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ORIGINAL ARTICLE

PREVENTION OF HYPOTENSION CAUSED BY INDUCTION OF ANAESTHESIA WITH PROPOFOL, A COMPARISON OF PRELOADING WITH 3.5% POLYMERS OF DEGRADED GELATIN SOLUTION, CRYSTALLOID (RINGER LACTATE) & INTRAVENOUS EPHEDRINE

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ABSTRACT: AIMS: In this study, we compared the effect of preloading with crystalloid, colloid and intravenous ephedrine against the hypotensive effects of propofol induction in ASA I-II patients scheduled for elective surgical procedures. **MATERIAL AND METHODS:** 120 patients aged 20yrs to 50yrs were randomly allocated to one of the four groups of 30patients each. Group-1 (control) did not receive any study medication, group-2 received degraded gelatin 10ml/kg over a period of 15min, group-3 received ringer lactate 20ml/kg over a period of 15 min and group-4 received intravenous ephedrine 0.2mg/kg prior to induction of anesthesia. Midazolam 1mg was given 1hour before induction of anesthesia. Anesthesia was induced with propofol 2.5mg/kg over 20 to 30 seconds. Heart rate and blood pressure were recorded before induction and then every minute for 5 minutes after induction of anesthesia. After the study period patients were intubated and anesthesia was continued as required. Hypotension was defined as a drop in systolic arterial pressure more than or equal to 20% of baseline. **RESULTS:** A significant decrease in systolic arterial pressure occurred in patients of group preloaded with ringer lactate as well as the control group. Less decrease in systolic arterial pressure was seen in the patients preloaded with degraded gelatine and ephedrine group. The incidence of hypotension was also lower in ephedrine group when compared with degraded gelatin group. **CONCLUSION:** We conclude that crystalloid preloading is not efficacious in preventing hypotension while as degraded gelatine and ephedrine markedly attenuates, but does not fully abolish the decrease in blood pressure caused by induction.

KEYWORDS: propofol, midazolam, degraded gelatine, ringer lactate and ephedrine.

INTRODUCTION: Induction of anaesthesia aims to produce a rapid and smooth transition from consciousness to unconsciousness by achieving adequate concentration of anaesthetic agents in the central nervous system. The induction of general anaesthesia and tracheal intubation can have significant hemodynamic consequences and many strategies have been used for limiting these.[1] Propofol (2, 6-diisopropylphenol) is a rapidly acting intravenous anaesthetic. Propofol has been used for both induction and maintenance of general anaesthesia. Administration of propofol 1.5 to 2.5mg/kg, as a rapid intravenous injection (15 sec) produces unconsciousness within 30 seconds.

Awakening is more rapid and complete than after induction of anaesthesia with other drugs used for intravenous induction of anaesthesia.[2] Anaesthesia induction with propofol is often associated with a significant decrease in arterial blood pressure. The precise mechanism of propofol induced hypotension is not known. Reduction in arterial blood pressure has been attributed to decrease in systemic vascular resistance or decrease in cardiac output caused by combination of

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venous and arterial vasodilatation,[3] depression of myocardial contractility and impaired baroreflex mechanism.[4] A propofol mediated decrease in sympathetic activity may explain all the hemodynamic changes, although direct vascular smooth muscle relaxation and direct negative inotropic effect may contribute to a lesser degree.[4,5] There are different ways to prevent propofol induced hypotension such as volume preloading and intravenous injection of various drugs available. Fluid preloading for caesarean section under regional anaesthesia has been established as routine and considered to be a safe and effective method of reducing the incidence of hypotension.[6] Ketamine and ephedrine have been co-administered in various studies to prevent or reduce this hypotension, with variable results. Low dose ketamine (0.5mg/kg) or ephedrine pretreatment can prevent hypotension due to propofol induction. Ketamine can also reduce the intensity of injection pain.[7] The present clinical study was undertaken to compare the effect of preloading with crystalloid (Ringer lactate) colloid (3.5% degraded gelatin) and the effect of prophylactic administration of vasopressin intravenous ephedrine in prevention of hypotension during propofol induction.

MATERIAL AND METHODS: After approval by institutional ethical committee and written informed consent from patients, the present study was conducted in the Postgraduate Department of Anaesthesiology and Critical Care Medicine, Government Medical College, Srinagar and associated hospitals during the study period of january 2011- September 2012. The study included 120 patients of either sex, aged between 20-50 years, belonging to either ASA Class I or II.

In order to avoid any interference with the results, following patients were excluded from this study:

- Patients with any significant cardiovascular, respiratory, endocrine or renal disease.
- Patients taking any drugs affecting the heart rate (e.g. -blockers) and blood pressure.
- Patients who are pregnant.
- Patients who are obese (BMI >40kg/m2).
- Patients with predicted difficult airway (Mallampati III or IV).

The present study compared the efficacy of fluid preloading with colloid “Haemaccel”, crystalloid “Ringer lactate” and intravenous ephedrine in prevention of hypotension during induction of anaesthesia with propofol.This study included 120 patients of ASA1 and ASA2 ageing 20 to 50 years of both gender undergoing routine elective surgery. All patients in this study were subjected to a detailed pre-anaesthetic evaluation and the presence of significant systemic disease and difficult airways were ruled out. All basic investigations according to our hospital protocol (like haemoglobin, fasting blood sugar, KFT, LFT, urine examination, chest x-rays and ECG) were checked. Patients were randomly allocated into four groups. All patients included in the study received 0.5mg of oral alprazolam as a premedication the night before surgery. Patients were kept fasting for 6-8 hours. Group 1 (Control group) comprised 30 patients received neither fluid nor ephedrine before induction of anaesthesia. Group 2 (Colloid group) comprised 30 patients received haemaccel 10ml/kg iv over a period of 15 minutes before induction of anaesthesia. Group 3 (Crystalloid group) comprised 30 patients received ringers lactate 20ml/kg iv over a period of 15minutes before induction of anaesthesia. Group 4 (Ephedrine group) comprised 30 patients received injection ephedrine 0.2mg/kg prior to induction of anaesthesia. All patients included in the study received 1mg midazolam intravenously 1 hour before the procedure as premedication. In the anaesthetic room,

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intravenous access was established using 18 guage cannula. Routine monitoring, i.e. electrocardiography, heart rate, pulse oximetry and NIBP was established. Baseline cardiovascular parameters i.e. heart rate, blood pressure (systolic, diastolic and mean) and oxygen saturation were recorded. Anaesthesia was induced with propofol 2.5mg/kg injected over 20-30 seconds. Propofol (2.5mg/kg) was used for induction of anaesthesia. In this period, bag and mask ventilation was used to maintain oxygen saturation greater than 95% and no endotracheal intubation was done. The usual maintenance and replacement fluid (ringer lactate) was started at the rate of 2ml/kg in all the patients. Heart rate and blood pressure (systolic, diastolic and mean) were recorded every minute starting 1minute after induction till 5minutes after propofol administration. Hypotension was defined as fall in blood pressure more than 20% from the base value. At the end of the study atracurium and tramadol was given to continue anaesthesia and surgery.

STATISTICAL ANALYSIS: Data was described as mean ± SD and percentage. Least significant difference for intergroup comparisons was measured at 95% CI. Student's t–test & Mann–Whitney U test were applied for intergroup comparisons, whereas overall comparison was done by F-test (ANOVA) and Kruskal-Wellis test. P-value upto three decimal places were considered. All the data was analysed in SPSS 11.5 software.

RESULTS: On comparison of the demographic data among the four groups no statistically significant difference was found (p value>0.05) as shown in table I.

Table II shows that on comparison of base line hemodynamic parameters among the four groups there was no statistically significant difference found (p value >0.05).

Table III shows that the heart rate decreased in group-I, group-II and group-III with respect to baseline, whereas the heart rate increased in group-IV. The change in heart rate at 1 minute was not statistically significant (p> 0.05) but significant at 2, 3, 4 and 5 minutes (p< 0.05).

As shown in table IV systolic blood pressure decreased in all the four groups after the induction of anaesthsia. The highest decrease in SBP was seen in group-I and lowest decrease was seen in group-IV.

Table V shows that diastolic blood pressure (DBP) decreased in all the four groups after induction of anaesthesia. The decrease in group-I was highest and group-IV was lowest among all the four groups. At 1 min the DBP was not statistically significant (p>0.05).

Table VI shows that the mean arterial pressure (MAP) decreased in all the four groups with time. The highest decrease in MAP was seen in group-I & lowest decrease was seen in group-IV. The difference was statistically significant (p<0.05).

Table VII shows the percentage of hypotension in the four groups. Percentage of hypotension was highest in control group (56.7%) at 3 minutes and lowest in ephedrine group (3.3%) at 5 minutes and was statistically significant (p<0.05).

DISCUSSION: Hypotension after induction of anaesthesia with propfol is well recognized.[8] The cause of this hypotension has been found to be a reduced systemic vascular resistance and a depression of myocardial contractility. The relaxation of smooth muscle produced by propofol is primarily due to inhibition of sympathetic vasoconstrictor nerve activity. For the purpose of this study we defined clinically significant hypotension as a decrease in blood pressure of greater than 20% below baseline measurements.[9]

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A number of techniques have been tried to counteract the hypotensive effects of propofol, for example slow administration of the drug, preloading with fluids (crystalloids and colloids) and admistration of different vasopressor drugs like ephedrine, dopamine, dobutamine and metaraminol to elevate blood pressure. The aim of our study was to evaluate the efficacy of fluid preloading (crystalloid and colloid) and intravenous ephedrine in prevention of hypotension during induction of anaesthesia with propofol. All the patients in the four groups were homogenous with respect to age, weight, sex distribution and baseline hemodynamic parameters (pulse, systolic blood pressure and diastolic blood pressure).

Group I patients did not receive any preloading fluid or vasoconstrictor before induction of anaesthesia with propofol. In group II fluid preloading was done with 10ml/kg body weight of 3.5% degraded gelatin intravenously over a period of 15 minutes before induction of anaesthesia with propofol. The same amount of preloading was used by Y Dhungana et al[9] and Mahendra Kumar et al[10] in patients without any cardiovascular side effects. Similarly in group III fluid preloading was done with 20ml/kg body weight of Ringer's lactate intravenously over a period of 15 minutes before induction of anaesthesia with propofol. The same amount of fluid preloading was used by Turner et al[11] and Kumar et al[10] in patients without any cardiovascular complications.

Numerous studies have been performed about the use of ephedrine before regional anaesthesia (spinal or epidural) and also about the use of intravenous ephedrine to prevent hypotension after induction of general anaesthesia with propofol. We used ephedrine in a dose of 0.2mg/kg body weight, given intravenous just before the induction of anaesthesia with propofol. The same dose was used by Iver Michelsenet al[12] and Dhungana et al[9] in ASA I and II patients without any cardiovascular adverse effects.

In our study, we observed that the systolic arterial pressure decreased in all the four groups during induction of anaesthesia with propofol. The highest decrease in systolic arterial pressure was seen in the control group and the lowest decrease in systolic arterial pressure was seen in ephedrine. We also demonstrated a significant reduction in the incidence of propofol induced hypotension with crystalloid preloading, colloid preloading and prior administration of intravenous ephedrine. We observed that crystalloid preloading, colloid preloading and ephedrine effectively maintained higher level of systolic blood pressure than control group. However, none of these three methods was fully effective in preventing the decrease in systolic blood pressure associated with propofol administration. Our findings are consistent with the findings of Turner et al[11] and Al-Ghamdi[13] who have shown lack of full effectiveness of preloading with crystalloids or colloids in preventing hypotension associated with propofol induction. In the studies conducted by Kumar et al[10] and Dhungana et al,[9] it was observed that fluid preloading attenuated the drastic fall of blood pressure but did not completely abolish the hypotension associated with propofol induction.

In our study, we observed that prophylactic intravenous ephedrine was more effective than crystalloid and colloid preloading in preventing the hypotension during propofol induction. But, ephedrine did not completely abolish the decrease in blood pressure associated with induction of anaesthesia with propofol. The results in the present study are comparable to those of Dhungana et al.[9] Michelsen et al[12] also found that prophylactic intravenous ephedrine 0.2mg/kg body weight significantly attenuated, but did not abolish the decrease in blood pressure during propofol and fentanyl induction. Gamlin et al[14] found that 15 or 20mg of ephedrine premixed with 20ml of 1% propofol maintained blood pressure at pre-induction values, whereas ephedrine 10mg was

insufficient to prevent hypotension. Similarly, El-Beheiryet al[15] found that ephedrine 0.07mg/kg given just before propofol induction and subsequent tracheal intubation maintained blood pressure at preinduction value for up to 6 minutes after induction. The reason that a smaller dose of ephedrine was effective is explained by the sympathoadrenal stimulating effect of intubation.

Although pre-induction ephedrine attenuated the hypotensive effects of propofol, some patients still experienced a decrease in blood pressure to >20% of baseline. The reason for this may be that ephedrine mainly maintains the blood pressure by increasing the cardiac output,[16] whereas propofol, under conditions similar to those in the present study, causes arterial hypotension by reducing peripheral vascular resistance.[6]

In our study, we observed decrease in heart rate in control group, crystalloid group and colloid group whereas heart rate is increased in the ephedrine group. Turner et al[11] reported decrease in heart rate in non-fluid preloaded and fluid preloaded patients after induction of anaesthesia with propofol. Kumar et al[10] observed that heart rate decreased in crystalloid preloaded patients after induction of anaesthesia with propofol and fentanyl. In our study, we observed increase in the heart rate in patients received ephedrine but it was less than 10% of the baseline and statistically significant. Gamlin et al[14] reported marked tachycardia associated with the use of ephedrine in combination with propofol in majority of patients. The difference in observations could be correlated with higher doses of ephedrine (20 and 25mg) in their study than in ours (0.2mg/kg). Dhungana et al[9] also reported insignificant increases in heart rate in patients receiving ephedrine.

We concluded that administration of ephedrine and preloading with haemaccel reduced the incidence of hypotension in significant number of patients as compared to preloading with ringer lactate and control group.

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Parameters	Control (mean ± SD)	Haemaccel (mean ± SD)	Ringer lactate (mean ± SD)	Ephedrine (mean ± SD)	p value
Age (years)	34.1±6.1 (25-50)	35.7±8.2 (23-50)	34.9±6.0 (28-49)	32.6±3.9 (25-40)	0.265 (NS)
Weight (kg)	(54.0±3.8) (45-60)	(54.7±4.6) (45-66)	(53.1±5.1) (40-65)	(55.3±4.7) (49-70)	0.238 (NS)
Gender (Male : Female)	5:25	5:25	4:26	6:24	

TABLE I: Comparison of demographic data of the four groups

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Parameters	Control	Haemaccel	Ringer Lactate	Ephedrine	p value
Pulse (beats/ minute)	83.4 ± 6.8 (71,98)	82.3 ± 5.8 (72,92)	82.0 ± 7.1 (72,104)	83.1 ± 6.8 (68,98)	0.830 (NS)
Systolic Blood Pressure(mmHg)	124.9 ± 8.7 (110,140)	124.5 ± 10.2 (110,140)	126.3 ± 8.9 (112,140)	125.5 ± 9.3 (110,140)	0.888 (NS)
Diastolic Blood Pressure(mmHg)	79.3 ± 5.9 (70,90)	78.3 ± 6.5 (70,90)	80.2 ± 6.8 (70,90)	79.4 ± 6.6 (70,90)	0.726 (NS)

Table II: Comparison (ANOVA) of Baseline Hemodynamic Parameters

Heart Rate	Control	Haemaccel	Ringer Lactate	Ephedrine	p value
15 min before Induction	84.8 ± 7.1 (70,102)	85.1 ± 5.7 (73,94)	84.3 ± 6.5 (74,95)	84.7 ± 7.9 (65,95)	0.976 (NS)
Induction (0 min)	86.7 ± 9.4 (72,113)	86.0 ± 5.9 (73,95)	85.7 ± 6.7 (76,99)	86.5 ± 10.3 (68,127)	0.960 (NS)
1 minute	87.9 ± 8.6 (69,112)	85.3 ± 5.6 (71,95)	85.7 ± 6.0 (74,96)	88.5 ± 11.0 (65,127)	0.343 (NS)
2 minute	84.1 ± 6.8 (67,96)	83.7 ± 5.7 (73,95)	85.4 ± 7.7 (77,110)	89.5 ± 9.1 (78,121)	0.011 (Sig)
3 minute	82.8 ± 6.0 (67,93)	81.8 ± 5.0 (69,91)	83.6 ± 7.1 (75,104)	89.3 ± 9.2 (78,118)	0.000 (Sig)

Table III: Overall Comparison (ANOVA) of Heart Rate

Systolic Blood Pressure	Control	Haemaccel	Ringer Lactate	Ephedrine	p value
15 min before Induction	120.9 ± 7.7 (110,136)	120.1 ± 5.4 (111,133)	120.3 ± 8.5 (109,137)	120.8 ± 8.7 (109,136)	0.973 (NS)
Induction (0 min)	122.2 ± 7.8 (111,137)	121.2 ± 5.8 (114,136)	121.7 ± 8.8 (110,140)	122.3 ± 8.7 (109,139)	0.947 (NS)
1 minute	99.1 ±6.3 (89,113)	110.0 ±6.6 (100,127)	103.5 ±11.5 (90,131)	115.6 ±8.6 (101,132)	0.000 (Sig)
2 minute	98.0 ±6.0 (88,113)	107.4 ±6.3 (97,124)	102.0 ±11.1 (90,129)	110.6 ±9.2 (95,127)	0.000 (Sig)
3 minute	96.9 ±5.9 (86,113)	105.5 ±6.0 (94,122)	101.1 ±11.5 (87,129)	108.3 ±9.1 (93,127)	0.000 (Sig)
4 minute	98.1 ±6.4 (87,115)	105.9 ±6.0 (94,122)	101.6 ±10.7 (87,127)	106.5 ±9.0 (90,122)	0.000 (Sig)
5 minute	98.8 ±6.3 (88,115)	106.0 ±8.7 (89,120)	101.4 ±9.6 (88,129)	107.0 ±8.7 (89,120)	0.000 (Sig)

Table IV: Comparison (ANOVA) of Systolic Blood Pressure

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Diastolic Blood Pressure	Control	Haemaccel	Ringer Lactate	Ephedrine	p value
15 min before Induction	81.0 ± 4.8 (70,88)	78.4 ± 5.3 (70,87)	79.3 ± 5.6 (71,88)	79.4 ± 5.9 (70,88)	0.328 (NS)
Induction (0 min)	81.9 ± 4.9 (70,90)	79.6 ± 5.6 (70,89)	80.8 ± 5.6 (71,90)	80.5 ± 6.0 (70,90)	0.461 (NS)
1 minute	60.1 ±5.7 (50,71)	63.2 ±6.0 (53,73)	63.3 ±5.7 (54,74)	64.1 ±6.2 (54,77)	0.050 (NS)
2 minute	56.7 ±6.0 (46,68)	60.5 ±5.6 (51,69)	59.9 ±6.1 (50,71)	61.8 ±6.2 (52,75)	0.011 (Sig)
3 minute	53.7 ±6.3 (42,66)	58.1 ±6.0 (48,67)	57.2 ±5.4 (48,68)	59.3 ±5.7 (50,73)	0.002 (Sig)
4 minute	52.8 ±6.9 (42,66)	58.5 ±5.9 (48,67)	55.2 ±4.7 (48,64)	58.3 ±5.8 (48,73)	0.000 (Sig)
5 minute	52.0 ±6.1 (42,63)	56.7 ±6.5 (46,67)	54.8 ±5.7 (45,67)	58.0 ±6.2 (48,71)	0.001 (Sig)

Table V: Comparison (ANOVA) of Diastolic Blood Pressure

Mean Arterial Pressure	Control	Haemaccel	Ringer Lactate	Ephedrine	p value
15 min before Induction	94.3 ± 4.1 (83.3,101.0)	92.3 ± 4.1 (85.0,99.0)	93.0 ± 4.7 (84.3,103.0)	93.2 ± 5.8 (83.7,102.0)	0.443 (NS)
Induction (0 min)	95.3 ± 4.3 (83.7,103.3)	93.5 ± 4.4 (86.0,101.3)	94.4 ± 4.8 (85.7,104.7)	94.4 ± 5.7 (85.0,102.0)	0.535 (NS)
1 minute	73.1 ±4.2 (65.3,82.7)	78.8 ±4.9 (71.7,87.7)	76.7 ±5.4 (69.0,89.7)	81.3 ±5.7 (72.0,92.3)	0.000 (Sig)
2 minute	70.5 ±4.2 (62.7,79.7)	76.1 ±4.5 (69.7,85.3)	73.9 ±5.4 (66.3,87.0)	78.1 ±5.8 (68.7,90.0)	0.000 (Sig)
3 minute	68.1 ±4.3 (59.7,77.7)	73.9 ±4.8 (67.0,83.3)	71.8 ±5.3 (64.7,85.0)	75.6 ±5.6 (66.3,87.3)	0.000 (Sig)
4 minute	67.9 ±4.7 (59.7,78.3)	74.3 ±4.6 (67.3,83.3)	70.6 ±4.8 (63.0,85.0)	74.4 ±5.6 (64.3,87.3)	0.000 (Sig)
5 minute	67.6 ±4.3 (59.3,77.0)	73.5 ±5.0 (66.0,82.0)	70.3 ±4.9 (63.0,84.3)	74.0 ±5.8 (63.0,86.7)	0.000 (Sig)

Table VI: Comparison (ANOVA) of Mean Arterial Pressure

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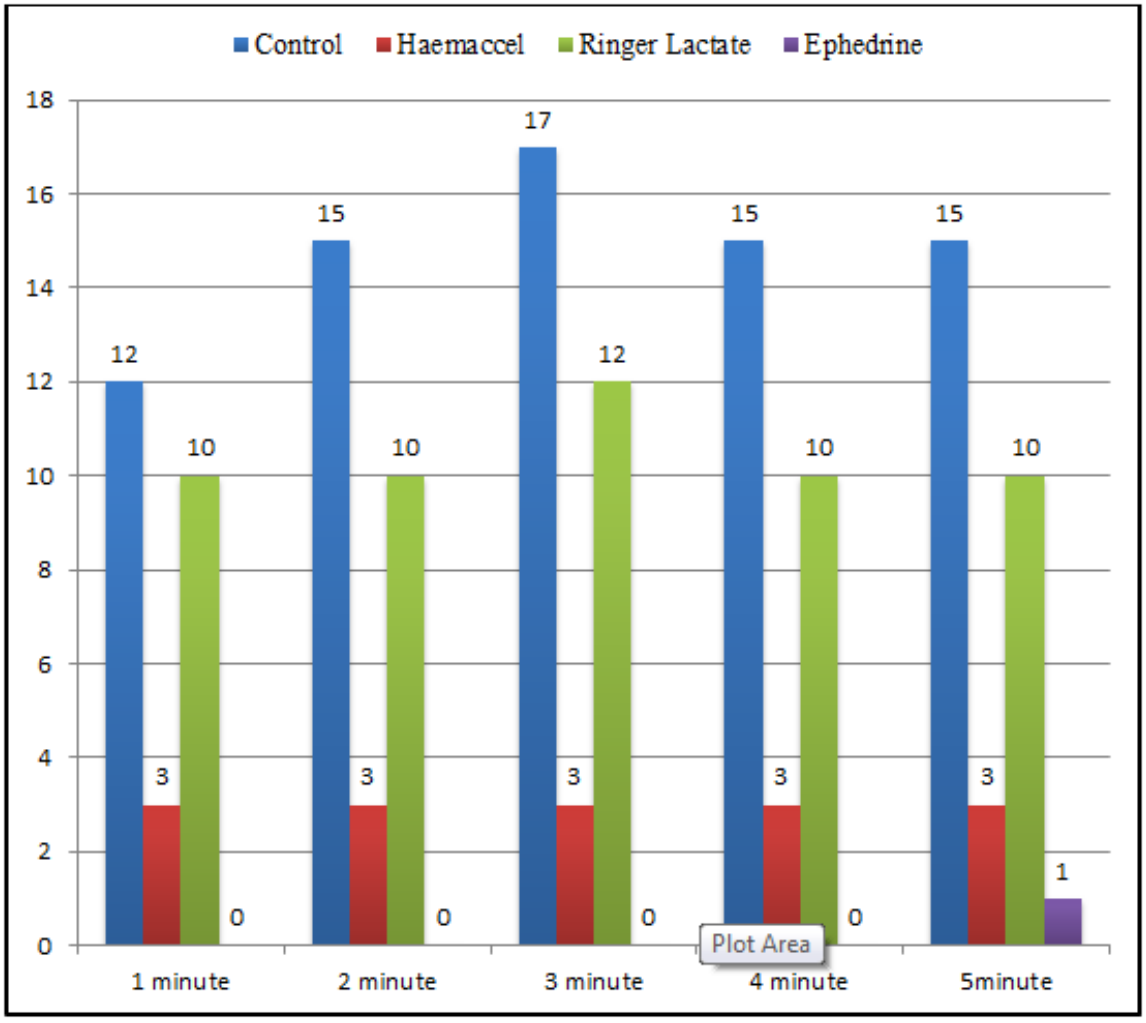
	Control		Haemaccel		Ringer Lactate		Ephedrine		p value
	n	%	n	%	n	%	n	%	
1 minute	12	40.0	3	10.0	10	33.3	0	0.0	0.000 (Sig)
2 minute	15	50.0	3	10.0	10	33.3	0	0.0	0.000 (Sig)
3 minute	17	56.7	3	10.0	12	40.0	0	0.0	0.000 (Sig)
4 minute	15	50.0	3	10.0	10	33.3	0	0.0	0.000 (Sig)
5 minute	15	50.0	3	10.0	10	33.3	1	3.3	0.000 (Sig)

Table VII: Percentage of hypotension in four groups



Percentage of hypotension in four groups

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