Original Research Article

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To Evaluate Hemodynamic Effect of Propofol and Etomidate as Induction Agents in Elective Surgeries

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Abstract

Introduction: One of the major concerns of the anaesthetist during induction of anesthesia is maintenance of haemodynamic stability. Various induction agents have been used during induction like thiopentone, propofol, ketamine and etomidate.

Aim: The aim of this study was to evaluate the hemodynamic effect ofPropofoland Etomidate as induction agents in elective surgeries undergeneral anesthesia.

Material and Methodology: Sixty (60) patients of age group (18-60 years) of ASA grade I and II were randomly divided into two groups of thirty patients each, as follows:

Group I: Propofol 2 mg/kg was given intravenously as induction agent.

Group II: Etomidate 0.3 mg/kg was given intravenously as induction agent. The various haemodynamic parameters were recorded and compared.

Result: A significant difference inhaemodyanamic parameters seen between both the groups. In group I (Propofol) HR, SBP, DBP, MBP significantly decrease at the time of induction and upto 60 minutes ($p<0.05$) whereas in group II (Etomidate) lesserfall in hemodynamics parameters seen as compared to group I ($p>0.05$).

Conclusion: Induction with etomidate is associated with lesser fall in haemodynamic parameters as compared to propofol .

Keywords: Induction; Haemodynamic; Parameters; Propofol; Etomidate.

Introduction

Hemodynamic stability at the time of induction of anaesthesia and during surgery has been a major concern for the anaesthetist. It depends not only on the basal tone of the autonomic nervous system but is also importantly influenced by baroreceptor reflex regulation of autonomic outflow influencing cardiac function and peripheral vascular resistance[1]. Pressor response to laryngoscopy and intubation is a documented fact in patients, under a variety of anaesthetic techniques [2-3]. These changes are due to stimulation of receptors at the base of the tongue which leads to hypertension, tachycardia and other arrhythmias in proportion to magnitude of the stimulus. There is an increase in the concentration of catecholamines like adrenaline and nor- adrenaline in response to the stimulus of laryngoscopy [5] and subsequent intubation stimulates the receptors in larynx and trachea withenhancement of hemodynamic and epinephrine response. The mean increase in arterial pressure due to laryngoscopy and intubation may be upto 20-25 mmHg and the peak response occurs approximately 30-35sec after laryngoscopy. These cardiovascular changes are

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transient, variable and unpredictable and usually well tolerated by healthy individuals, but may be fatal in patients with hypertension, coronary artery disease or raisedintracranial tensions. Laryngoscopy produced the major contribution to the stresss response whereas tracheal intubation on its own contributed only a little. Various drugs and techniques have been utilized to blunt this response with variable degree of success. Thiopentone, propofol, etomidate, ketamine andetofol (admixture of propofol and etomidate) are used as induction agents induction to lower the stress response to laryngoscopy and intubation and to maintain better hemodynamic stability.

Induction of anaesthesia with propofol is smooth and decreases airway reflexes but is associated with significant blood pressure reduction and hemodynamic instability. Propfolis commonly used in outpatient anaesthesia for its rapid and smooth onset of action, short recovery period and minimal per operative side effects [6] but it decreases the cardiac parasympathetic tone in a dose dependent manner [1]. Etomidate as an intravenous induction agent has minimal cardiovascular side effects making it especially suitable for cardiac compromised patients and for those in whom hypotension must be avoided during induction and surgery.

Although etomidate provides stable hemodynamics and minimal respiratory depression, it is associated with several side effects when used for induction of anaesthesia or continuous infusion , including nausea and vomiting [7].

In this study, we aim to evaluate the hemodynamic effects of propofol and etomidate during induction, laryngoscoy, intubation and during surgery under general anaesthesia in elective surgeries.

Aims and Objectives

To evaluate and compare the efficacy of propofol and etomidate as induction agents in maintaining haemodynamic stability in elective surgery under general anesthesia.

Material and Methods

This randomized double blind clinical study was conducted in the department of anaesthesiologyat a tertiary care centre in India, after approval from Institutional Ethical Committee on 60 ASA grade I and II patients aged 18 to 60 years, of either sex, undergoing elective surgery lasting for approximately 2 hrs undergeneral anesthesia.

Patient having cardiac disease, hypertension, respiratory disease, cerebello vascular disease, Mallampati grade III-IV, epilepsy and pregnant patients were excluded from the study. All patients were kept nil per oral for 8 hours. Written informed consent was taken from all patients. On arrival in the operation theatre standard anaesthesia monitors including pulse oxymetry, electrocardiogram, non invasive blood pressure (NIBP) were attached and hemodynamic parameters were recorded. An 18 G intravenous cannula was secured and Ringer Lactate infusion was started. Midazolam 0.025 mg/kg i/v and Nalbuphine 0.1mg/kgi/v were given 2 minutes before induction. The patients were randomly divided into two groups. Randomization was done by computer generated random numbertables. Group I received Propofol 2mg/kg i/v and group II Etomidate 0.3mg/kg i/v for induction. All study drugs were prepared by an anesthesiologist who was blinded to the details of the study. Volume of medication and speed of injection were equal in both the groups. Injection Rocuronium 1.2mg/mg i/v was given as muscle relaxant. Laryngoscopy and endotracheal intubation was done by experienced anesthesiologist and the duration of laryngoscopy was keptto less than 10 seconds. Proper placement of ETT was confirmed by capnography and bilateral auscultation of chest. Anesthesia was maintained by Isoflurane 1-1.5% and equal mixture of Oxygen-Nitrous Oxide (4 L/min) along with intermittent bolus of injection Rocuronium as required throughout the surgery. Heart rate (H.R), systolic blood pressure(SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MBP)and oxygen saturation were continuously monitored and recorded before induction, at induction and after induction at 1, 2, 5,10, 20, 30 and 60 minutes by an anaesthesiologist who wasblinded to the study .

Statistical Analysis

Data was analysed by computer software package Statistical Package for Social Sciences (SPSS) version 20.0 for windows. Categorial data like gender was presented as number. Age, weight, heart rate and blood pressure were presented as Mean±standard deviation (S.D). Inter group comparison of blood pressure and heart rate was done using ANOVA. p value of <0.05 was considered significant.

Results

The *patient's characteristics* i.e. age, sex and weight were statistically similar in both the groups. [Table 1]. The mean baseline heart rate, SBP, DBP,

MBP in both the groups were comparable to each other and statistically non significant. The heart rate in groupI at 1 minute decreased to 69.2±10.5 and in group II to74.8±8.7 and the difference in heart rate betweenboth the groups was statistically significant ($p<0.05$). No significant difference in heart rate was observed at 0 minute, and from 2 minutes till 60 minutes. (p-value>0.05) of induction. Statistically significant fall in *SBP* was observedin group I at 0 minute (at time of induction), and from1 minute, 2minute, 5minute, 10minute, 30minute, 60 minutes ($p=0.000$) of induction [Table 3]. Statistically significant fall in DBP was observed in group I at 0 minute (at time of induction) and at1minute, 2 minute, 5 minute, 10 minute, 30 minute and 60 minutes (p-value=0.000) after induction [Table 4]. Statistically

Table 1: Comparison of Demographic variables of patients in both the groups

Variables	Group I(n=30)	Group II(n=30)	p value	Statistical significance
Age (years)	37.62±9.06	37.60±9.64	0.265	NS
Gender (male/female)	20/10	18/12	0.279	NS
Weight (kg)	58.1±1.8	57.5±1.6	0.232	NS

Table 2: Comparison of Heart rate between both the groups

Time	Group I (N=30)	Group II (N=30)	P value	Statistical Significance
Baseline	81.5±12.3	84.5±9.8	**0.310**	**NS**
O minute	73.0±11.4	76.7±8.3	0.144	NS
1 minute	69.2±10.5	74.8±8.7	0.024	S
2 minute	79.0±8.5	80.8±10.2	0.452	NS
5 minute	76.5±9.0	79..6±9.6	0.167	NS
10 minute	75.2±10.2	78.4±9.2	0.175	NS
30 minute	74.5±10.3	78.5±10.5	0.113	NS
60 minute	74.6±9.7	78.5±10.0	0.102	NS

Table 3: Comparison of Systolic blood pressure (SBP) between both the groups

Time	Group I (N=30)	Group II (N=30)	P value	Statistical Significance
Baseline	137±4.3	131.1±8.7	0.167	NS
O minute	102.2±8.1	118.6±14.4	0.000	S
1 minute	94.2±9.4	112.9±17.1	0.000	S
2 minute	94.8±7.4	127.3±10.9	0.000	S
5 minute	95.7±6.4	125.4±9.2	0.000	S
10 minute	96.7±6.5	124.3±7.7	0.000	S
30 minute	98.5±5.3	124.4.±9.6	0.000	S
60 minute	99.6±5.5	124.9±7.8	0.000	S

Table 4: Comparison of Diastolic blood pressure between both the groups

Time	Group I (N=30)	Group II (N=30)	P value	Statistical Significance
Baseline	87.4±4.0	86.4±4.5	0.317	NS
O minute	58.1±6.2	74.7±10.6	0.000	S
1 minute	56.2±5.7	69.7±12.0	0.000	S
2 minute	57.7±5.5	80.4±7.6	0.000	S
5 minute	57.0±4.6	80.2±6.2	0.000	S
10 minute	56.5±4.2	79.6±5.9	0.000	S
30 minute	58.5±5.1	79.9±5.9	0.000	S
60 minute	58.0±4.4	80.2.±6.7	0.000	S

Table 5: Comparison of Mean blood pressure between both the groups

Time	Group I (N=30)	Group II (N=30)	p value	Statistical Significance
Baseline	104.3±3.9	102.1±4.9	0.057	NS
O minute	72.4±6.4	89.0±11.5	0.000	S
1 minute	68.4±6.3	84.2±13.4	0.000	S
2 minute	69.6±5.1	96.0±8.2	0.000	S
5 minute	69.5±4.7	95.2±6.8	0.000	S
10 minute	69.6±4.1	94.6±5.6	0.000	S
30 minute	70.9±4.7	95.1±6.1	0.000	S
60 minute	71.6±3.8	95.3±5.8	0.000	S

significant fall in MBP was observed in group I at 0minute (at time of induction), and after 1minute, 2minute, 5minute, 10minute, 30minute and 60 minutes (p-value=0.000) of induction [Table 5].

Discussion

General anesthetic induction agents may decrease arterial blood pressure via cardio vascular depression and attenuation of autonomic nervous system activity. On the other hand laryngoscopy and endotracheal intubation elicit vasopressor responses such as hypertension and tachycardia. Various attempts have been made to attenuate hemodynamic instability duringinduction, larynoscopy, and intubation. In many studies induction agents, eitheralone or in combination have been used to achieve minimumcardiovascular effects. Propofol is widely used as an intravenous induction agent but induction with propofol alone causes remarkable reduction in blood pressure. Various studies show that etomidate provides better hemodynamic profile during induction but for its adverse effects like myoclonus and adrenal suppression.

Patients in both the groups were comparable with respect to age, sex and mean weightconsistent with findings of K Meena et. al. who also hadcomparable age group and comparable gender distribution [8].

In our study significant difference in heart rate was seen at 1minute in group I vs II after induction (p = 0.024). No significant difference in heart rate was observed at 0 min, after 1min till 60 mintues. (p>0.05) Masoudifar M et al concluded there were no significant difference among groups I (Propofol) and II (Etomidate) in terms of HR (P = 0.47) [9]. Whereas Singh R et. al. found a significant increase from baseline in heart rate (P = 0.001) at 1 minute after intubation in their study. This statistically significant difference can be due to ASA grade III patients with coronary artery diseaseand left ventricular dysfunction [10].

In our study statisticallysignificant fall in SBP was observed in group I at 0min which persisted till 60 minutes. A significant fall in SBP was observed in group I vs II at 2min, 5min,10min, 30min and 60 minutes (p=0.000).

Geeta Karki et al concluded that Etomidate offers superior hemodynamic stability during induction compared to thiopentone and propofol, similar to our study [11].

Stastically significant fall in DBP was found in group I vs group II at 0 minute, 1minute, 2minute, 5minute, 10minute, 30minute and 60 minutes (p-value 0.000) of induction. In 2015 Ozgur Yagan et. al. measured the haemodynamic responses using etomidate, propofol and combination of etomidate-propofol. In all 3 groups, a significant decrease in MAP values were seen at T2 and T3 compared to the baseline values, and this decrease was greater in group P compared to that in group E and PE ($P < 0.001$, $P<0.01$)[12] Findings of our study are comparable with, Ozgur Yagan et. al.

Statistically significant difference was found in MBP in both the groups at 0 min (at the time of induction) and after 1 minute of induction. Significant change (fall in MBP) was observed in group I vs II at 2minute, 5minute,10minute, 30minute and 60 minutes (p-value 0.000). Bendel SI et. al. found that MBP decreased to a greater extent in patients receiving propofolthan in those receiving etomidate ($P = 0.006$) [13]. Erdil F et. al. concluded that MBP were lower at T3 and T4 in the propofol group than in the etomidate group ($P < 0.05$) [14]. Findings of our study are consistent with the study of Bendel et. al. and Erdil et. al.

Conclusion

Induction with etomidate provides stable hemodynamic parametersas compared to propofol which causes noteworthy hypotension when used for' induction of anesthesia.

References

1. Ebert TJ, Muzi M, Berens R, Goff D, Kampine JP. Sympathetic responses to induction of anaesthesia in humans with propofol or etomidate. Anaesthesiology 1992;76:725-33.
2. King BD, Harris LC, Griefenstein FE et. al. Reflex circulatory response to direct layrngoscopy andintubation performed during anesthesia Anaesthesiology 1951;12:556-66.
3. Wycoff CC. Endotracheal intubation effects on blood pressure and pulserate. Anaesthesiology 1960;21:153-8.
4. Burnstien CL, Pinto Lo FJ and Newman W. Electro-cardiographic studiesduring tracheal intubation. I: effect during usual routine technics. Anaesthesiology 1950;11:224-37.
5. Russel W J, Morris RG, Frewin D B et .al. Changes in plasma catecholamine concentrations during endotracheal intubation. Br J Anaesth 1981;53:837-9.
6. Ghabash M, Matta M & Kehhaleh J,Depression of excitatory of propofol induction by fentanyl,Middle East J Anaesthesiol, 1996;13(4):419-25.

7. St Pierre M, Dunkel M, Rutherford A, Hering W. Does etomidate increase postoperative nausea? A double-blind controlled comparison ofetomidatein lipid emulsion with propofol for balanced anaesthesia.Eur J Anaesthesiol 2000;17:634-641.
8. Meena K, Meena R, Nayak SS, Prakash S, Kumar A . A Comparative Study of Effect of Propofol, Etomidate and Propofol Plus Etomidate Induction on Hemodynamic Response to Endotracheal Intubation: A RCT. J AnesthClin Res 2016;(7):622. doi: 10.4172/2155-6148.1000622.
9. Masoudifar M1, BeheshtianE.Comparison of cardiovascular response to laryngoscopy and tracheal intubation after induction of anesthesia by Propofol and Etomidate.J Res Med Sci.2013;18(10):870-4.
10. Singh R, Choudhury M, Kapoor PM, Kiran U. A randomized trial of anesthetic induction agents inpatients with coronary artery disease and left ventricular dysfunction. Ann Card Anaesth. 2010; 13:217-23.
11. Geeta Karki, Vishwadeep Singh, Abhishek Barnwal, Lalit Singh. A Comparative Evaluation of Hemodynamic Characteristics of the Three Induction Agents – Etomidate, Thiopentone and Propofol. Journal of Evolution of Medical and Dental Sciences 2014 Aug 11;3(34):9133-41.
12. Yagan O, Tas N, Kucuk A, Hanci V, Yurtlu BS. Haemodynamic Responses to Tracheal Intubation Using Propofol, Etomidate and Etomidate-Propofol Combination in Anaesthesia Induction. J Cardiovasc Thorac Res 2015(7):134-40.
13. Bendel SI, Ruokonen E, Pölönen P, Uusaro A. Propofol causes more hypotension than etomidate in patients with severe aortic stenosis: Adouble-blind, randomized study comparing propofol andetomidate. ActaAnaesthesiol Scand 2007;51:284-9.
14. Erdil FL, Demirbilek S, Begec Z, Ozturk E, Ersoy MO. Effects of propofol or etomidate on QT interval during electroconvulsive therapy. J ECT. 2009Sep;25(3): 174-7.