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OPTIMIZING HEMODYNAMICS DURING INDUCTION: CRYSTALLOID PRELOAD OR EPHEDRINE?

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Abstract
In this study, we compared the effect of preloading with crystalloid and intravenous ephed rine against the hypotensive effects of propofol and fentanyl induction in ASA I-II patients scheduled for elective surgical procedures. 150 patients aged 18yrs to 60yrs were randomly allocated to one of the three groups of 50 patients each. Group-A (control) did not receive any study medication, group-B received Ringers lactate 20ml/kg over 10-15min and group-C received intravenous ephedrine 0.2mg/kg prior to induction of anesthesia. Anesthesia was induced with propofol 2.5mg/kg, fentanyl 1.5µg/kg and atracurim 0.5mg/kg. Heart rate and blood pressure were recorded before induction and then every min for 5min 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. A significant decrease in systolic arterial pressure occurred in both the fluid loaded and the control group. Least decrease in systolic arterial pressure was seen in the ephedrine group. The incidence of hypotension was also lower in ephedrine group when compared with control group. We conclude that crystalloid preloading is not efficacious in preventing hypotension and ephedrine markedly attenuates, but does not fully abolish, the decrease in blood pressure caused by propofol and fentanyl induction.

Keywords: propofol, fentanyl, preloading and ephedrine.

Introduction

Propofol (2, 6 diisopropylphenol) is a rapidly acting IV anesthetic agent widely used for induction of general anesthesia [1]. Fentanyl is commonly used as a short acting analgesic agent with propofol. The induction of general anesthesia with propofol, however, has been associated with a decrease in systolic arterial pressure [2]. The mechanism of this hypotension is not well understood. The hypotensive effects of propofol has been attributed to a decrease in systemic vascular resistance caused by combination of venous and arterial vasodilatation [3]. Depression of myocardial contractility and impaired baroreflex mechanism also play a role [4,5]. The cardiovascular depressant effects of propofol are increased when fentanyl is added [6]. Various strategies have been attempted to prevent this hypotension with inconclusive evidence. Ketamine, ephedrine, atropine, glycopyrrolate, dopamine, dobutamine and metaraminol have

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been administered in various studies to prevent this hypotension, with variable results [7-10]. Fluid preloading with colloid and crystalloid has also been used to prevent the hypotensive effects of induction of anesthesia with these drugs [11, 12].

The present study was undertaken to compare, the effect of preloading with crystalloid (Ringer lactate) and the effect of prophylactic administration of intravenous ephedrine against the hypotensive effects of induction of anesthesia with propofol and fentanyl.

I. Patients and methods

After obtaining approval from the hospital ethics committee and informed consent we studied 150 patients, ASA I or II, scheduled for elective surgical procedures under general anesthesia. Patients with history of any cardiac, cerebrovascular, respiratory, endocrine, hepatic or renal disease were excluded from the study. Patients allergic to study medication, taking any drugs affecting heart rate or blood pressure, patients with anticipated difficult airway, morbid obesity (BMI>35) and pregnant females were also excluded. Patients were allocated using sealed envelope technique into three groups, to receive, no drug or fluid preload (Control groupgroup-A), 20ml/kg of ringers lactate over15-20min (Crystalloid group-group-B), or 0.2mg/kg of ephedrine (Ephedrine group-group-C).

The patients received no premedication. In the anesthetic room, intravenous access was established using a 18 gauge cannula. The usual maintenance and replacement fluid (normal saline) was started at the rate of 2ml/kg in all the patients. On shifting the patient to the operating room, 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. Noninvasive blood pressure was measured by using Datex-Engstrom Cardiocap II monitor. Patients allocated to receive a fluid preload were infused over 20min with ringers lactate, 20ml/kg. Patients allocated to ephedrine group received ephedrine 0.2mg/kg just prior to induction.

Anesthesia was induced with fentanyl 1.5µg/kg followed by propofol 2.5mg/kg injected over 30sec. Patients were given atracurium besylate 0.5mg/kg as muscle relaxant. We measured the heart rate, arterial blood pressure (systolic, diastolic and mean) and oxygen saturation every minute, starting 1min after induction till 5min after propofol injection. In this period, bag and mask ventilation was used to maintain oxygen saturation greater than 95% and no endotracheal intubation was done. 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. Hypotension was treated with rapid infusions of ringers lactate. The statistical analysis of categorical data was done by using Chi-square test. The quantitative data of the three groups was analyzed by using one way analysis of variance (ANOVA). All tests were referred for Pvalues for their significance. Any P-value less than 0.05 ($P<0.05$) was taken to be statistically significant. Data was presented as mean (±SD).The analysis of data was performed

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using comprehensive statistical software i.e. statistical package for social sciences (SPSS ver. 17.0), Chicago, USA for windows.

Results

150 patients were recruited to the study. All the groups were comparable with respect to age and body weight. The three groups were comparable with regard to baseline hemodynamic variables (TABLE 1).

Table-1: Demographic data and baseline hemodynamic parameters.

Parameters	Group-A mean±SD	Group-B mean±SD	Group-C mean±SD	P value
Age(years)	39.48±10.84	40.78±9.61	40.76±11.25	0.80
Weight(kg)	59.18±8.13	63.08±6.68	60.80±7.86	0.13
Heart rate(beats/min)	89.06±9.59	88.26±13.30	85.70±12.40	0.33
Systolic blood pressure(mmHg)	126.36±5.12	124.08±8.51	123.30±8.48	0.15
Diastolic blood pressure(mmHg)	76.50±3.91	75.98±7.06	77.46±7.94	0.13
Mean arterial pressure(mmHg)	93.18±3.64	92.14±7.16	92.86±7.85	0.11

Systolic blood pressure (SBP) decreased in all the three groups after the induction of anesthesia. The drop in systolic blood pressure over the study period was similar in group-A and group-B. In group-A SBP decreased to 95mmHg at 5min (75% of the baseline), in group-B SBP decreased to 97mmHg (78% of the baseline) and in group-C systolic blood pressure decreased to 103mmHg (84% of the baseline). The decrease in systolic blood pressure was highest in group-A and the lowest in group-C (TABLE 2).

Table-2: Comparison of systolic blood pressure during the study period.

Time (min)	Group-A mean±SD	Group-B mean±SD	Group-C mean±SD	P value
Baseline	126.36±5.12	124.08±8.51	123.30±8.48	0.15
1	102.36±7.10	105.38±8.91	106.56±12.76	0.01
2	93.28±8.67	96.58±8.72	98.94±13.39	0.01
3	94.12±8.60	95.72±15.26	100.28±8.30	0.03
4	95.38±6.87	96.68±13.65	100.40±6.31	0.04
5	95.38±6.55	97.22±9.73	103.72±5.44	0.00

Decrease in diastolic blood pressure (DBP) and mean arterial pressure (MAP) was also compared. DBP and MAP were similar in group-A and group-B. There were no significant differences in DBP and MAP between group-A and group-B. The decrease in DBP in group-A and group-B was similar and more than groupC. At 5min the DBP was statistically comparable among the three groups (TABLE 3).

Table-3: Comparison of diastolic blood pressure during the study period

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.Time (min)	Group-A mean±SD	Group-B mean±SD	Group-C mean±SD	P-value
Baseline	79.68±5.98	75.98±7.06	77.46±7.94	0.13
1	56.22±6.97	55.72±7.03	58.14±9.82	0.02
2	48.64±10.40	49.38±7.54	51.58±8.33	0.02
3	48.68±5.07	48.10±8.52	51.34±4.30	0.04
4	48.94±4.38	49.60±11.69	54.76±5.40	0.00
5	53.30±5.37	53.48±8.05	53.66±5.32	0.08

MAP decreased in all the three groups after the induction of anesthesia. The decrease was similar in group-A and group-B. The decrease in MAP in group-C was significantly less when compared to group-A and group-B (TABLE-4).

Table-4: Comparison of mean arterial pressure during the study period.

Time (min)	Group-A mean±SD	Group-B mean±SD	Group-C mean±SD	P-value
Baseline	93.18±3.64	92.14±7.16	92.86±7.85	0.11
1	72.36±7.04	73.98±7.45	73.80±10.23	0.01
2	63.56±8.92	64.88±7.32	66.74±9.60	0.02
3	63.86±5.18	63.70±10.03	66.92±4.23	0.04
4	64.64±4.43	65.24±11.89	69.62±4.11	0.02
5	68.52±4.92	67.78±7.16	69.78±4.75	0.01

Baseline heart rate (HR) was comparable in the three groups. In group-A and group-B it decreased following anesthetic induction. In group-C it increased from baseline following anesthetic induction (TABLE-
5).

Table-5: Comparison of heart rate during the study period.

Time (min)	Group-A mean±SD	Group-B mean±SD	Group-C mean±SD	P-value
Baseline	89.06±9.59	88.26±13.30	85.70±12.40	0.33
1	90.46±12.58	89.72±18.98	87.16±9.91	0.48
2	79.38±11.94	77.78±15.68	89.74±7.19	0.00
3	78.98±14.92	73.98±15.25	85.06±7.26	0.00
4	74.82±12.29	71.16±12.42	84.48±7.45	0.00
5	74.84±12.59	73.86±12.71	88.46±8.57	0.00

The incidence of hypotension in the three groups during the study period was also compared. The number of patients developing hypotension at 1min was not significant when compared among the three groups ($P>0.05$). The incidence of hypotension was significant at 2min, 3min, 4min and 5min ($P<0.05$). The

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incidence of hypotension during the study period was highest in group-A followed by group-B and group-C (TABLE-6).

Table-6: Number of patients developing hypotension and time of onset on hypotension.

Time	Number of patients developing hypotension			P value
	Group-A	Group-B	Group-C	
1min	11(22%)	10(20%)	9(18%)	0.13
2min	42(84%)	26(52%)	22(44%)	0.00
3min	42(84%)	26(52%)	23(46%)	0.00
4min	41(82%)	26(52%)	18(36%)	0.00
5min	36(72%)	27(54%)	10(20%)	0.00

Discussion

The present study confirms that induction of anesthesia with propofol and fentanyl in ASA-I and II patients is often associated with significant systemic arterial hypotension. The infusion of 20ml/kg of crystalloid preload does not prevent or attenuate the decrease in blood pressure after induction of anesthesia with propofol and fentanyl. Preinduction IV injection of ephedrine 0.2mg/kg significantly attenuated, but did not fully abolish the decrease in blood pressure.

Hypotension after induction of anesthesia with propofol is well recognized [2]. The cause of this hypotension has been found to be a reduced systemic vascular resistance and a depression of myocardial contractility [13]. Fentanyl was used to supplement induction of anesthesia with propofol. Fentanyl in low doses has minimal cardiovascular effects [14]. However when used with propofol for induction of anesthesia it may accentuate the hypotensive and bradycardic effects of propofol[6]. Significant decrease in systolic blood pressure from the baseline was observed in all the groups after propofol administration in our study also.

Our findings are consistent with the findings of Turner et al [11] and Al-Ghamdi [15] who have shown lack of full effectiveness of preloading with crystalloid or colloids in preventing hypotension associated with propofol. In the studies conducted by Kumar et al[12] and Dhungana et al[16] 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 IV ephedrine was more effective than crystalloid preloading in preventing the hypotension during propofol induction. But, ephedrine did not completely abolish the decrease in blood pressure associated with induction of anesthesia with propofol and fentanyl. The results in the present study are comparable to those of Michelsen et al[17]. They found that prophylactic IV ephedrine 0.2mg/kg significantly attenuated, but did not abolish, the decrease in blood pressure during propofol and fentanyl induction. Gamlin et al[18] found that 15 or 20mg of ephedrine premixed with 20ml of 1% propofol maintained blood pressure at preinduction values, whereas ephedrine 10mg was insufficient. Similarly, ElBeheiry et al[19] found that ephedrine 0.07mg/kg given just before propofol

induction and subsequent tracheal intubation maintained blood pressure at preinduction values for up to 6min after induction. The reason that a smaller dose of ephedrine is effective depends on the sympathoadrenal-stimulating effect of intubation. Although preinduction ephedrine attenuated the hypotensive effects of propofol, some patients still experienced a decrease in blood pressure to <80% of baseline. The reason for this may be that ephedrine mainly maintains the blood pressure by increasing the cardiac output[20], whereas propofol, under conditions similar to those in the present study, causes arterial hypotension by reducing peripheral vascular resistance[21,2].

In our study, we observed decrease in heart rate in control group and crystalloid group whereas heart rate 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 anesthesia with propofol. Kumar et al[12] observed that heart rate decreased in crystalloid preloaded patients after induction of anesthesia with propofol and fentanyl. In our study, we observed increase in the heart rates in patients receiving ephedrine but it was less than 10% of the baseline and statistically insignificant. Gamlin et al[22] 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 [16] also reported insignificant increases in heart rate in patients receiving ephedrine.

In conclusion we found that the administration of crystalloid preload does not prevent the decrease in arterial blood pressure after induction of anesthesia with propofol and fentanyl. The prophylactic intravenous injection of ephedrine 0.2mg/kg significantly attenuated, but did not abolish, the decrease in systolic blood pressure associated with induction of anesthesia with propofol and fentanyl.

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