Original article

ROLE OF FENTANYL IN ATTENUATION OF HAEMODYNAMIC CHANGES DURING ELECTROCONVULSIVE THERAPY

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Key words: **FENTANYL, HAEMODYNAMICS ,ELECTROCONVULSIVE THERAPY**

Abstract

BACKGROUND AND AIMS :
The use of Electroconvulsive therapy as a treatment modality has increased over the recent years. Electroconvulsive therapy (ECT) is associated with transient episodes of hypertension and tachycardia.Various agents like Beta-blocking agents, Opioids, Nitroglycerine and Clonidine are used to prevent cardiovascular response. Aim of our study was to assess the efficacy of Fentanyl on cardiovascular response and seizure duration during ECT. MATERIAL AND METHOD : 60 patients aged 18-60 years with ASA Grade I or II undergoing ECT were divided into Group A patients who received Inj.Fentanyl 1 mcg/kg intravenously and Group B patients who did not received Inj. Fentanyl 1 mcg/kg. The haemodynamic changes and seizure duration were recorded and tested by Mean, Standard deviation, Percentage, P value for significance. RESULTS : The haemodynamic changes after giving ECT were less in Fentanyl Group A ($P<0.05$) as compared to Nonfentanyl Group B and vitals came to baseline value earlier in Fentanyl group than Nonfentanyl group. Seizure duration was not affected by Fentanyl ($P>0.05$). CONCLUSION : Those patients receiving ECT when given Inj. Fentanyl 1 mcg/kg intravenously were seen to be more haemodynamicaly stable as compared to those patients who did not received Inj.Fentanyl intravenously.
KEYWORDS :

Electroconvulsive therapy (ECT), cardiovascular response, seizure duration, Fentanyl

INTRODUCTION

Electroconvulsive therapy (ECT) is a widely used and effective treatment for severe depression, schizophrenia, bipolar mood disorder, especially when alternative methods of treatment have failed. [2,5] ECT is a simple procedure, when appropriately administered. It is a safe and effective procedure in a wide variety of high-risk patients. However, ECT is accompanied by a cardiovascular response that can be dangerous in patients with cardiovascular disease. This response consists of an initial parasympathetic and a subsequent sympathetic reaction.[1,2,4,6,7] The essential elements of anesthesia for ECT include rapid loss of consciousness, effective attenuation of the hyperdynamic response to the electrical stimulus, avoidance of gross movements, minimal interference with seizure activity and prompt recovery of spontaneous ventilation and consciousness.[2,4] Fentanyl can be used as premedication to reduce haemodynamic response during ECT.[2,3,5,6,8] When it is used, there is transient increase in Heart rate and Blood pressure but it comes to preoperative status soon.

MATERIAL AND METHOD

This Comparative observational study was undertaken after Institutional Review Board approval.The present study was conducted on 60 patients of American society of Anesthesiologist Grade I or II, between 18-60 years of either sex posted for ECT.

The indications for ECT were Depression, Schizophrenia, Bipolar mood disorder etc. The patients with ASA grade III & above, like uncontrolled Hypertension, Diabetis mellitus, Thyroid dysfunction, Increased Intracranial pressure, Recent history of Ischemic heart disease, Pheochromocytoma, Retinal detachment or Glaucoma were excluded from the study.

All sixty patients were divided into two groups with thirty patients in each group. Group A patients who had received Inj.Fentanyl 1mcg/kg intravenously & Group B patient who did not received Inj.Fentanyl 1 mcg/kg.

In all patients, proper preoperative assessment was done. Routine investigations were done. Patients were asked to be Nil by mouth and a written informed consent of patient and relative was taken. All equipments for cardio-pulmonary resuscitation were kept ready.

After arrival to the Operation Theatre, baseline vital parameters of the patients were recorded using ECG monitor, Pulse oximetry and Blood pressure monitoring. An intravenous canula was inserted. Patients were premedicated with Inj. Glycopyrrolate 0.04 mg/kg and Inj. Fentanyl 1 mcg/kg intravenously. Patients were induced with Inj.Thiopentone sodium 3-4 mg/kg and Inj.succinylcholine 0.5-1 mg/kg intravenously. Ventilation was assisted with a face mask with a magill's circuit. Patients were pre-oxygenated with 100% Oxygen. Bite block was kept before application of the electrical stimulus to prevent injury in oral cavity, teeth , gum bleeding and laceration of tongue. Patients were held tight for immobilization to prevent fracture, joint dislocation and other complications. Then ECT was given.

During ECT noninvasive monitors were used to record Heart rate, Blood pressure, Oxygen saturation. Seizure duration was also noted. Patients were given 100% Oxygen after convulsion. During procedure awareness was assessed by PRST (Pressure, Rate, Sweating, Tear) score. Recovery was observed in the form of regain of reflexes, response to pain, and following verbal command. Patients were shifted in post –procedure room when they were following verbal command.

For statistical data processing tests were used: Mean, Standard deviation, Percentage, P value for significance.

RESULTS AND OBSERVATIONS

In our study in both the groups age, weight and sex ratio are not significant at 95% confidence limit ($p>0.05$).(Table-1)

There is no statistical difference found (Pulse, Systolic BP, Diastolic BP, SpO_2) between two groups before procedure. So both groups are appropriate for matching.

There is no statistical difference found even after Fentanyl premedication before ECT.

Non-Fentanyl Group B shows higher Pulse rate, Systolic BP and Diastolic BP than Fentanyl Group A at 30 sec, 60 sec, 90sec, 2min, 3min, 5min which is **statistically significant** at 95% confidence limit($p<0.05$).

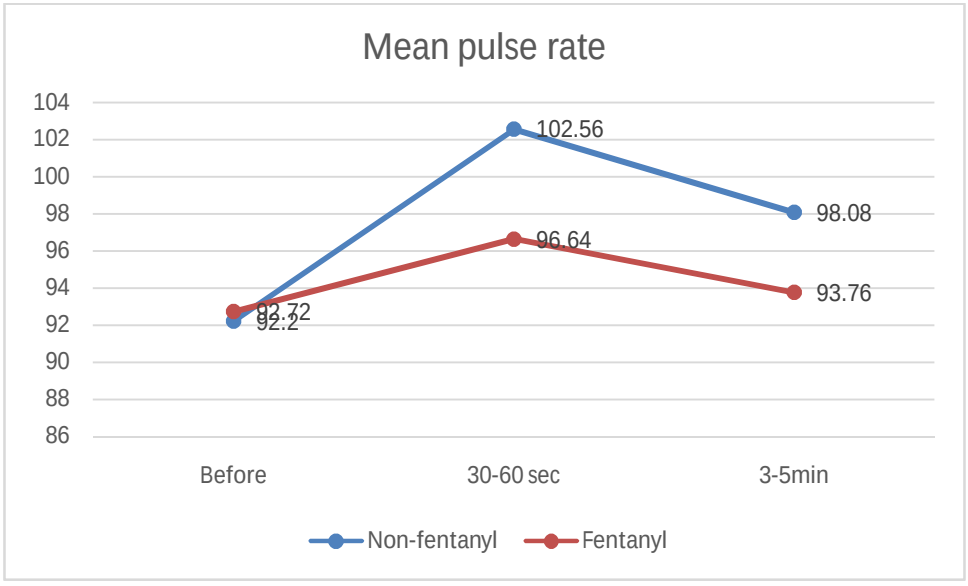
Control Group B (Non-Fentanyl) shows longer seizure duration than Fentanyl Group A at 30 sec, 60 sec, 90sec, 2min, 3min, 5min but it is **statistically NOT significant** at 95% confidence limit($p>0.05$).

There is no difference found in SpO_2 value between two groups.

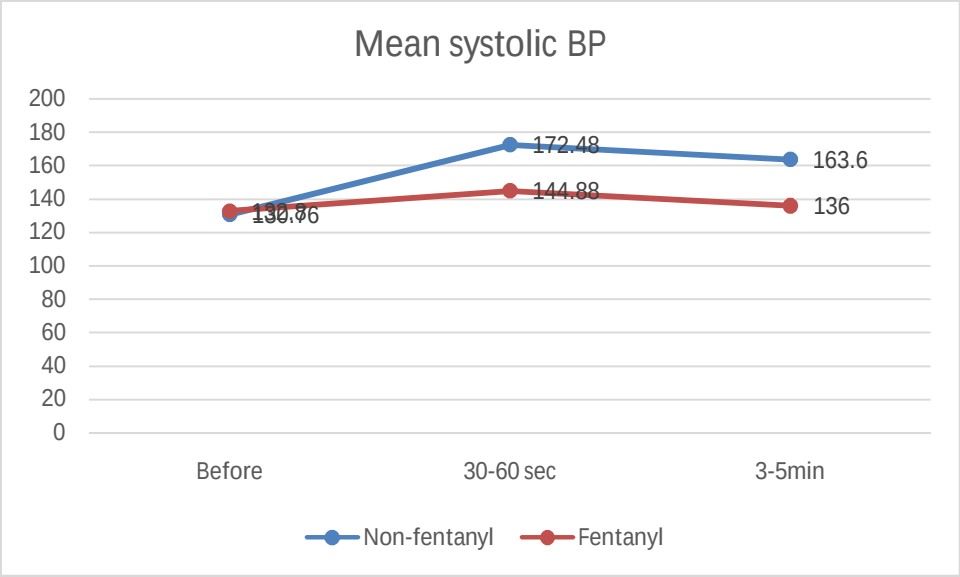
Thus our study indicates that the rise in BP and Pulse rate following ECT is less in Fentanyl group A than Non Fentanyl group B and attenuation of haemodynamic parameters occur earlier in Fentanyl group A than Non Fentanyl group B. The effect on saturation is not significant in both groups. So patients receiving ECT after giving Fentanyl show more haemodynamically stable profile.

	Fentnyl Group A	Non Fentanyl Group B
Age (Years)	36±0.4	35±0.7
Weight (Kg)	49±0.3	51±0.6
Sex (M:F)	27:33	29:31

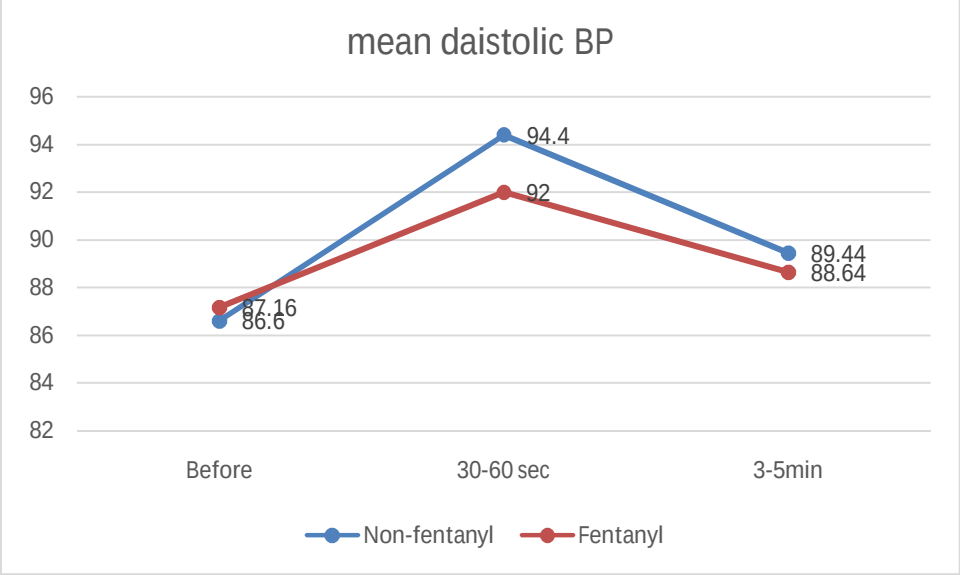
Table-1 : Demographic characteristics



Graph-1:Trend showing mean pulse rate in both group.



Graph-2:Trend showing mean systolic blood pressure in both group.



Graph-3:Trend showing mean diastolic blood pressure in both group.

DISCUSSION

The use of Eletroconvulsive therapy (ECT) to provoke a generalized epileptic seizure was first described byItalian neurologist, lucio bini and ugo cerletti in 1938 and was performed without anesthesia for almost 30 years. [2,4] Now the number of ECT procedures performed each year under general anesthesia.[2] The mechanism of action of ECT is not fully known. ECT affects multiple central nervous system components, including hormones, neuropeptides, neurotrophic factors and neurotransmitters.[7] During the electrical stimulus there is an immediate, brief and intense parasympathetic activity lasting 10–15 sec, which may cause a transient sinus bradycardia, hypotension

or rarely asystole.[2,6] So Atropine or Glycopyrrolate are given before induction of anesthesia to attenuate this vagal effect. Glycopyrrolate has superior anti-sialogogue effects, no adverse central nervous system effects as it does not cross blood brain barrier, and results in less Post-ECT tachycardia. Routine atropine premedication is not recommended due to detrimental effects on myocardial work and oxygen demand.[2,6] This transient vagal discharge is followed by sympathetic discharge, generally peaking at 3–5 min, amplified by adrenal release, which is responsible for the tachycardia and the hypertension observed after the stimulation. Myocardial oxygen consumption, as determined by the Rate–Pressure Product (RPP), therefore increases. RPP increases are more marked with ECT, in older patients and during hyperventilation-induced hypocapnia. Simultaneously, seizure activity increases tissue oxygen consumption, potentially reducing myocardial oxygen supply. Myocardial ischaemia and infarction can therefore occur, particularly with pre-existing disease.[2,6] Cerebral oxygen consumption, blood flow, and intracranial pressure also increase.[2,6] Cognitive adverse effects like Disorientation, impaired attention, and memory problems are frequent post-ictally, and short-term memory impairment may occur. This can be reduced by altering the stimulus intensity or waveform, using unilateral electrode placement, and lengthening the inter-ECT interval.[2,6] During the recovery period, the most common side effects are confusion, agitation, amnesia, and headache.[2] Nearly every Neurotransmitter system is affected by ECT, including Beta-adrenergic, Serotonin, Muscarinic, Cholinergic, and Dopaminergic systems.[7] It is proved that after ECT Epinephrine, Norepinephrine, Adrenocorticotrophic hormone (ACTH), Arginine vasopressin (AVP), and Cortisol level increased.[8]

Pretreatment screening and adequate management of cardiovascular risk factors remain the most important methods of preventing cardiovascular complications caused by ECT. In addition, attenuation of the cardiovascular response during ECT can be important in patients with cardiovascular disease. Many antihypertensive drugs have been administered in an attempt to attenuate the acute autonomic response to ECT. Previously Diazoxide, Hydralazine, β blockers (Esmolol, Labatolol, Propanolol) , Calcium-channel blockers (Nicardipine, Nifedipine) Direct vasodilators(Nitroglycerine) and α2 Agonists/Antagonists(Clonidine), Opioid analgesics(Remifentanyl, Alfentanil) have been used ; [2,4,6] however, the duration of action of these agents is longer than the ECT procedure and anesthesia time. However, when seizure duration is less than 15 seconds in both motor and EEG manifestations , the seizure was limited by insufficient electrical stimulation and that the treatment was inadequate. EEG seizure activity lasting from 25 to 50 s is alleged to produce the optimal antidepressant response. Patients experiencing an initial seizure duration of <15 s or >120 s achieve a less favorable response to ECT and the treatment may be inadequate.[2]

Fentanyl Citrate is a potent Opioid agonist with a rapid onset and short duration of action. It is a potent agonist of μ-opioid receptors in the brain which can be administered by the intravenous or intramuscular routes. The principal actions of therapeutic value are analgesia and sedation. Alterations in respiratory rate and alveolar ventilation associated with opioid analgesic may last longer than the analgesic effect. Fentanyl preserves cardiac stability and blunts stress-related hormonal changes. [8]

Pretreatment with fentanyl resulted in significant attenuation of the Norepinephrine peak after seizure ($P < 0.05$). Only Esmolol significantly attenuated ECT-induced Epinephrine secretion, whereas Fentanyl pretreatment significantly reduced release of ACTH after ECT.[8] The onset of action is from two to three minutes and the duration of action is one to two hours. The peak effect of a single intravenous dose of Fentanyl citrate is noted 5 to 15 minutes following injection. Fentanyl decreased heart rate and arterial blood pressure but did not significantly change stroke volume, cardiac output, central venous pressure, or peripheral arterial resistance. When given as premedication in ECT, it attenuates ECT induced cardiovascular response like tachycardia and hypertension.

CONCLUSION

This comparative observational study of 60 patients, with one group receiving Inj.fentanyl 1 mcg/kg as pre medication has shown more stable haemodynamic profile in terms of changes in Systolic BP and Heart rate than other group (Non-fentanyl). In Fentanyl group Systolic blood pressure and Heart rate increases minimally and returns to normal earlier than Non-fentanyl group. The seizure duration following ECT is also reduced in Fentanyl group though statistically not significant. In studies previously done by other researchers, were seen other effects of Fentanyl like respiratory inhibition and sedation. During this study respiratory parameters changes and sedation score are non-significant in both groups. Thus Fentanyl given in controlled dosage as pre medication smoothens induction and recovery of anesthesia as well as provides more stable cardiovascular response. It also provides efficient intra and post procedural analgesia. Thus as shown by this study opioids have shown a vital role as pre medication in short procedures like ECT.

REFERENCES

1. Weinger MB, Partridge BL, HaugherMirow A.:Prevention of the cardiovascular and hemoendocrine response to ECT, effectiveness of pretreatement regimen on Shemodynamics – Anaes. Analg.:1991.Nov;73(5):556-62
2. Ding,ZhengnianMD, White, Paul F.PhD, MD. Anaesthesia for ECT- : Anaes and anal : May 2002-vol 94, isssue 5:1351-64
3. Recart A, Rawal S, White PF, Byerly S, Thornton L. The effect of remifentanyl on seizure duration and acute hemodynamic responses to elecroconvulsive therapy.- Anaes. Analg.:2003 Apr;96(4);1047-50
4. Rashmi Pal, Virendra Singh Pal. Anaesthesia for ECT : A Noble Approach –
    a. Int J Med Res Health Sci. 2015;4(4):890-895
5. De Santis C, Delmante D, Stankovic S, Barbini B, Colombo C. IRCCS San RaffaleTurro University Vita – Salute San Raffale, Fentanyl with sodium thiopental or propofol in ECT Anaesthesia : Pilot Study
6. Vishal Uppal, MBBS DA FRCA,Jonathan Dourish, MB ChB MRCPsych,Alan Macfarlane, BSc MBChB MRCP FRCA, Anaesthesia for electroconvulsive therapy , :Contin Educ Anaesth Crit Care Pain (2010) 10 (6): 192-196.
7. Raj K Kalatapatu,MD; Chief Editor : Dennis M Popeo, MD more….Electroconvulsive Therapy medscape, Updated : Jan 12,2015.
8. Weinger MB[1], Partridge BL, Hauger sR, Mirow A, Brown M. Prevention of the cardiovascular and neuroendocrine response to electroconvulsive therapy: II. Effects of pretreatment regimens on catecholamines, ACTH, vasopressin, and cortisol. Anesth Analg. 1991 Nov;73(5):563-9.