



## EFFECTS OF INTRAVENOUS CLONIDINE ON HEMODYNAMIC RESPONSE TO ELECTRO CONVULSIVE THERAPY

### KEYWORDS

ECT, Hemodynamic response, clonidine.

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### ABSTRACT

**Background and Aims:** ECT stimulates sympathetic nervous system thereby causing tachycardia and hypertension. In our study, we evaluated the effects of IV clonidine on hemodynamic response to ECT. **Materials and methods:** This study was a randomized prospective double blinded cross over study. Thirty patients received 100ml of plain NS intravenously (Group S) and same patients received clonidine 1 µg/kg in 100ml NS (Group C). Hemodynamic parameters were recorded before and after the ECT. **Results:** In our study, there was statistically significant attenuation of raise of mean arterial pressure in clonidine group compared to NS group at different time intervals ( $P < 0.05$ ). There was statistically significant fall in heart rate in clonidine group compared to NS group ( $P < 0.0001$ ). **Conclusion:** clonidine at a dose of 1µg/kg attenuates the hemodynamic response of modified ECT.

### Introduction

Electroconvulsive therapy (ECT) is a procedure where a generalized epileptic seizure is purposely induced for the treatment of psychiatric disorders.<sup>1</sup> The indications for ECT are acute and medication resistant chronic depression, mania, schizophrenic patients with affective disorders, suicidal drive, delusional symptoms, vegetative dysregulation, inanition, and catatonic symptoms.<sup>2</sup>

ECT is performed under general anaesthesia with muscle relaxation to avoid the risks of long bone and vertebral fractures from violent muscle contractions.<sup>3</sup> ECT stimulates autonomic nervous system, with an initial parasympathetic induced bradycardia lasting 10 to 15 secs followed immediately by a more prominent sympathetic response that results in tachycardia and hypertension lasting 5 minutes or longer.<sup>4,5</sup> The hemodynamic response to ECT can produce myocardial ischemia and even infarction,<sup>6</sup> as well as transient neurologic ischemic deficits, intra-cerebral hemorrhages, and cortical blindness.<sup>7,8</sup>

Many drugs like beta blockers,<sup>9,10</sup> calcium channel blockers, local anaesthetics and  $\alpha_2$  agonist,<sup>11,12</sup> have been used by various routes in an attempt to blunt the hemodynamic effects of ECT. In our study, we evaluated the effects of intravenous clonidine a partial  $\alpha_2$  agonist on hemodynamic response to ETC.

### Materials and methods:

This study was a randomized, prospective double blinded cross over study. It was done at KAPV Govt medical college hospital from February 2016 to August 2016 after approval from the Medical Ethics Committee.

Thirty patients of ASA physical status I and II of either sex, between the ages of 18 to 60 years posted for ECT under general anaesthesia were assigned into two groups

Group S - Received 100ml of plain normal saline intravenously over 10minutes

Same patients were allocated as

Group C - Received clonidine 1 µg/kg in 100ml of normal saline intravenously over 10minutes

Patients with history of any cardiovascular, respiratory or central nervous system disorders were excluded from the study. Patients with heart rate less than 60 beats/min, systolic blood pressure less than 100 mm of Hg and difficult airway were also excluded from the study.

Pre- anaesthetic evaluation was done on the day before the procedure included history, general physical examination and routine investigations. A written informed consent was taken from their relatives. Patient were kept NPO for 8 hours and antipsychotic drugs continued. The study was done in repeated crossover pattern.

Patients were shifted to ECT room and connected to pulse oximeter, non-invasive blood pressure monitor and electrocardiographic monitor. Baseline vital parameters were recorded. An IV line (20G) was started, and all the patients were premedicated with IV glycopyrrolate 0.2 mg.

In ECT room study drugs were prepared by an Assistant professor who was not involved in this study. Administering drugs and monitoring were done by the principal investigator who did not know about the preparation. Study drug was administered over 10minutes as intravenous infusion.

All the patients were initially preoxygenated with 100% oxygen for 3 minutes and then induced with propofol 1mg/kg intravenously. immediately after the induction in one arm, the other arm was isolated by inflating a BP cuff above the systolic BP to assess the duration of motor seizure activity. Succinylcholine 0.5 mg/kg IV was then administered, and ventilation was assisted via a face mask with 100% oxygen. An adequate sized oral soft bite block was inserted to prevent tongue bite. Monitored Electroconvulsive Therapy Apparatus (MECTA) using bilateral stimulation was used to deliver a brief pulse stimulus for about 1to3 sec ( frequency 60–90 Hz - pulse width of 0.75msec) to produce seizures.

The effectiveness of ECT current was verified by appearance of tonic-clonic seizures in the isolated arm. Controlled or assisted ventilation was continued with 100 % oxygen until adequate spontaneous respiration returned.

HR and BP were recorded before the administration of the study drug (baseline) and 10 minute after the administration of infusion. HR and BP was then recorded before the ECT and at 1 minute, 3 minutes, 5 minutes and 10 minutes after the ECT shock. If the heart rate fall more than 20% from baseline 0.6mg of Atropine was given intravenously and if the systolic blood pressure fall more than > 20% from baseline 6mg of Ephedrine was given intravenously. Motor Seizure duration was noted and tourniquet deflated. Time to recover from anaesthesia was noted.

Data are expressed as mean  $\pm$  standard deviation. Paired students' t test was used for evaluation of demographic data, hemodynamic changes, seizure duration and recovery time.  $P < 0.05$  was considered statistically significant. All statistical analyses were done using SPSS version 16.0 statistical software

**Results:**

In our study thirty patients completing total of sixty ECT application had an average age of 37.70 ± 7.929 years, weight of 55.00 ± 6.843 Kg and male female ratio of 17:13.

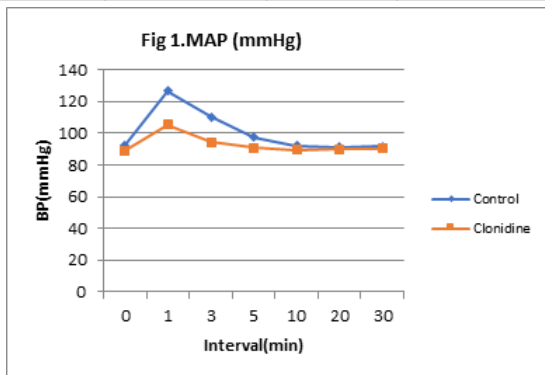
The mean durations of seizure activity was 36.57±3.42 sec in group S and 35.40±3.41 sec in in group C. The difference between both the groups were statistically not significant (p=0.191).

The mean basal heart rate in both the group were comparable (P>0.05), there was fall in mean HR 1 min,3,5,10 minutes after ECT in group C compared to raise of mean HR in group S and this difference was extremely statistically significant (P<0.0001). (Table 1).

	Group s	Group c	p value
Basal	84.32±5.31	84.35±5.34	0.9827
0 min	94.07±8.34	76.40±6.72	0.0001
1 min	126.50±9.41	83.80±5.56	0.0001
3min	101.03±8.70	81.10±5.03	0,0001
5 min	92.43±5.70	77.90±4.64	0.0001
10 min	88.17±5.29	78.47±4.91	0.0001

The difference in the mean arterial blood pressure(MAP) in the group C and the group S in the initial period after ECT (1minute,3minutes,5 minutes,10 minutes after ECT) were found to be statistically significant (p<0.05). (Table 2)(Figure 1).

	Group s	Group c	p value
0 min	92.6±4.86	89.17±4.34	0.0055
1 min	126.73±11.81	105.43±4.81	0.0001
2min	110.27±11.13	94.23±3.25	0,0001
5 min	97.67±9.01	90.67±2.63	0.0001
10 min	92.27±6.7	89.47±2.16	0.0334



The mean duration of recovery after ECT was 27.13±3.62 min in group C and 10.03±2.28 min in group S and this difference was statistically significant (P<0.0001).

**Discussion:**

ECT induced cardiovascular changes are due to central activation of the autonomic nervous system. Plasma epinephrine increases to 15 times normal level, and plasma norepinephrine peaks to 3 times normal level, with peak levels occurring within 60s of electrical stimulation.<sup>13</sup> Systolic blood pressure is transiently increased by 30–40% and heart rate is increased by 20% or more. clonidine is a selective α2 agonist at adrenoceptors. Its cardiovascular effects are mediated by central α2 adrenergic receptors and may involve inhibition of the sympathetic outflow and potentiation of parasympathetic nervous activity.<sup>11</sup>

In our study, there was significant fall in mean heart rate in clonidine group after ECT which is consistent with results obtained by

Ravichandra Dodawad et al,<sup>14</sup> but in study done by Fu Wen et al<sup>11</sup> there is no significant difference in heart rate.

In our study the increase in mean arterial pressure after ECT was more in plain normal saline group compared to clonidine group which is consistent with study done by Fu Wen et al<sup>11</sup> on oral clonidine effects on ECT. similar results were obtained by Ravichandra Dodawad et al,<sup>14</sup> on effects of 0.6µg/kg of dexmedetomidine in the attenuation of stress response after electroconvulsive therapy.

In our study, there is no significant change in seizure duration between two groups. same results were obtained by Fu Wen et al.<sup>11</sup> oral clonidine 0.05-0.3 mg did not alter the duration of either motor or EEG seizure activity after ECT in humans.

Time to recovery does not show significant changes in study by Fu Wen et al,<sup>11</sup> But in our study time to recovery is more in clonidine group which may be advantageous as it reduces postictal agitation in patients undergoing ETC.

**Conclusion:**

To conclude, clonidine at a dose of 1µg/kg attenuates the hemodynamic response of modified ECT without any change in the seizure duration with a slight delay in time for recovery.

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