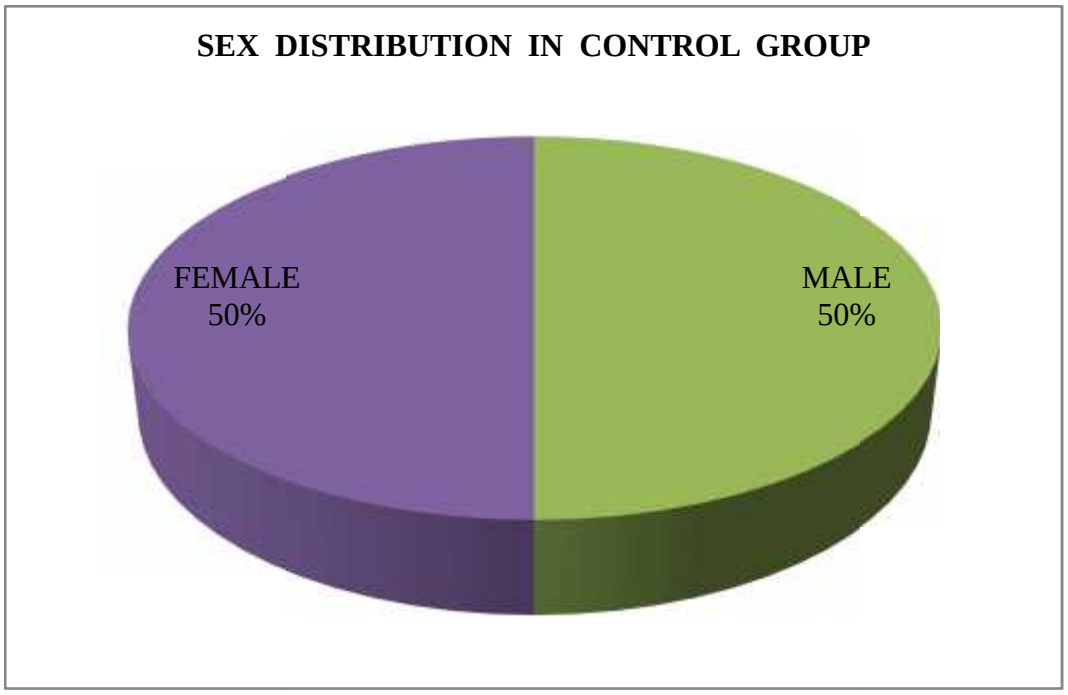



The Weight of the patients in both the groups is presented in the table III. The two groups were comparable and there was no significant difference between the two groups

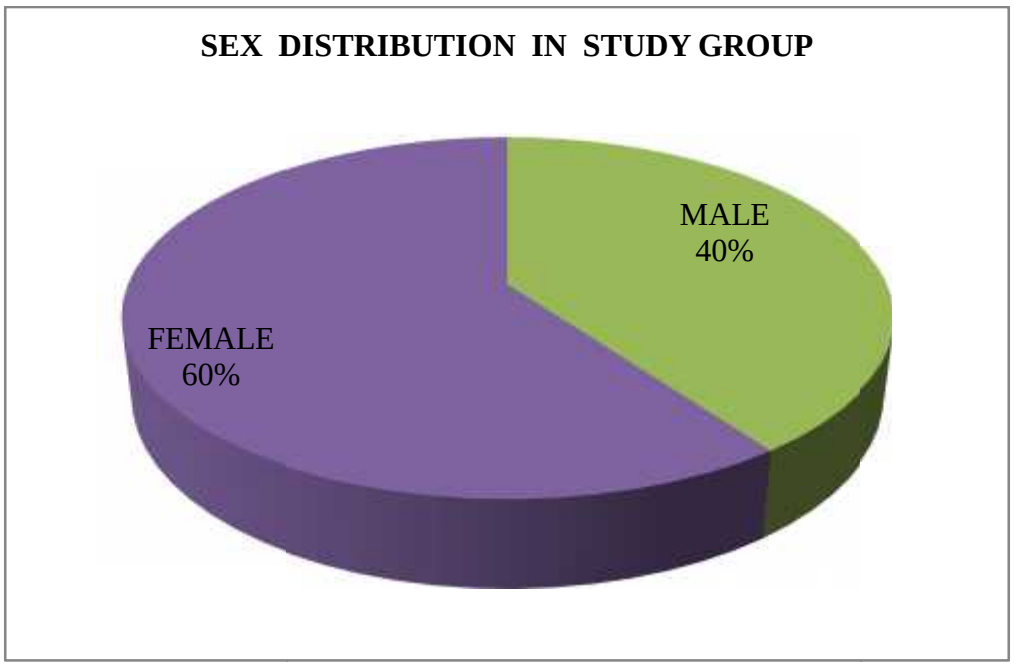
**TABLE 4 : Sex Distribution**

| | Group C | | Group D | | P VALUE | Statistical significance |
|---|---|---|---|---|---|---|
| | | % | | % | | |
| Male | 15 | 50% | 12 | 40% | 0.436 | Not significant |
| Female | 15 | 50% | 18 | 60% | | |
| Total | 30 | | 30 | | | |

**GRAPH 3: Sex Distribution in control group**



**GRAPH 4: Sex Distribution in study group**

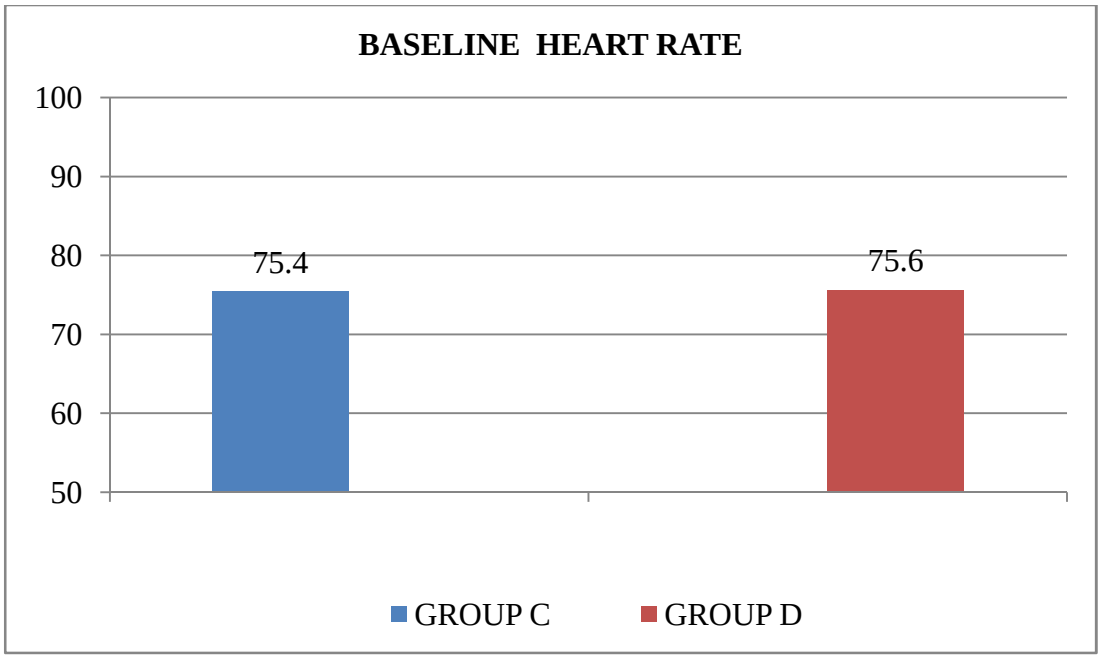



The male to female distribution in the two groups was as shown in table IV. There were 15 males and 15 females in control and 12 males and 18 females in study group. Female patients outnumbered male patients in study group.

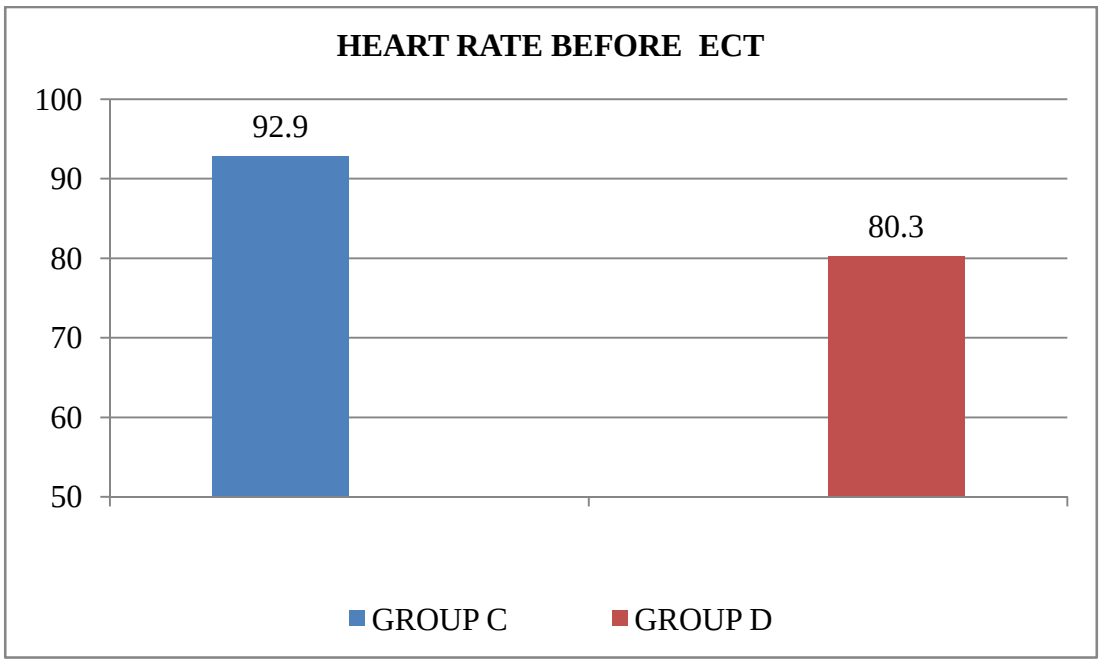
**TABLE 5: Heart rate Variability In Both Groups**

| | GROUP C | GROUP D | P VALUE | INFERENCE |
|---|---|---|---|---|
| Baseline Heart Rate(bpm) (*Before starting infusion of study drug)* | 75.40 ± 3.53 | 75.66 ± 2.83 | 0.75 | Not Significant |
| Heart Rate before ECT (bpm) | 92.96 ± 2.95 | 80.30 ± 3.49 | 0.001 | Significant |
| Peak Heart Rate Following ECT within 5 min (bpm) | 137.53 ± 6.30 | 93.30 ± 4.06 | 0.001 | Significant |

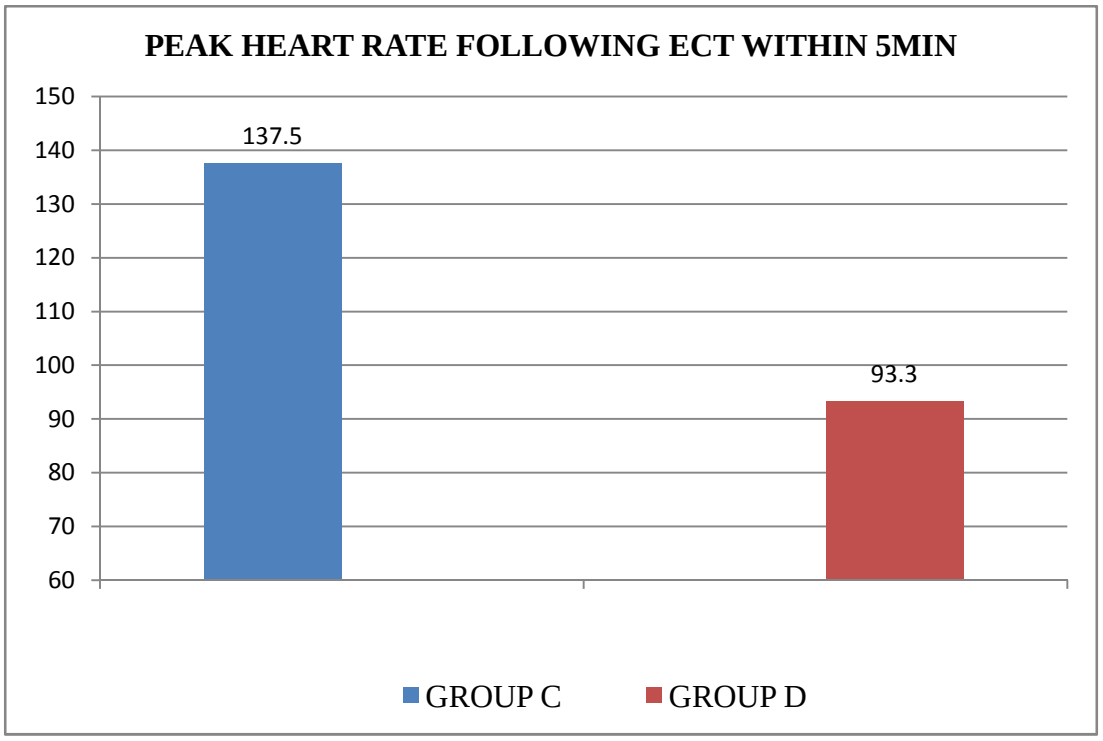
**GRAPH 5: Baseline Heart Rate**



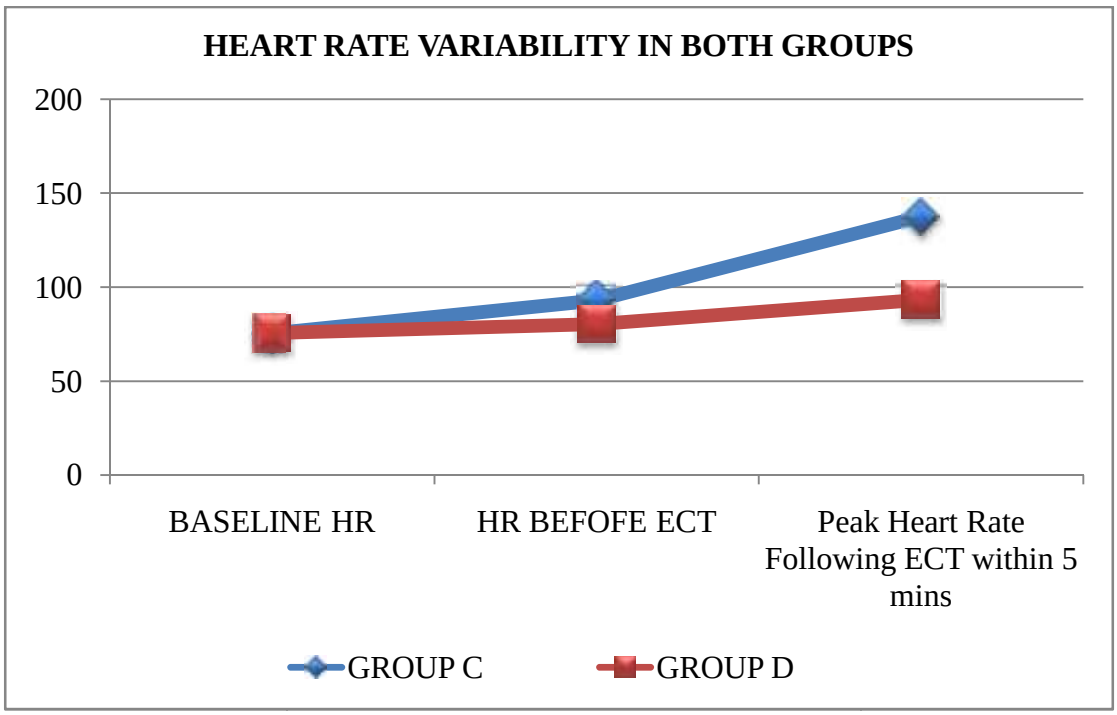
**GRAPH 6: Heart Rate Before ECT**




**GRAPH 7: Peak Heart Rate Following ECT within 5 minutes**



**GRAPH 8: Heart Rate Variability In Both Groups**




The mean heart rates in the two groups are as shown in table V. The baseline heart rate were similar in the two groups with no statistical difference. The mean heart rate in the control group before ECT was 92.96 and that of the study group was 80.30 respectively, while after ECT the heart rate increased in both groups. The mean peak heart rate in the control group after ECT was 137.53 while it was 93.30 in the study group. Though there was increase in heart rates in both the groups but the rise in study group was significantly less as compared to control group.

# DISCUSSION

ECT has a well-established role in the management of patients who have not responded to psychopharmacological treatment [46,47,48]. Many studies documenting the efficacy of ECT for depressive illnesses have been published, finding ECT superior to medications in the treatment of patients with severe depressive illnesses[46], particularly those with psychotic and suicidal symptoms [49].

The procedure consists of programmed electrical stimulation of the central nervous system to initiate seizure activity. The electroconvulsive shock is applied to one or both the cerebral hemisphere to induce seizure. The goal is to produce the therapeutic generalised seizure of 30-60 sec duration. The electric stimuli are usually administered until the therapeutic seizure is induced. A good therapeutic effect is generally not achieved until a total of 400-600 seizures seconds have been induced.

When the efficacy of ECT was discovered enthusiasm was tempered in the medical community because drugs were not used to control the violent seizure caused by the procedure, thus endangering a relatively high incidence of musculoskeletal injuries. Moreover, when a neuromuscular blocker was used alone, patients sometimes recalled being paralysed and awake just prior to the shock. So the routine use of general anaesthesia to ensure amnesia and neuromuscular blocakade to prevent injuries during ECT was started.

Seizure activity is characteristically associated with an initial parasympathetic discharge followed by a more sustained sympathetic dischrage. The initial phase is characterised by bradycardia and increased secretions. The hypertension and tachycardia that follow are typically sustained for several minutes. Transient

autonomic imbalance can produce arrhythmias and T wave abnormalities on the electrocardiogram. Cerebral blood flow, ICP, intragastric pressure and intra ocular pressure all transiently increase.

In the present study, 60 patients belonging to ASA I and II physical status of either gender, in the age group of 15 to 50 years who were scheduled to undergo modified electroconvulsive therapy were included.

They were randomly divided into two groups of 30 each as Control group and Dexmedetomidine group by computer generated randomization chart.

On the day of electroconvulsive therapy, each patient`s over night fasting was confirmed.

On arrival of the patient in the electroconvulsive therapy room, ECG, pulse oxymeter and non invasive blood pressure monitors were attached and baseline heart rate, systolic, diastolic and mean arterial pressures were recorded using a non invasive blood pressure monitor. An intravenous line was secured using a 20 G or 18 G intravenous cannula.

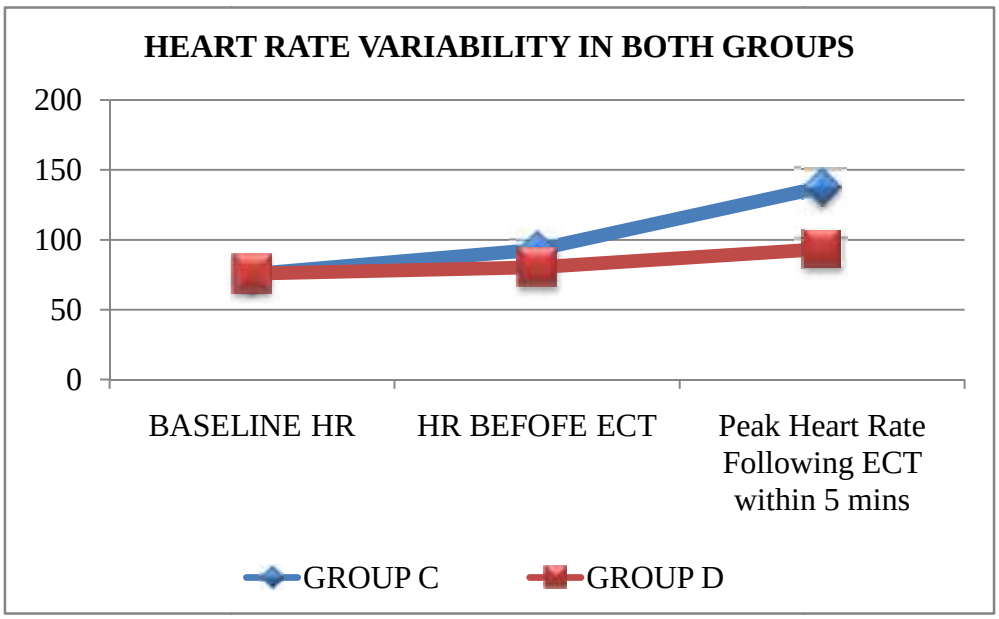
Ten minutes prior to the electroconvulsive therapy procedure, all patients received premedication of Inj. Glycopyrrolate 0.2 mg i.v for the antisialogogue effect as there is increase in secretions followed by electroconvulsive therapy and infusion of normal saline for control group and dexmedetomedine for study group were started and completed in 10 minutes.

| GROUP | Group C | Group D |
|---|---|---|
| | 50 ml Normal saline | 50ml Normal saline with Dexmedetomidine (1 mcg/kg) |

Following preoxygenation, patients were induced using thiopental sodium (3mg/kg) and muscle relaxation was achieved by Injection suxamethonium (0.5 mg / kg) and patients were ventilated with 100 % $O_2$ until fasciculations subsided. A mouth prop was inserted and a bitemporal ECT was administered by the psychiatrist. Seizure activity was noted in seconds. The mouth prop was changed to Guedel airway after the seizure and ventilation was assisted with the face mask and 100% oxygen until return of spontaneous respiration. The patient was observed for 10 minutes in the ECT room and later monitored in the recovery room for an hour.

Heart rate was recorded every 15 seconds for 5 minutes following modified electroconvulsive therapy and the peak heart rate was noted.

In our study baseline heart rates in Control and Dexmedetomidine groups were 75.40 bpm and 75.66 bpm respectively. Just before electoconvulsive therapy (pre ECT) heart rates in both Control and Dexmedetomidine groups were 92.96 bpm and 80.30 bpm respectively. This relatively lesser heart rate in Dexmedetomidine group was due to the effect of Dexmedetomidine which was statistically significant (92.96 bpm vs 80.30 bpm, p value is <0.001) . Peak Heart Rates following electroconvulsive therapy in 5 min in the Control and Dexmedetomidine groups were 137.53 bpm and 93.30 bpm respectively.




Increase in heart rate was seen in both the group but relative stability and trend of heart rate changes in Dexmedetomidine group was less as compared to control group which was statistically as well as clinically significant. (137.53 bpm vs 93.30 bpm and p value is <0.001).

Prolonged sympathetic response following the ECT is not well tolerated by majority of the patients. It results in tachycardia, hypertension, increased myocardial oxygen demand and possible dysrhythmias. The magnitude of this sympathetic discharge is highest following ECT.

To gain insight in their magnitude, a retrospective analysis of 23,000 clinical data points in 227 patients at Massachusetts General Hospital has revealed that post ECT, more than a quarter of patients had systolic blood pressures exceeding 220mmHg, heart rates were between 120 and 140 bpm in 20% of patients and between 140 and 160 bpm in 15% of patients. Such a profound hyperdynamic state may be explained by enhanced sympathetic outflow in the brain, augmented by significant increase in circulating norepinephrine and epinephrine[20].

Heart is a vital organ of circulatory system which pumps blood by rhythmic contractions. Heart being an aerobic organ, depends on oxygen for its activity and consumes oxygen proportional to its workload.

The two most prominent factors that determine this workload are heart rate and systolic blood pressure. The product of heart rate and systolic blood pressure termed as rate pressure product[50] (RPP) is a very reliable indicator of myocardial oxygen demand and is widely used clinically**.**

Monitoring peak heart rate following electroconvulsive therapy is the best noninvasive, cost effective and time sparing method of assessing degree /magnitude of neuroendocrine response.

Muhammed.et.al in their study opined that hyperdynamic cardiovascular response that occurs after ECT is a result of central activation of the autonomic nervous system. A brief period of parasympathetic discharge occurs immediately for the first 10–15 seconds after application of electrical current followed by sympathetic discharge. Within 10–12 seconds of the sympathetic surge caused by epinephrine and norepinephrine release, sinus tachycardia and arterial hypertension usually develop. Plasma epinephrine increases to fifteen times of the normal level, while plasma norepinephrine peaks can become three times higher than those under normal resting conditions, with their peak levels being attained within 60 s of electrical stimulation . Systolic blood pressure is transiently increased by 30–40% and heart rate is increased by 20% or more, resulting in a two- to fourfold increase in the rate-pressure product, an index of myocardial oxygen consumption. Hence these hemodynamic changes produce an abrupt increase in myocardial oxygen consumption. It may be beneficial

to administer drugs which blunt the hemodynamic stress response. Thus reinforcing the need for a suitable drug to attenuate stress response to ECT[51].

Dexmedetomidine, a highly selective α2 agonist, has significant sympatholytic and haemodynamic stabilizing property[52]. It causes dose-dependent decrease in heart rate and blood pressure[53]. Earlier studies have shown that dexmedetomidine attenuates stress response to intubation by decreasing central sympathetic outflow, thereby decreasing serum epinephrine and norepinephrine levels[54]. These findings are consistent with our study results which showed a significant decrease in change in heart rate in patients who were pretreated with Dexemedetomidine[55,56]

Dexmedetomidine has become one of the frequently used drugs in anaesthetic practice due to its stable haemodynamic profile, sympatholytic, sedative, anxiolytic, analgesic, neuroprotective and anaesthetic sparing effects. Other claimed advantages include minimal respiratory depression with cardioprotection, neuroprotection and renoprotection, thus making it useful in various situations including offsite procedures[57] such as in ECT.

Zakine Begec et.al. studied the effect of pre-treatment of Dexmedetomidine on post ECT hemodynamic response. The results of this study showed lower heart rate in Dexmedetomidine group at 0,1,3,10 minutes after ECT, the results of which are consistent with our results and observation[58].

Tanskanen *et al*. in their study showed that intraoperative infusion of dexmedetomidine at a rate of 0.4$\mu$g/kg/h maintained heart rate and blood pressure in acceptable range for a longer duration as compared to placebo group[53]. The decrease in heart rate and blood pressure was similar to the findings by Feld *et al*. who

compared dexmedetomidine with fentanyl in bariatric surgery[59]. Thus, showing that dexmedetomidine by its sympatholytic activity attenuates various stress responses during surgery and maintains haemodynamic stability.

Anish Sharma N.G et.al, compared the effects of premedication dose of dexmedetomidine with clonidine in attenuating pressor response to laryngoscopy & endotracheal intubation. The study concluded that both clonidine and dexmedetomidine attenuate the pressor response during laryngoscopy and intubation but Dexmedetomidine is better in attenuating the tachycardia response[60].

Cho JS observed that sympathetic hyperactivation during tracheal intubation prolonged the QT interval and increased the risk of arrhythmia and concluded that pretreatment with dexmedetomidine suppressed sympathetic hyperactivity and attenuated QTc prolongation during intubation[61].

Siddareddigari observed that infusion of dexmedetomidine 1.0 μg/kg prior to induction of anesthesia suppressed the hemodynamic response to tracheal intubation in normotensive patients and suppression in cardiovascular responses was found to be greater with dexmedetomidine than that resulted from infusion of esmolol 2.0 mg/kg[62]. Their study reinforces the importance of dexmedetomidine and superiority of dexmedetomidine over esmolol in attenuating sympathetic response.

## LIMITATIONS AND FUTURE SCOPE OF THE STUDY

The present study has some limitations such as small sample size, so further studies with larger number of sample sizes are needed to test the efficacy and safety of dexmedetomidine in electroconvulsive therapy.

Achieving therapeutic seizures (30-60 sec) is prerequisite for success of electroconvulsive therapy. Seizure threshold for electroconvulsive therapy is defined as “minimum amount of energy required to elicit seizures”. It depends on various factors like oxygenation, acid base status, electrolyte balance and anaesthetic drugs. The literature supporting actual role of dexmedetomidine on seizure duration is scanty and mixed. The effect of present study drug i.e, dexmedetomidine on seizure threshold can be measured by post-electroconvulsive therapy seizure duration and this parameter was not included in the methodology of present study.

# CONCLUSION

Pre-treatment with dexmedetomidine significantly attenuates sympathetically mediated hemodynamic responses to modified electroconvulsive therapy.

# SUMMARY

The aim of the study was to know the effectiveness of dexmedetomidine on acute haemodynamic response in patients undergoing modified electroconvulsive therapy.

We observed that the baseline heart rate were similar in the two groups. After ECT the heart rate increased in both groups. The mean heart rate in the control group after ECT was 137.53 bpm while it was 93.30 bpm in the study group. Though there was increase in heart rates in both the groups but the rise in study group was less as compared to control group which is statistically as well as clinically significant.

Based on the results obtained from our study we conclude that, pre treatment with dexmedetomidine significantly attenuates sympathetically mediated hemodynamic responses to modified electroconvulsive therapy.

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**ANNEXURE III – PROFORMA**

**"EVALUATION OF EFFECTIVENESS OF DEXMEDETOMIDINE ON ACUTE HAEMODYNAMIC RESPONSE IN PATIENTS UNDERGOING MODIFIED ELECTROCONVULSIVE THERAPY – A RANDOMISED CONTROL TRIAL"**

Name & address of the patient : _____

Age of the patient : Gender :

IP No. : Weight of the patient :

Random No :

Anaesthesiologist :

**PRE-ANAESTHETIC EVALUATION :**

Chief Complaints :

Past History :

1. Hypertension / Diabetes Mellitus / Asthma / Drug allergy
2. Previous exposure to anaesthesia

**Family History**

**General Physical examination:**

Weight: Height Temperature:

Pallor: Cyanosis: Pedal edema:

Clubbing:

Pulse: BP: RR:

M.P. Grading:

Mouth opening

Thyromental distance :

**SYSTEMIC EXAMINATION :**

Respiratory System :

Cardiovascular System :

Central Nervous System :

Per Abdomen :

Spine assessment :

**INVESTIGATIONS:**

Hb %:

Urine routine: (Sugar, Albumin, Micro.)

Blood sugar

Serum creatinine

ECG

Chest X-RAY

**ASA STATUS:**

**DIAGNOSIS:**

**ANAESTHETIC PROCEDURE:**

After obtaining the written informed consent, a total of 60 patients confirming to the inclusion and exclusion criteria will be included in the study.

Patients will be randomly divided into two groups, Group D who will receive dexmedetomidine and Group C who will receive 0.9 % normal saline, by using computer generated table. An investigator who is not otherwise involved in the study will prepare syringes containing saline or dexmedetomidine.

On the day of electroconvulsive therapy, each patient`s over night fasting will be confirmed.

On arrival of the patient in the electroconvulsive therapy room, ECG, pulse oxymeter and non invasive blood pressure monitors will be attached and baseline heart rate, systolic, diastolic and mean arterial pressures will be recorded using a non invasive blood pressure monitor. An intravenous line will be secured using a 20 G or 18 G intravenous cannula.

Ten minutes prior to the electroconvulsive therapy procedure, all patients will receive premedication of Inj. Glycopyrrolate 0.2 mg i.v. and infusion of normal saline for control group or Dexmeditomedine for study group as per the following table.

| **GROUP** | **Group C** | **Group D** |
|---|---|---|
| | 50 ml Normal saline | 50ml Normal saline with Dexmedetomidine (1 mcg/kg) |

Following preoxygenation, patients will be induced using thiopental sodium (3mg/kg) and muscle relaxation will be achieved by Injection suxamethonium (0.5 mg / kg). Patients will be ventilated with 100 % $O_2$ until fasciculations subside. A mouth prop will be inserted and a bitemporal ECT will be administered by a psychiatrist. The mouth prop will be changed to Guedels airway after the seizure subsides and ventilation will be assisted with the face mask and 100% oxygen till the return of spontaneous respiration. The patients will be observed for 10 minutes in the ECT room and later monitored in the recovery room for an hour.

**Readings will be recorded in the following manner:**

| | **GROUP C** | **GROUP D** |
|---|---|---|
| Baseline Heart Rate | | |
| Heart Rate before ECT | | |
| Peak Heart Rate Following ECT within 5 min | | |

**MONITORING**:

1. Baseline Heart rate will be noted before premedication.
2. Heart rate will be recorded just before Modified electroconvulsive therapy.
3. Heart rate will be recorded at every 15 seconds following modified electroconvulsive therapy for 5 minutes and the peak heart rate change will be noted.

**Signature of staff in charge:** **Signature of Guide**

## ANNEXURE IV – PHOTOGRAPHS


**PHOTOGRAPH 1. DEXMEDETOMIDINE**


**PHOTOGRAPH 2. ECT DEVICE**


**PHOTOGRAPH 3. HEMODYNAMIC MONITORING**

**PHOTOGRAPH 4. PATIENT RECEIVING ECT**

# ANNEXURE – V – KEY TO MASTER CHART

| | | |
|---|---|---|
| Age | - | Age In Years |
| Bpm | - | Beats per minute |
| ECT | - | Electro Convulsive Therapy |
| F | - | Female |
| HR | - | Heart Rate |
| IP.NO | - | In Patient Number |
| M | - | Male |
| Sl. NO | - | Serial Number |
| Weight | - | In kilograms |