

## PREVENTION OF HYPOTENSION DURING PROPOFOL INDUCTION: A COMPARISON OF PRELOADING WITH RINGER LACTATE AND INTRAVENOUS EPHEDRINE

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**ABSTRACT:** The induction of general anaesthesia with propofol, however, has been associated with a decrease in systolic arterial blood pressure. Various measures to prevent hypotension include preloading with fluids (colloids and crystalloids) and use of vasopressors including ephedrine, dopamine, dobutamine, and metaraminol. The aim of the present study was to compare the three different regimes for the prevention of hypotension during induction of anaesthesia. **MATERIALS AND METHODS:** Ninety patients, classified as ASA physical status I or II, male/female, aged 20-50 years, body weight 45-85 kg, scheduled for various elective surgeries under general anaesthesia gave written informed consent to participate in this study. Patients were randomly allocated into one of three groups of 30 patients each to receive either normal saline Group C, ephedrine 70µg/kg, or Group E 10ml/kg Ringer Lactate Group RL prior to induction of anaesthesia with propofol (2mg/kg). Parameters analyzed were: heart rate and systemic arterial pressure noninvasively before induction, after propofol administration, immediate post intubation and then at 3min, 5min and at 10 post intubation. **RESULTS:** In all the groups there was an increase in the systemic arterial pressure post intubation; ephedrine > RL > control group. On comparing the groups at varied intervals the decrease was statistically significant in control group. (P<0.00). On comparing ephedrine group with RL decrease was statistically significant in the ephedrine group at 10mins. (P<0.01). **CONCLUSION:** We concluded, preoperative administration of ephedrine failed to prevent propofol-induced hypotension, but preoperative volume loading with 10ml/kg of ringer lactate successfully antagonised it, hence provide more haemodynamic stability.

**KEYWORDS:** Propofol, hypotension, ephedrine, ringer lactate, blood pressure, heart rate.

**INTRODUCTION:** Propofol (2, 6 di-isopropyl phenol) is a rapidly acting i.v. anaesthetic agent that has gained wide acceptance for the induction and maintenance of general anaesthesia. Propofol is ideal for short and ambulatory surgical procedures requiring general anaesthesia, as recovery is rapid with fewer unwanted side effects, such as drowsiness on recovery, disorientation and nausea, when compared with other agents such as thiopentone. The induction of general anaesthesia with propofol, however, has been associated with a decrease in systolic arterial blood pressure, <sup>1, 2, 3</sup> especially in patients with advanced age (>50 years), prior hypotension (mean arterial pressure <70mmHg) and higher American Society of Anaesthesiologists' Physical Status (ASA-PS) class (> II). <sup>4, 5, 6, 7</sup> The hypotensive effect of propofol has been attributed to decrease in systemic vascular resistance<sup>1, 8</sup> and /or in cardiac output <sup>9</sup> caused by a combination of arterial and venous vasodilation<sup>1, 10</sup> impaired baroreceptor reflex mechanism<sup>11</sup> and depression of myocardial contractility<sup>12, 13</sup>. A propofol

mediated decrease in sympathetic activity may explain all the hemodynamic changes<sup>10, 11</sup> although direct vascular smooth muscle relaxation<sup>14</sup> and direct negative inotropic<sup>13, 15</sup> effect may contribute to a lesser extent. This decrease in blood pressure may not be clinically significant in young and healthy individuals, but significant hypotension during induction has been reported to correlate with longer post operative stay<sup>6</sup>. So far, various measures to prevent hypotension include preloading with fluids (colloids and crystalloids)<sup>16, 17</sup> and use of vasopressors including ephedrine, dopamine, dobutamine, and metaraminol has been tried.<sup>18, 19, 20</sup> Preloading with colloids (like Haemaccel®) prevents hypotension by increasing venous return and filling pressure of the right atrium and left ventricle to augment cardiac output but can have many disadvantages including long administration time, high cost, risk of haemodilution, fluid overload and anaphylactoid reactions.<sup>21</sup>

Similarly sympathomimetics (like ephedrine) prevent and correct hypotension by increasing peripheral vascular resistance and/or cardiac contractility with their advantages of low cost and ease of administration. But they also have disadvantages such as tachycardia and increased risk of arrhythmias with concomitant use of volatile anaesthetics<sup>22</sup>.

The aim of the present study was to compare the three different regimes which were propofol-placebo, propofol ephedrine and propofol crystalloid infusion in prevention of hypotension during induction of anaesthesia.

**METHOD AND MATERIALS:** Approval for the study was obtained from our institutional ethical committee. Ninety patients, classified as ASA physical status I or II, male/female, aged 20-50 years, body weight 45-85 kg, gave written informed consent to participate in this randomized, prospective, double-blinded trial.

All the patients who were scheduled for various elective surgeries under general anaesthesia such as pelvic laparoscopies: laparoscopic assisted vaginal hysterectomy, orthopaedic surgeries: upper limb humerus interlocking, laparoscopic cholecystectomy and appendicectomy were included in this study.

Subjects were excluded if they had a history of allergy to the study medication, pregnancy, morbid obesity, uncontrolled cardiovascular, respiratory, hepatic or renal disease, controlled or uncontrolled hypertension, diabetics, therapy with diuretics or vasoactive medications and patients posted for emergency surgeries under general anaesthesia.

Patients were advised pre-operative fasting for a period of 8 h and were premedicated with tab midazolam 0.1 mg/kg body weight, tab pantoprazole 40mg and tab ranitidine 150mg the night before and 2hr prior to the surgery with sips of water. On arrival in the induction room, a 16-gauge cannula was inserted into a peripheral vein at the dorsum of the hand by the first anesthetist who was not involved in charting the changes in heart rate and mean arterial pressure. Patients were assigned by pre randomized; sealed envelopes into three study groups and receive the following:

**Group P (control group) (n=30):** Propofol (2mg/kg) and 1ml of normal saline

**Group E (Ephedrine group) (n=30):** Propofol (2mg/kg) and 1ml of normal saline and ephedrine 70µg/kg.

**Group RL (Ringer Lactate) (n=30):** Propofol (2mg/kg) and 1ml of normal saline and 10ml/kg Ringer Lactate was given 10-15 minutes prior to induction of anaesthesia

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To achieve blinding, for the patients in the RL group, the fluid preload was given outside the operation theater by one investigator, and the empty fluid bags replaced with fresh fluid bags. Thus all patients arrived in the theater with full fluid bags attached to the i.v. fluid administration sets, and the anesthesiologist was not aware that whether the patient had received a fluid overload. The usual maintenance and replacement fluid (Ringer's Lactate solution) was started at the rate of 2ml/ kg in all the patients. After connecting standard monitoring: ECG, noninvasive arterial blood pressure (NIBP-Mean BP), pulse oximetry (SPO<sub>2</sub>) baseline measurements were recorded.

After preoxygenation for three minutes, injection fentanyl 1.5µg/kg was given as i.v bolus. Anaesthesia was induced 2mins later by using propofol 2mg/kg in 20-30 sec. If required, further increments of propofol 0.5mg/kg were given and repeated every 30 sec until loss of consciousness and eyelash reflex. Patient breathed oxygen-enriched air, and respiration was assisted by mask in all the patients in the mean time. Injection succinylcholine 1.5mg/kg intravenously was given thereafter. Laryngoscopy, orotracheal intubation and cuff inflation were carried out 30 sec later. After confirming the position and fixation of the endotracheal tube, positive- pressure ventilation was started with the administration of 0.25-0.5% isoflurane in a 2:1 N<sub>2</sub>O/O<sub>2</sub> gaseous mixture. Vecuronium, 0.06mg/kg, was given once neuromuscular function had recovered from succinylcholine. Parameters analyzed were pulse rate (PR), systolic blood pressure(SBP), diastolic blood pressure(DBP) and mean blood pressure (MBP), saturation (SpO<sub>2</sub>) and percentage change in pulse rate (PR), systolic blood pressure(SBP), diastolic blood pressure(DBP) and mean blood pressure (MBP) from the pre operative values. Measurements were made before the induction, after administration of propofol, post intubation and then at 3min, 5min and at ten min post intubation. No surgical stimulation was performed until the first ten minutes after induction was completed to ensure no untoward extraneous effect on patient's physiological variables during the study period. Analysis was performed on raw data using ANOVA test .Results were presented as mean ±SD.P<0.05 defined as statistical significant.

**RESULTS:** 40 women and 50 men were included in the study. The mean age and weight were 34.53±8.75 yr and 57.07±3.81kg for the control group; 35±7.11 yr and 56.30±4.76kg for the ephedrine group and 33.27±7.06yr and 57.77±7.07 kg for the ringer lactate group and were statistically insignificant. (P> 0.05)

In all the groups there was an increase in the mean heart rate, SBP, DBP, MBP, post intubation which was maximum in ephedrine group followed by RL and then control group. On comparing the control, ephedrine and RL group at 3mins, 5mins and 10 mins the decrease in the mean heart rate, SBP, DBP, MBP was statistically significant in control group (P =0.00). On comparing ephedrine group with RL, decrease was statistically significant in the ephedrine group (P =0.00).

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(Table 1, 2, 3 and 4)

**Table 1: Comparison of mean pulse rate among the groups**

<b>Pulse Rate (beats/min)</b>	<b>Group C (n = 30)</b>	<b>Group E (n = 30)</b>	<b>Group RL (n = 30)</b>	<b>P value C v/s E</b>	<b>P value C v/s RL</b>	<b>P value E v/s RL</b>
Pre- Induction	78.10±3.86	79.40±4.79	79.23±5.38	0.25	0.35	0.89
Post-Induction	89.77±4.36	101.00±5.98	86.77±6.43	0.00	0.00	0.00
Immediate-Intubation	83.93±3.49	112.17±7.97	88.43±6.34	0.00	0.00	0.00
3 min Post-Intubation	82.33±3.72	103.87±6.65	85.23±5.81	0.00	0.02	0.00
5 min Post -Intubation	79.87±3.74	95.73±5.46	84.13±5.71	0.00	0.06	0.00
10 min Post-Intubation	77.40±3.24	70.80±5.67	72.63±5.19	0.00	0.06	0.00

(Test applied: ANNOVA)

(P < 0.05 is significant)

**Table 2: Comparison of mean systolic blood pressure (SBP) among the groups**

<b>SBP mmHg</b>	<b>Group C (n = 30)</b>	<b>Group E (n = 30)</b>	<b>Group RL (n = 30)</b>	<b>P value C v/s E</b>	<b>P value C v/s RL</b>	<b>P value E v/s RL</b>
Pre- Induction	123.0±4.50	131.90±7.55	123.47±6.17	0.91	0.73	0.71
Post-Induction	112.13±4.44	136.37±9.67	113.47±6.65	0.00	0.00	0.00
Immediate-Intubation	140.40±4.10	148.27±8.22	137.13±8.71	0.00	0.02	0.00
3 min Post-Intubation	114.0±5.85	126.47±7.84	131.80±8.11	0.00	0.02	0.00
5min Post -Intubation	93.93±5.41	122.83±7.05	126.03±8.32	0.00	0.04	0.00
10 min Post-Intubation	81.13±5.02	107.80±6.06	121.87±7.84	0.00	0.04	0.00

(Test applied: ANNOVA)

(P < 0.05 is significant)

**Table 3: Comparison of mean diastolic blood pressure (DBP) among the groups**

<b>DBP mmHg</b>	<b>Group C (n = 30)</b>	<b>Group E (n = 30)</b>	<b>Group RL (n = 30)</b>	<b>P value C v/s E</b>	<b>P value C v/s RL</b>	<b>P value E v/s RL</b>
Pre- Induction	72.83±1.49	74.67±6.29	74.10±3.88	0.12	0.10	0.67
Post-Induction	67.87±1.68	81.30±6.55	69.47±4.09	0.00	0.03	0.04
Immediate-Intubation	81.00±2.60	93.10±8.43	88.57±5.42	0.00	0.02	0.01

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3 min Post-Intubation	66.53±3.37	86.63±7.04	83.70±5.31	0.00	0.02	0.01
5min Post -Intubation	57.90±1.58	79.53±6.84	81.90±5.36	0.00	0.02	0.02
10 min Post-Intubation	50.57±1.85	67.27±6.06	72.07±5.32	0.00	0.02	0.06

(Test applied: ANNOVA)

(P < 0.05 is significant)

**Table 4: Comparison of mean blood pressure (MBP) among the groups**

MBP mmHg	Group C (n = 30)	Group E (n = 30)	Group RL (n = 30)	P value C v/s E	P value C v/s RL	P value E v/s RL
Pre- Induction	91.03±2.13	89.53±6.01	90.83±5.28	0.20	0.84	0.37
Post-Induction	78.33±1.99	98.40±6.61	85.20±4.96	0.00	0.05	0.00
Immediate-Intubation	100.70±3.47	111.20±7.65	104.30±5.83	0.00	0.00	0.00
3 min Post-Intubation	80.93±2.79	102.90±6.92	99.83±5.44	0.00	0.02	0.04
5min Post -Intubation	72.70±2.17	94.80±6.67	92.30±5.81	0.00	0.00	0.03
10 min Post-Intubation	64.20±3.03	80.50±5.27	84.93±5.48	0.00	0.00	0.00

(Test applied: ANNOVA) (P < 0.05 is significant)

In the control group the mean percentage of change of heart rate from base line has increased from 78.10±3.86 /min to 115.9±1.45/min post induction and has decreased to 2.30±2.22/min at 5 min and to -0.85±1.64 at 10 mins. In the ephedrine group the percentage of change of heart rate from base line has increased from 79.40±4.79/min to 27.31 ±5.10/min post induction and has decreased to 20.66±4.30/min at 5 min and to 11.89± 4.22/min at 10 mins. In the RL group the heart rate increased from 79.23 ± 5.38/min to 7.28± 1.87/min post induction has decreased to 6.20±2.17 /min at 5mins 3.06±1.23 /min at 10min. Therefore, all the groups there was an increase in the heart rate post intubation which was maximum in ephedrine group followed by RL and then control group. On comparing the control ephedrine and RL group at varied intervals the decrease was statistically significant in control group. (P<0.00) On comparing ephedrine group with RL decrease is statistically significant in the ephedrine group. (P<0.01) (Fig 1).

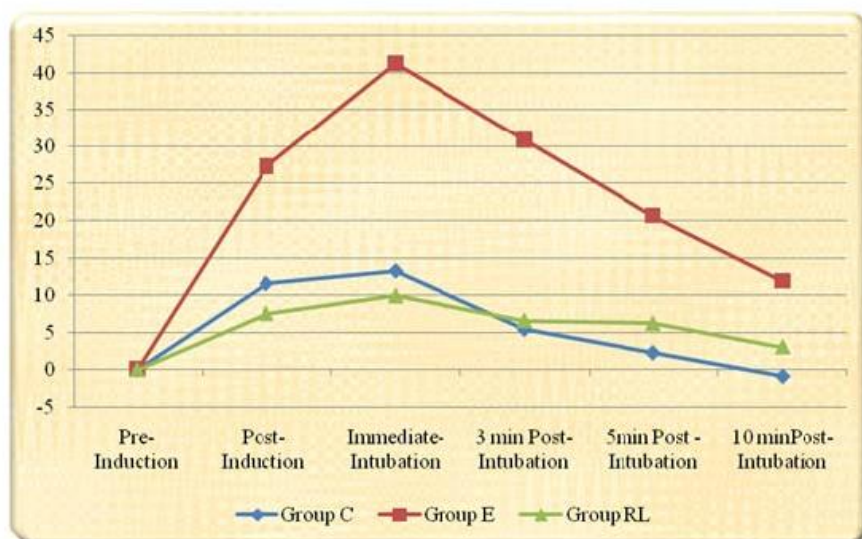
The mean percentage of change of systolic blood pressure in the control group from base line was 128.27±3.72 mmHg to -11.04±5.42 mmHg post induction and -26.73±4.39 mm Hg at 5 min and to -36.71±4.07 at 10 mins. In the ephedrine group it has increased from the baseline 120.53±6.38mmHg to 9.41 ±0.81mmHg post induction and the 4.88±1.49mmHg at 5 min and to -10±1.28mmHg at 10 mins .In the RL group it has decreased from 120.13± 7.45mmHg to -5.52± 1.56mmHg post induction and to 4.89±0.75 at 5mins 1.43±0.80 mmHg at 10min. Hence, the percentage change in the systolic blood pressure was maximum post intubation which was noticed

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in the ephedrine group followed by RL and then control group. However, on comparing them at varied intervals the decrease was statistically significant in control group. ( $P < 0.00$ ) On comparing ephedrine group with RL decrease was statistically significant in the ephedrine group. ( $P < 0.01$ ) (Fig 2)

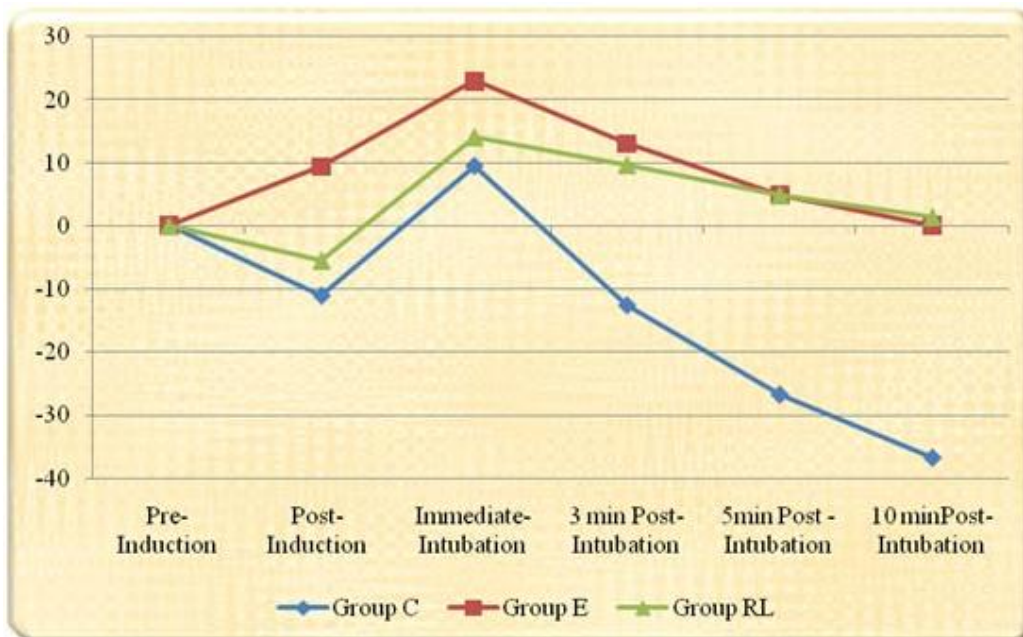
In the control group the mean percentage of change of diastolic blood pressure from base line has decreased from  $72.83 \pm 1.49$  mmHg to  $-6.82 \pm 1.28$  mmHg post induction and  $-0.51 \pm 1.24$  mmHg at 5 min and to  $-0.56 \pm 2.41$  at 10 mins. In the ephedrine group the percentage of change of diastolic blood pressure from base line has increased from  $74.67 \pm 6.29$  mmHg to  $8.96 \pm 3.07$  mmHg post induction and  $6.51 \pm 1.23$  mmHg at 5 min and to  $-9.94 \pm 1.51$  mmHg at 10 mins. In the RL group the diastolic blood pressure has decreased to from  $74.10 \pm 3.88$  mmHg to  $-6.26 \pm 2.23$  mmHg post induction  $10.54 \pm 4.85$  at 5 mins  $6.70 \pm 4.44$  mmHg at 10 min. In all the groups there was an increase in the diastolic blood pressure post intubation which was maximum in ephedrine group followed by RL and then control group. On comparing the control ephedrine and RL group at varied intervals the decrease was statistically significant in control group. ( $P < 0.00$ ) On comparing ephedrine group with RL decrease was statistically significant in the ephedrine group. ( $P < 0.01$ ) (Fig 3).

In the control group the mean percentage of change of mean blood pressure from base line has decreased from  $91.03 \pm 2.13$  mmHg to  $-6.26 \pm 0.84$  mmHg post induction and  $-20.14 \pm 1.28$  mmHg at 5 min and to  $-29.47 \pm 3.07$  at 10 mins. In the ephedrine group the percentage of change of mean blood pressure from base line has increased from  $89.53 \pm 6.01$  mmHg to  $9.91 \pm 0.09$  mmHg post induction and the has decreased to  $5.87 \pm 1.19$  mmHg at 5 min and to  $-10.08 \pm 1.03$  mmHg at 10 mins. In the RL group the mean blood pressure from has decreased to  $90.83 \pm 5.28$  mmHg to  $-6.19 \pm 1.21$  mmHg post induction  $6.01 \pm 0.86$  at 5 mins  $2.31 \pm 0.55$  mmHg at 10 min. In all the groups there was an increase in the mean blood pressure post intubation which was maximum in ephedrine group followed by RL and then control group. On comparing the control ephedrine and RL group at varied intervals the decrease was statistically significant in control group. ( $P < 0.00$ ) On comparing ephedrine group with RL decrease was statistically significant in the ephedrine group. ( $P < 0.01$ ) (Fig 4).

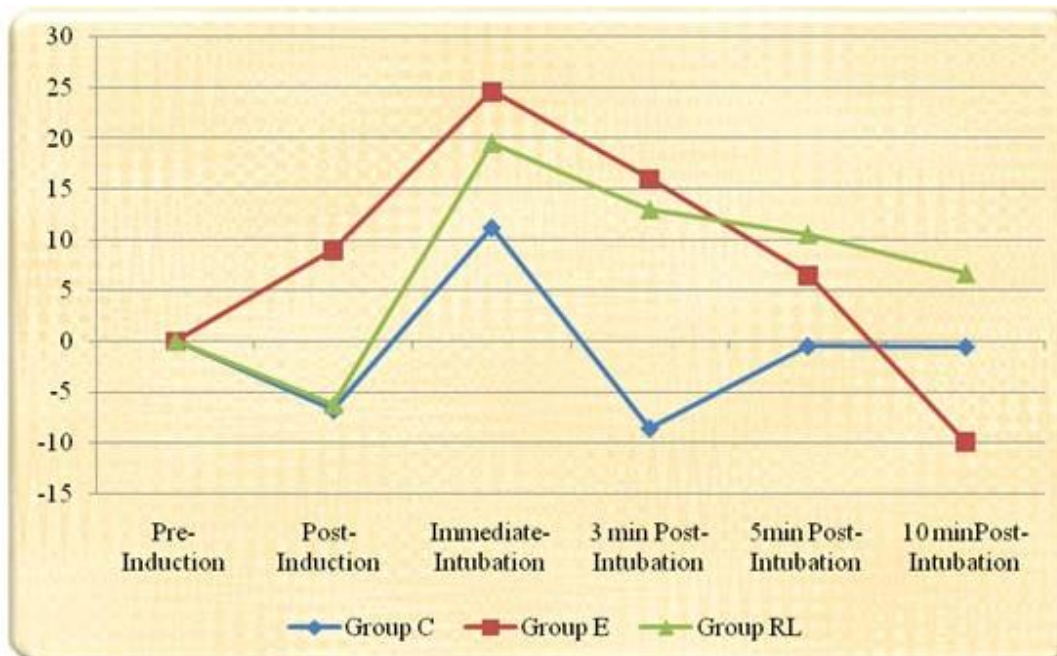


**Comparison of percentage of change of heart rate from base line among the groups (figure 1)**

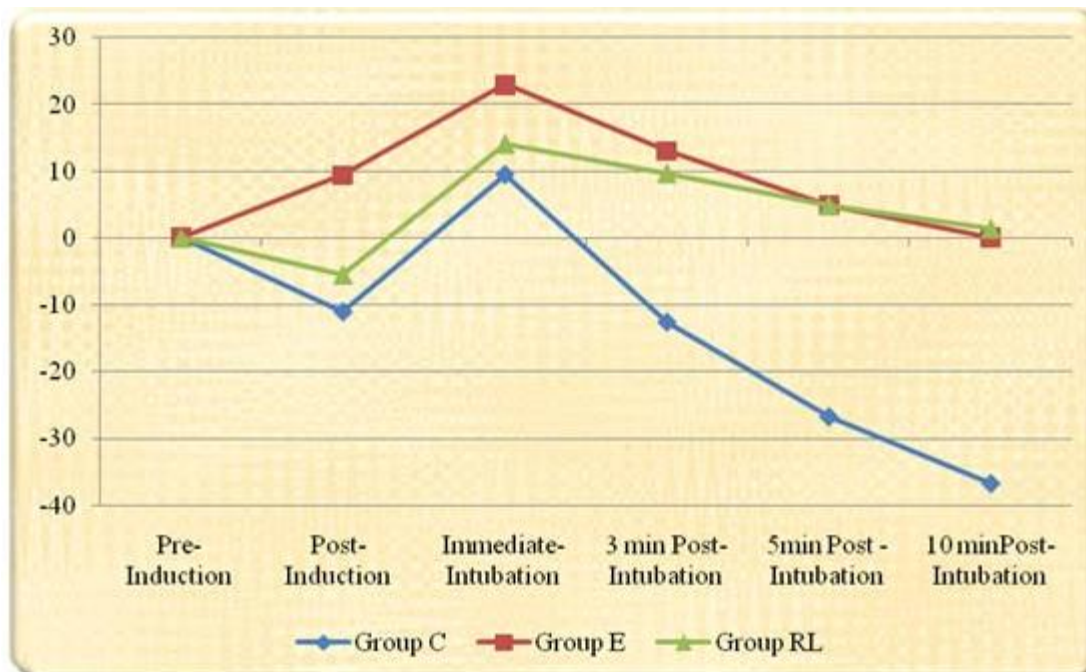
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**Comparison of percentage of change of systolic blood pressure from baseline among the groups (figure 2)**



**Comparison of percentage of change of diastolic blood pressure from baseline among the groups (figure 3)**



**Comparison of percentage of change of mean blood pressure from baseline among the groups (figure 4)**

**DISCUSSION:** The aim of the present study was to compare the three different regime which were propofol-placebo, propofol ephedrine and propofol crystalloid infusion in prevention of hypotension during induction of anaesthesia.

Propofol has been shown to cause hypotension due to its effects of peripheral vasodilatation by increased endothelial production and release of nitric oxide.<sup>23</sup>

The rationale for the prophylactic administration of sympathomimetics or crystalloid loading is to attenuate the anticipated decrease in the systemic vascular resistance or to maintain the right ventricular filling pressure respectively. Ephedrine and crystalloid infusion have been previously used safely and studied to combat the systemic hypotension after conduction of neuraxial blocks<sup>24</sup> and high dose of opioid induction.<sup>25</sup>

In our study, we have demonstrated a significant reduction in the arterial blood pressure i.e.  $11.04\% \pm 5.42$  after induction of anaesthesia with propofol in the control group which was in disagreement with 20-30% reduction in SBP as demonstrated by Edelist G. A<sup>26</sup>, Hugg CC.<sup>27</sup> but is in accordance with the study conducted by El Beheiry<sup>28</sup> who also reported a decrease of  $4.2 \pm 3\%$  in the control group as compared to ephedrine group and crystalloid group. The post intubation increase in the arterial systemic pressure all the groups represented the antagonism between the propofol-induced hypotension and the stress response to laryngoscopy and intubation. Our study also reported a mean peak decline in SBP by  $\sim 29\%$  at ten minutes after intubation this could be attributed to the induction dose of propofol along with the administration of positive pressure ventilation and administration of isoflurane (0.25%-0.5%) and this was in consistent with the study conducted by El beheiry et al<sup>28</sup> who also reported a decrease of mean arterial pressure of 30%.

Modest post-induction hypertension after the administration of ephedrine sulphate immediately before induction was in consistent with the study conducted by Michelsen et al<sup>18</sup> who



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have also observed attenuation of drastic decrease of blood pressure but not complete abolition of hypotension associated with propofol induction with the use of prophylactic ephedrine. Similar effects have been observed with the use of metaraminol.<sup>19</sup> Our findings are consistent with the findings of Gamlin et al<sup>29</sup> who have reported effectiveness of ephedrine in obtunding hypotensive effects of propofol when it was mixed with propofol.

Pre induction administration of ephedrine results in post-intubation hypertension, however, still there was a 10% decrease in the arterial blood pressure at 10 mins from the preinduction value. Therefore, it failed to protect against the delayed hypotension observed after propofol induction. There also was considerable increase in the heart rate 27% from the preinduction value to a mean of 41% just after intubation. Our findings were similar to the findings of El beheiry et al<sup>28</sup> who reported that the mean arterial blood pressure decrease of 12% from the preinduction value and a rise of heart rate to 41% post intubation from the baseline heart rate. Hence, preoperative ephedrine sulphate failed to prevent the delayed post intubation hypotension and led to excessive increase in the heart rate that may not be tolerated in the high risk patients.

On the other hand, pre-induction volume loading with Ringer's lactate does not fully abolished the post-induction decrease in SBP but the decrease was less as compared to the control group and at ten minute post intubation the decrease was 2% from the baseline value. The slight heart rate increases in the volume loading group were not different from preoperative values or from those of the control group but were less than those from the ephedrine group.

Hence, preoperative volume loading with 10ml/kg ringer lactate over 10-15 min successfully antagonized propofol induced hypotension without increments in the heart rate. Therefore, volume loading with Ringer's lactate provides more haemodynamic stability than the pre-induction administration of ephedrine sulphate. But, fluid preloading has its own limitations and side effects. We need to be cautious regarding preloading the patients with ringer lactate considering the preoperative status.

We have limited selection of our patients' upto the age group of 50 years only. So could not recommend using ringer lactate preloading for the prevention of propofol induced hypotension more than 50 years of age.

Our observation period was limited to 3min, 5min, and 10min post intubation. So we could not observe the percentage change in the hemodynamic variables every min.

In conclusion, preoperative administration of ephedrine failed to prevent delayed post intubation hypotension observed after induction with propofol and is associated with increase in the heart rate. On the other hand, preoperative volume loading with 10ml/kg of ringer lactate successfully antagonised propofol-induced hypotension without an increase in the heart rate, hence provide more haemodynamic stability.

### REFERENCES:

1. Claeys MA, Gepts E, Camu F. Haemodynamic changes during anaesthesia induced and maintained with propofol. *British Journal of Anaesthesia* 1988; 60: 3-9.
2. Grounds RM, Twigley AJ, Carli F, Whitwam JG, Morgan M. The haemodynamic effects of intravenous induction. *Anaesthesia* 1985; 40: 735-740.

## ORIGINAL ARTICLE

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3. Coates DP, Monk CR, Prys-Roberts C, Turtle M. Haemodynamic effects of infusions of the emulsion formulation of propofol during nitrous oxide anaesthesia in humans. *Anesthesia and Analgesia* 1987; 66: 64-70.
4. Muzi M, Berens RA, Kampine JP, Ebert TJ. Venodilation contributes to propofol-mediated hypotension in humans. *Anesth Analg* 1992; 74: 877-83
5. Yamaura K, Hoka S, Okamoto H, Kandabashi T, Akiyoshi K, Takahashi S. Changes in left ventricular end-diastolic area, end-systolic wall stress, and fractional area change during anesthetic induction with propofol or thiamylal. *J Anesth* 2000; 14: 138-42.
6. Reich DL, Hossain S, Krol M et al. Predictors of hypotension after induction of general anesthesia. *Anesth Analg* 2005; 101:622-8.
7. Benson M, Junger A, Fuch C, Quinzio L, Bottger S, Hempelmann G. Use of an anesthesia information management system (AIMS) to evaluate the physiologic effects of hypnotic agents used to induce anesthesia. *J Clin Monit Comput* 2000; 16: 183-9
8. Larsen R, Rathgeber J, Bagdahn A, et al. Effects of propofol on cardiovascular dynamics and coronary blood flow in geriatric patients. *Anaesthesia* 1988; 43(Suppl):25-31.
9. Brussel T, Theissen IL, Viefusson G. et al. Hemodynamic and cardio dynamic effects of propofol and etomidate: negative inotropic properties of propofol. *Anesth Analg* 1989; 69:35-40.
10. Robinson BJ, Ebert TJ, O'Brien TJ, et al. Mechanisms whereby propofol mediates peripheral vasodilation in humans. *Anesthesiology* 1997; 86:64-72.
11. Sellgren J, Ejnell H, Elam M, et al. Sympathetic muscle nerve activity, peripheral blood flows, and baroreceptor reflexes in humans during propofol anesthesia and surgery. *Anesthesiology* 1994; 80:534-44.
12. Coetzee A, Fourie P, Coetzee J, et al. Effect of various propofol plasma concentrations on regional myocardial contractility and left ventricular afterload. *Anesth Analg* 1989; 69:473-83.
13. Page1 I'S, Warltier DC. Negative inotropic effects of propofol as evaluated by the regional preload recruitable stroke work relationship in chronically instrumented dogs. *Anesthesiology* 1993;78:100-8
14. Chang KSK, Davis RF. Propofol produces endothelium independent vasodilation and may act as a Ca<sup>2+</sup> channel blocker. *Anesth Analg* 1993; 76:24-32.
15. Park WK, Lynch C III. Propofol and thiopental depression of myocardial contractility: a comparative study of mechanical and electrophysiologic effects in isolated guinea ventricular muscle. *Anesth Analg* 1992; 74:395-405.
16. Turner RJ, Gatt SP, Kam PC, Ramzan I, Daley M. Administration of a crystalloid fluid preload does not prevent the decrease in arterial blood pressure after induction of anaesthesia with propofol and fentanyl. *Brit J Anaesth* 1998; 80: 737-41.
17. Al- Ghamdi A. Hydroxyethyl starch 6.0% preload does not prevent the hypotension following induction with propofol and fentanyl. *Middle East J Anesthesiol* 2004; 17: 959-68.
18. Michelsen I, Helbo-Hansen HS, Kohler F, Lorenzen AG, Rydlund E, Bentzon MW. Prophylactic ephedrine attenuates the hemodynamic response to propofol in elderly female patients. *Anesth Analg* 1998; 86: 477-81.

# ORIGINAL ARTICLE

19. Chiu CL, Tew GP, Wang CY. The effect of prophylactic metaraminol on systemic hypotension caused by induction of anaesthesia with propofol in patients over 55 years old. *Anaesthesia* 2001; 56: 893-7.
20. Kasaba T, Yamaga M, Iwasaki T, Yoshimura Y, Takasaki M. Ephedrine, dopamine or dobutamine to treat hypotension with propofol during epidural anaesthesia. *Can J Anesth* 2000; 47:237-41.
21. Weeks S. Reflection of hypotension during caesarean section under spinal anaesthetic: do we need to use colloid? *Can J Anaesth* 2000; 47: 607-10.
22. Omoigui S. Sota Omoigui's Anesthesia Drug Handbook. 3<sup>rd</sup> ed. Malden: Blackwell Science 1999; 147.
23. Basu S, Mutschler DK, Larsson K, Kiiski R, Nordgren A, Eriksson MB. Propofol counteracts oxidative injury and deterioration of the arterial oxygen tension during experimental septic shock. *Resuscitation* 2001; 501: 341-8.
24. Clark RB, Thompson DS, Thompson CH. Prevention of spinal hypotension associated with cesarean section. *Anesthesiology* 1976; 45: 670-4.
25. Lowenstein E, Hallowell P, Levine FH, Daggett WM, Austen G, Laver MB. Cardiovascular response to large doses of intravenous morphine in man. *N Engl J Med* 1969; 281: 1389-93.
26. Edelist G. A comparison of propofol and thiopentone as induction agents in outpatient surgery. *Can J Anaesth* 1987; 34: 110-6.
27. Hugg CC Jr, McLeskey CH, Narwold ML, et al. Hemodynamic effects of propofol: data from over 25, 000 patients. *Anesth Analg* 1993~ 77: \$21-9.
28. El-Beheiry H, Milne B, Seegobin R Prophylaxis against the systemic hypotension induced by propofol during rapid-sequence intubation *Can J Anaesth* 1995 / 42:10 / pp 875-829
29. Gamlin F, Freeman J, Winslow L, Berridge J, Vucevic M. The haemodynamic effects of propofol in combination with ephedrine in elderly patients (ASA groups 3 and 4). *Anaesth Intensive Care* 1999; 27: 477-80.

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