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Comparative Study of Hemodynamic Changes during Induction of Anesthesia between Etomidate and Propofol

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Abstract

An ideal inducing agent for general anesthesia should have hemodynamic stability minimal respiratory side effect and rapid clearance with minimal side effects and drug interactions this study, is an attempt to evaluate and compare the beneficial and also side effect of propofol and etomidate. The patients were randomly allocated into two groups (group E and group P) with equal n = 40. A prospective double blind, parallel randomized controlled clinical study was done and result were analyzed, Inetomidate group heart rate did not significantly changed compared to pre-induction (p>0.05). But in propofol group, post induction heart rate significantly decreased (p<0 001) and after intubation, significantly increased (p<0.001) compared to the pre-induction. There was no significant change in systolic blood pressure in post induction and after intubation compared to pre induction value, as p value is >0 05. But in propofol group SBP decreased significantly in post induction (p<0.0001), and after intubation (p<0. 0001) SBP again increased. It was seen that among Group E patients, no patient had pain on injection and among Group P patients 8 had grade I pain and 18 had grade II pain P value <0.0001. There is no significant ECG and SPO_2 changes seen between two groups. Side effects like pain on injection, myoclonus and nausea was more pronounced with propofol. Only vomiting is more frequent with etomidate, Also further studies need to be conducted with a larger sample size to corroborate the finding of this study, which may enlighten further the usefulness of etomidate as inducing agent specially in hemodynamically unstable patient.

Keywords: General Anesthesia; Etomidate; Propofol; Myoclonus; Pain

Introduction

General anesthesia is usually induced most commonly in an anesthetic room adjacent to the operation theatre Most general anesthetics are induced either intravenously or by inhalation. Intravenous injection works faster than inhalation, taking about 10-20 sec to induced total unconsciousness. This minimizes the excitatory phase and thus reduces complications related to the induction of anesthesia. Inducing agents are drugs that are given intravenously in an appropriate dose, causes rapid loss of consciousness. Induction agent are used to induce anesthesia prior to other drugs being given to maintain anesthesia. An ideal inducing agent for general anesthesia should have hemodynamic stability minimal respiratory side effect and rapid clearance with minimal side effects and drug interactions. Etomidate is a carboxylate imidazole containing compound characterized by hemodynamic stability, minimal respiratory depression and cerebral protective effects. Its lack of effect of sympathetic nervous system and its effect of increased coronary perfusion even of patient with moderated cardiac dysfunction makes it and induction agent of choice. Etomidate is a hypnotic drug without analgesic activity, Intravenous injection of Etomidate produces hypnosis characterized by a rapid onset of action, usually within one minute. Duration of hypnosis is dose dependent but rela-

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tively brief, usually three to five minutes when an average dose of 0.3 mg/kg is employed. Immediate recovery from anesthesia (as assessed by awakening time, time needed to follow simple commands and time to perform simple tests after anesthesia as well as they were performed before anesthesia), based upon data derived from short operative procedures where intravenous, Etomidate was used for both induction and maintenance of anesthesia, Etomidate formulations for clinical use contain the purified R (+)-enantiomer[1]. Etomidate has a pK a of 4.2 and is hydrophobic at physiologic pH To increase solubility, it is formulated as a 0.2% solution either in 35% propylene glycol or lipid emulsion (Etomidate-Lipuro; B Braun, Melsungen, Germany), Etomidate appears to facilitate GABA minergic neurotransmission by increasing the number of available GABA receptors, possibly by displacing endogenous inhibitors of GABA binding. At a dose of 0.2 to 0.3 mg/kg, etomidate reduces Cerebral blood flow by 34% along with cerebral metabolic rate by 45% and reduces intracranial pressure, while cerebral perfusion pressure is maintained or increased during etomidate-induced anesthesia. Electroencephalographic changes during hypnosis with etomidate are similar to those seen with barbiturates, Bispectral index monitor values drop following etomidate bolus administration and return to baseline during recovery of consciousness. During brief etomidate infusions, bispectral index values correlate well with sedation scores, Etomidate increases latency and decreases amplitude of auditory evoked potentials[2]. The duration of epilepti form activity following electroconvulsive therapy is longer after anesthetic induction with etomidate versus methohexital or propofol. Somatosensory evoked potential amplitudes are enhanced by etomidate, and motor evoked potential amplitudes are suppressed less by etomidate than propofol, thiopental, or methohexital, Etomidate has very minimal cardiovascular effects Etomidate has less effect on ventilation than other anesthetics used to induce anesthesia. It does not induce histamine release either in healthy patients or in patients with reactive airway disease. Ventilatory response to carbon dioxide is depressed by etomidate Induction with etomidate produces a brief period of hyperventilation, sometimes followed by a similarly brief period of apnea which results in a slight (±15%) increase in $PaCO_2$, but no change in the partial pressure of arterial oxygen (PaO_2) [3]. Ledingham and Watt in 1983 first raised concerns regarding long-term etomidate infusion in critically ill patients. They postulated that adrenocortical suppression secondary to long-term etomidate infusion was the cause of the increased mortality. The specific endocrine effects manifested by etomidate are a dose-dependent reversible inhibition of the enzyme 11 β-hydroxylase. The blockade of the cytochrome P-450–dependent enzyme 11 β-hydroxylase also results in decreased mineralocorticoid production and an increase in intermediaries (11-deoxycorticosterone) Propofol is insoluble in water and therefore was initially prepared in with cremophor EL because of anaphylactoid reactions associated with cremophor EL, the drug was reformulated using soya been oil emulsion. Propofol is non barbiturate short acting intravenous anaesthetic agent. It is a phenol derivative (2,6-Diisopropylphenol) Propofol injection is a sterile, nonpyrogenic emulsion containing 10 mg/ml of Propofol suitable for intravenous administration[5]. Propofol produces decrease in systemic arterial blood pressure due to decrease in sympathetic mediated systemic vascular resistance (Robinson et al, 1997). It is more effective than Thiopentone in blunting the hypertensive response of intubations, Propofol decreases cardiac contractility and preload[4]. Profound bradycardia and asystole after administration of Propofol have been described in healthy adult patients despite prophylactic anticholinergic (Egan and Brock, 1991, James et al, 1989). Propofol decreases blood pressure, cardiac output as systemic vascular resistance due to inhibition of sympathetic vasoconstriction and impairment of baroreceptor reflex regulatory system[6]. Propofol causes dose dependent respiratory depression (first reduction in tidal volume associated with tachypnoea followed by apnoea), Propofol infusion inhibits hypoxic ventilatory drive and depresses the normal response to hypercarbia (Blouin et al, 1993)[6]. Apnoea occurs in 25-30% of patients depending on dose Propofol decreases cerebral metabolic rate for oxygen ($CMRO_2$), cerebral blood flow, cerebral perfusion pressure and intracranial pressure (ICP) (Pinaud et al , 1990). There is decrease in Cerebral metabolic oxygen consumption, Cerebral blood flow and ICP along with decrease in cerebral perfusion pressure[7]. Cerebrovascular auto regulations response to change in systemic blood pressure and reactivity of cerebral blood flow to change in carbon dioxide partial pressure are not affected by Propofol[8,9]. In higher doses it produces burst suppression in EEG (Smith et al, 1996) Conclusively during general anesthesia, it's desirable to have a stable intraoperative hemodynamic status and minimal respiratory depression. Hence in this study, it has been attempted to evaluate and compare the beneficial and also side effect of propofol and etomidate. The patients were randomly allocated into two groups (group E and group P) with equal n = 40. A prospective double blind, parallel randomized controlled clinical study was done to compare the hemodynamic parameters, pain on injection, any adverse effects and to define better inducing agent in terms of time of onset and efficacy of induction at tertiary centre between July 2015 to July 2016. The study population comprised of 80 patient of ASA grade I, II, and III aged between 20-60 year written informed consent was taken from each patient. The patients were randomly assigned into two groups comprising of 40 patients in each group Group E (Etomidated Group, n=40) – received 0.3 mg/kg etomidated for induction Group P (Propofol group, n=40) – received 2 mg/kg propofol for induction.

1. Inclusion criteria
- Age group between 20-60 years
- Both sex
- ASA grade I, II and III
- Patient who had not eaten solid food within 8 hr before or liquid within 2 hr before

2. Exclusion criteria
- Patient refusal
- Patient allergic to any drug
- History of seizure disorder
- Presence of known primary and secondary adrenal insufficiency or on steroid medications
- Presence of hypotension

The ethical clearance for the study was obtained from institutional ethics committee. Patient undergoing elective surgery under general anesthesia were screened for the eligibility,

Patient fulfilling selection criteria were selected for this study and briefed about the nature of study and explained about anesthesia procedure. A written informed consent was obtained from the patient, the dose of etomidate and propofol were intended to be equipotent All prefilling, coding and decoding was done with the help of department of clinical pharmacy. The investigators involved in the study did not know about the content of the syringe. Patient were explained about the study, but did not knew which drug was used I V line with 18G cannula in one hand were secured HR, SBP, DBP, MAP & SpO_2 were monitored before, during and after the surgery. Patient were premedicated with injection glycopyrrolate 0.2mg i v, ondansetron 4mg i v and injection fentanyl 2 g/kg followed by pre-oxygenation for three minutes. After shifting to operating room monitors, ECG, NIBP, pulse oximeter was attached, Patients were induced with either propofol 2 mg/kg or etomidate 0.3 mg/kg, muscle relaxation was facilitated with injection Atracurium 0.5 mg/kg Patient were intubated using and appropriated size endotracheal tube and maintained on O_2:N_2O (30:70) and Isoflurane (1%) was started. Throughout the procedure any 20% change in MAP above or below basal MAP, isoflurane concentration was increased or decreased/stop to maintain basal MAP HR less than 50 bpm was treated with atropine 0 6 mg iv Parameters like HR, SBP, DBP, MAP and SpO_2 was measured before induction; After induction; At intubation ; At 1 min – post intubation ; At 3 min – post intubation; At 5 min – post intubation and At 10 min – post intubation After surgery patients were received with injection glycopyrrolate 0.01 mg/kg and injection neostigmine 0.05 mg/kg patient was extubated and time to recovery was measured Recovery being defined as the time to vocalize after extubation Data was expressed a mean and standard deviation (SD) The homogenecity in two group of mean and SD was analysed using SSPS version 17.0, one way analysis of variance for each parameter Scheffe's test is used to compare pair wise data A p value of less than or equal to 0.05 was considered as significant.

Results

The mean age in group P (propofol) was 29.16 and in group E (etomidate) group was 27.86. The mean weight in group P (propofol) was 56.02±11 03 kg and in group E (etomidate) was 57±11 16 kg. Table 1 shows the mean changes in heart rate in two study groups and comparison among them to pre-induction (baseline value) Post-induction and after intubation The change in Mean heart rate, where it was seen that among group E (etomidate) patients the basal mean HR i e pre induction was 82 1±5 24, post induction was 80.76±5.27 followed by 83.56 ±5 06, 82.2±4.91, 82.73 ±4.98, 81.63±4.8, 82.33±5.2 at 0,1,3,5,10 minutes after intubation respectively Among group P (propofol) the basal mean HR in beats per minute was 82±5.77, post induction was 75.93±5.36 followed by 85 .8±5.65, 84.56±5.29, 81.26±5.05, 76.83±4.78, 82.56±4.93 at 0,1,3,5,10 minutes after intubation respectively. Statically evaluation with base line value, in etomidate group shows heart rate did not significantly change compared to pre-induction ($p>0.05$), But in propofol group, post induction heart rate significantly decreased ($p<0.001$) and after intubation, significantly increased ($p<0.001$) compared to the pre-induction Mean heart rate returned to normal at 10 minutes in both the group.

Table 1: Change in Mean Heart Rate

Observation time		Group E	p value	Group P	p value
Pre induction		82.1±5.24		82.2±5.77	
Post Induction		80.76±5.27	p>0.05	75.93±5.36	P<0.001
After Intubation	0 min	83.56±5.06	p>0.05	85.8±5.65	P<0.001
	1 min	82.2±4.91	p>0.05	84.56±5.29	P<0.001
	3 min	82.73±4.98	p>0.05	81.26±5.05	P<0.001
	5 min	81.63±4.8	p>0.05	76.83±4.78	P<0.001
	10 min	82.33±5.2	p>0.05	82.56±4.93	P<0.001

In table 2 changes in systolic blood pressure shown in etomidate group pre induction SBP was 123.7±5.5, In post induction SBP 121.93±5.43. Again SBP after intubation 0 min, 1 min, 3 min, 5 min,10 min were 124.03±5.65, 123.93 ±5.66, 123.43±5.3, 122.56±5.13, 123.13±5.7 respectively. Similarly, in Group P pre induction SBP was 124.33±3.71, Post induction SBP, 94.1±6.76 followed by after intubation 0, 1, 3, 5, 10 mins were 121±4.74, 119.6±4.08, .107±7.18, 96.6±5.7, 122.80±3.82 respectively. Statically analysis of these two group shows in etomidate group, there was no significant change in SBP in post induction and after intubation compared to pre induction value, as p value is >0.05, but in propofol group SBP decreased significantly in post induction ($p<0.0001$), and after intubation ($p<0.0001$) SBP again increased, In propofol and etomidate group, at 10 minutes SBP returned to pre induction value ($p>0.05$).

Table 2: Changes in Mean Systolic Blood pressure

Observation time		Group E	p value	Group P	p value
Pre induction		123±5.50		124.33±3.71	
Post Induction		121.93±5.43	p>0.05	94.1±6.76	p<0.0001
After Intubation	0 min	124.03±5.65	p>0.05	121±4.74	p<0.001
	1 min	123.93±5.66	p>0.05	119±4.08	p<0.0001
	3 min	123.43±5.3	p>0.05	107±7.18	p<0.0001
	5 min	122.56±5.13	p>0.05	96.6±5.7	p<0.0001
	10 min	123.13±5.7	p>0.05	122.80±3.82	p>0.05

Table 3 Shows mean change in DBP in three study groups and comparison of them with pre-induction value (Baseline value) where it was seen that among etomidate group, pre induction DBP was 81.5±4.82. In post induction, DBP was 79.16±4.68 after intubation 0 min, 1 min, 3 min, 5 min, 10 min DBP were 81.36±4.77, 81.32±4.78, 81.3±4.7, 79.9±4.9 and 81.93±5.0 respectively, Among group P (propofol) the basal MDBP was 80.3±3.8, in post induction, DBP was 79.8±4.26, and it was 79.8±4.26 at 0 minutes, 79.23±4.19 at 1 min, 75.26±3.48 at 3 min, 64.83±5.06 at 5 min, 78.80±3.90 at 10 minutes. Statiscal evaluation in etomidate group; Post-induction and after intubation, DBP did not change significantly ($p>0.05$). But in propofol DBP decreased after induction ($p<0.05$) and again increased at 0 and 1 min ($p>0.05$) after intubation it significantly decreased again at 3 and 5 mint ($p<0.0001$). In etomidate and propofol group DBP returned to pre-induction (base line value) at 10 minutes.

Table 3: Change in Mean Diastolic Blood Pressure

Observation time		Group E	p value	Group P	p value
Pre induction		81.5±4.82		80.3±3.8	
Post Induction		79.16±4.68	p>0.05	61.3±2.89	p<0.05
After Intubation	0 min	81.36±4.77	p>0.05	79.8±4.26	p>0.05
	1 min	81.32±4 78	p>0.05	79.23±4.19	p>0.05
	3 min	81.3±4 7	p>0.05	75.26±3.68	p<0.0001
	5 min	79.9±4 9	p>0.05	64.83±5.06	p<0.0001
	10 min	81.93±5	p>0.05	78.80±3.90	p>0.05

Table 4 shows - MAP in two study groups, Pre-Induction was the baseline value. The change in Mean arterial pressure,where it was seen that among Group E (etomidate) pre induction MAP was 95.99±4.88. In post induction MAP was 93.4±4.7 and after intubation 0 min, 1 min, 3 min, 5 min and 10 min MAP were 95.85±4.82, 95.29±4.65, 95.29±4.64, 95.29±4.64 and 95.99±4.8 respectively. Among group P, pre induction MAP was 94.76±3.15, in post induction MAP was 72.19±3.31, followed by 93.83±3.26 at 0 min, 94.48±3.5 at 1 min, 86.39±3.48 at 3 min, 73. 07±3.40 at 5 mins, 93.40±3.24 at 10 mins after intubation Statiscal evaluation in etomidate group, Post -induction and after intubation, MAP did not change significantly (p>0.05). But in propofol group, post-induction, MAP decreased significantly (p<0.05) and after intubation again MAP increased, at 0 and 1 min (p>0.05) but it decreased significantly at 3 and 5 mins (p<0.0001). In propofol and etomidate group, MAP returned to baseline at 10 minutes. The incidence of myoclonus among study population, where it was seen that among Group E patients 4 showed grade I and among Group P, 8 showed grade I and 7 showed grade II P value=0.04, which shows significance It was seen that among Group E patients, no patient had pain on injection and among Group P patients 8 had grade I pain and 18 had grade II pain P value <0.0001, which shows significance Injection on pain is graded as:

Grade 0 – No pain
Grade 1- Verbal complain of pain
Grade 2 – Withdrawal of arm
Grade 3 – Both verbal complain and withdrawal of arm

Table 4: Change in Mean arterial blood pressure

Observation time		Group E	p value	Group P	p value
Pre induction		95.99±4.88		94.76±3.15	
Post Induction		93.4±4.7	p>0.05	72.19±3.31	p<0.0001
After Intubation	0 min	95.85±4.82	p>0.05	93.83±3.26	p>0.05
	1 min	95.29±4.65	p>0.05	94.48±3.5	p>0.05
	3 min	95.29±4.64	p>0.05	86.39±3.48	p<0.0001
	5 min	95.29±4.64	p>0.05	73.0±3.40	p<0.0001
	10 min	95.99±4.8	p>0.05	93.40±3.24	p>0.05

The incidence of nausea, where Group E patients 12 had nausea as compared to 7 in Group P, P value= 0.08, which shows no significance The incidence of vomiting, where Group E showed more incidence of vomiting than Group P, P value= 0.34, which shows no significance.

Discussion

Anesthesia-induced hemodynamic fluctuations are a matter of concern for anesthesiologists. Laryngoscopy and endotracheal intubation can cause sympathetic stimulation often manifested as an increase in systolic and diastolic blood pressures and heart rate. Research evidence indicates that these hemodynamics alterations are independently associated with postoperative complications in patients undergoing surgery[10]. In etomidate group there was no significant change in heart rate in post induction and after intubation as compared with pre induction. Also there was no significant change after intubation as compared with post induction. As p value is >0.05 which is statically insignificant but in case of propofol group, pre induction heart rate was 82.2±5.77. In post induction heart rate decreased to 75.93±5.36, which was highly significant (p<0.0001) as compared with pre induction value again heart rate increased significantly after intubation compared with pre-induction and post-induction and returned to pre-induction value in 10 mins and post induction heart rate value in 5 mins. In our study we found heart rate is more stable with etomidate induction in comparision to induction with propofol. Results were comparable to the result obtained by Moller et al[11] which is used propofol and etomidate in G A induction accompanied by BIS monitoring, the MAP, CI, SVRI values of 48 patients were compared. The hemodynamic data were found to be higher in the etomidate group up to 7 minutes after intubation a significantly high level of hypotension incidence was found in the propofol group and a significant high level of hypertension incidence in the etomidate group. Compared with etomidate, the use of propofol was determined to have caused less hypertension and tachycardia after intubation. In etomidate group pre induction SBP was 123.7±5.54. In post induction SBP 121.93±5.43 again SBP after intubation 0 min, 1 min, 3 min, 5 min, 10 min were 124.03±5.65, 123.93±5.66, 123.43±5.3, 122.56±5.13, 123.13±5.7 respectively. There was no significant change in SBP in post induction and after intubation compared to pre induction value and also no significant change after intubation compared to pre induction and post induction value, as p value is >0.05. Similarly, in group P pre induction SBP, 124.33±3.71, Post induction SBP, 94.1±6.76, after intubation, SBP again increased at 10 min after intubation blood pressure returned to pre induction value. The SBP nearly unchanged with etomidate, Pre-induction was taken as baseline value In etomidate group, Post-induction and after intubation, SBP did not change significantly as compared to preinduction (P>0.05) But in propofol group, SBP decreased significantly in post induction (p<0.0001) and after intubation SBP again increased. In propofol and etomidate group, at 10 minutes SBP returned to pre-induction value (p>0.05). Our study shows mean change in DBP in two study groups and comparison of them with pre-induction value (Baseline value). In etomidate group; Post-induction and after intubation, DBP did not change significantly (p>0.05) But in propofol DBP decreased after induction (p<0.05) and again increased after intubation. In etomidate and propofol group DBP returned to pre-induction (base line value) at 10 minute , This is supported by A Gauss (1991) noticed the change in SBP by 1 mm Hg, DBP by 1 mmHg with Etomidate lipuro and SBP decreased by 13 mmHg, DBP by 4 mmHg in

Propofol group[12]. In our study mean MAP pre-Induction was the baseline value In etomidate group, Post -induction and after intubation, MAP did not change significantly (P>0.05), But in propofol group, post-induction, MAP decreased significantly (P<0.0001) and after intubation again MAP increased (p>0.05) In propofol and etomidate group, MAP returned to baseline at 10 minute. We observed that propofol caused significant hypotension and tachycardia at induction in comparison to etomidate, Hypotension occurs with propofol is mainly due to reduction of sympathetic activity causing vasodilation or its direct effect on vascular smooth muscles. Sudden hypotension and tachycardia has deleterious effects on maintaining the circulation to vital organs in on another side hemodynamic stability observed with etomidate may be due to its unique lack of effect on the sympathetic nervous system and on baroreceptor functions Mayer et al and Wu et al also concluded that etomidate preserve hemodynamic stability during anesthesia[13,14]. Similar results obtained by Singh R et al, they compared the hemodynamic effects of etomidate and propofol in patients with coronary artery disease with LV dysfunction. After induction there was a significant decrease in the variable to compared to the base line HR (-7 to -15 %, p=0.001), MAP (-27 to -32%, p= 0. 001), CI (-36 to -38%, p=0.001) and SVI (-27 to -34%, p=0.001) in the etomidate group, there was a significant increase from the base line in both HR (p=0.001) and MAP (p=0.001) at one minute after intubation. All the four agents were acceptable for induction in patient's coronary artery disease with LV dysfunction, despite a 30-40% decrease in the cardiac indices these findings of our study corroborates with the study reports of Gooding JM et al[9] Vanacker et al[17], Kulka et al[15], Ebert et al[6], Zaugg et al (2002) and Paris et al[16] Similar results were obtained in study of Mehrdad at el[18] conducted a study including patients of 18 to 45 years of age that were admitted for elective orthopedic surgeries. Patients were divided in two groups, their cardiovascular response including SBP, DBP, MAP, HR and saturation were measure before the laryngocopy during anaesthesia induction with etomidate (0.3 mg/kg) in groups A propofol (2-2.5 mg/kg) in group B and at 1, 3, 5, 10 minutes after the induction. They concluded that patients receiving etomidate have more stable hemodynamic condition, if there would be no contraindication, it could be preffered over propofol for general anaesthesia. Another study which supports our observation done by Saricaoglu et al[19], after studying the hemodynamic effects of an induction dose of propofol and etomidate found that propofol was associated with significant decrease SBP and MBP. They attributed this hypotension to the negative inotropic effect of propofol. A similar study was done by Petrun M et al in which they noticed that there were no significant differences between the two groups regarding the hemodynamics before intubation, After intubation, MAP (P¼0.019) was significantly higher in the E group CI was significantly higher in the Group E after intubation Pandey AK et al compared the effects of propofol and etomidate induction on hemodynamic parameters and serum cortisol levels in patients undergoing coronary artery bypass graft. It was found that propofol group had a significant reduction in SAP, DAP, and SVRI, On the other hand, there was no change in cardiac output following induction of anesthesia (P<0.05) with etomidate. Pain during injection of anesthetic agent is a bad experience for patient while it quite embarrassing situation for an anesthesiologist Etomidate shown a favorable outcome and it was very well supported by Saricaoglu et al[19] and Wu et al[13]. Present study shows the incidence of myoclonus among study population, where it was seen that among Group E patients, 4 showed grade I myoclonus And among Group P, 8 showed grade I and 7 showed grade II A study by Mirakhur RK[20] showed that Myoclonus occur but less frequently with propofol than after etomidate lipuro, Present study shows the incidence of nausea, where Group E patients 12 had nausea as compared to 7 in Group P In a double blind randomized study, M Stpierre[21] studied the incidence and severity of post-operative nausea and vomiting was investigated with etomidate-lipuro and propofol. They concluded that etomidate lipuro does not show increase incidence of nausea than propofol during early post-operative period Group E showed more incidence of vomiting than Group P. In a double blind randomized study, M Stpierre[21] studied the incidence and severity of post-operative nausea and vomiting was investigated with etomidate-lipuro and propofol. They concluded that etomidate lipuro does not show increase incidence of vomiting than propofol during early post-operative period These findings are consistent with a finding obtaind by A study by J G Reves et al[22] showed that the cardiovascular effects of propofol have been evaluated after its use for induction and for maintenance of anesthesia The most prominent effect of propofol is a decrease in arterial blood pressure during induction of anesthesia.

Conclusion

Based on the present clinical comparative study, the following conclusion can be made etomidate is better inducing agent than propofol with regard to cardiovascular stability. There is no significant ECG and SPO_2 changes seen between two groups Side effects like pain on injection, myoclonus and nausea was more pronounced with propofol. Only vomiting is more frequent with etomidate, Although etomidate causes adrenocortical suppression, we did not examine the cortisol label of the group E. Also further studies need to be conducted with a larger sample size to corroborate the finding of this study, which may enlighten further the usefulness of etomidate as inducing agent specially in hameodynamically unstable patient.

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