Retraction Notice

Title of retracted article: A Comparison Study of the Effect of Different Doses of Ephedrine on Prevention of Hemodynamic Changes Associated with Induction of General Anesthesia by Propofol and Fentanyl

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Retraction initiative (multiple responses allowed; mark with X):

☐ All authors
☐ Some of the authors: X Editor with hints from
☐ Journal owner (publisher)
☐ Institution:
☐ Reader:
☐ Other:

Date initiative is launched: 2018-2-8

Retraction type (multiple responses allowed):

☐ Unreliable findings
☐ Lab error
☐ Inconsistent data
☐ Analytical error
☐ Biased interpretation
☐ Other:
☐ Irreproducible results
☐ Failure to disclose a major competing interest likely to influence interpretations or recommendations
☐ Unethical research

☐ Fraud
☐ Data fabrication
☐ Fake publication
☐ Other:
☐ Plagiarism
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☐ Editorial reasons
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Results of publication (only one response allowed):

☐ are still valid.
X invalid.

Author's conduct (only one response allowed):

☐ honest error
☐ academic misconduct
X none (not applicable in this case)
History
Expression of Concern:
yes, date: none

Correction:
yes, date: none

Comments:
The paper does not meet the standards of "Open Journal of Anesthesiology".

This article has been retracted to straighten the academic record. In making this decision the Editorial Board follows COPE's Retraction Guidelines. Aim is to promote the circulation of scientific research by offering an ideal research publication platform with due consideration of internationally accepted standards on publication ethics. The Editorial Board would like to extend its sincere apologies for any inconvenience this retraction may have caused.

Editor guiding this retraction: Professor Dr. Praveen Kumar Neema (EiC of OJAnes)
A Comparison Study of the Effect of Different Doses of Ephedrine on Prevention of Hemodynamic Changes Associated with Induction of General Anesthesia by Propofol and Fentanyl

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Abstract

**Background:** Propofol and fentanyl are commonly combined for general anesthesia. However, hypotension and bradycardia are common during anesthetic induction. The purpose of this study was to compare the response of different doses of ephedrine for attenuation of the hemodynamic changes following anesthetic induction without adverse effects. **Materials and Methods:** This was a randomized, double blinded, case-controlled clinical trial. One hundred and twenty adult patients allocated into one of the four groups: receiving IV, saline, ephedrine 0.05 mg/kg, 0.1 mg/kg, or 0.2 mg/kg respectively. Anesthesia was induced with fentanyl 2 mic./kg and propofol 2.5 mg/kg. Changes in systolic and diastolic blood pressures (SBP, DBP), mean arterial pressure (MAP), and heart rate (HR) were measured 1 min after induction, and 2, 3, 4 and 5 min then, intubation was done. **Results:** There was no significant difference between the four groups as regard to baseline hemodynamic variables. Patients received 0.1 mg/kg, and 0.2 mg/kg experienced less decrease in SBP, DBP, MAP, and HR with no significant increase in adverse effects. Numbers of patients with hypotension were significantly less in group received ephedrine 0.2 mg/kg. Use of IV ephedrine 0.1 mg/kg was effective for attenuation of hemodynamic changes but did not fully abolish the decrease in blood pressure. Ephedrine 0.2 mg/kg was better without causing any adverse effects. They **concluded** that 0.1 mg/kg of ephedrine is suitable for minimizing hemodynamic changes during propofol-fentanyl induction of anesthesia but 0.2 mg/kg was better without causing any adverse effects.


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1. Introduction

Propofol (2,6 diisopropylphenol) is a rapidly acting ideal IV anesthetic agent widely used for induction of general anesthesia [1]. Fentanyl is popular and 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 exact mechanism of this hypotension is not well understood. The hypotensive effects of propofol have 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 plays a role [4] [5]. The cardiovascular depressant effects of propofol are increased when fentanyl is added. Various strategies have been attempted to prevent this hypotension with inconclusive evidence. Ketamine, ephedrine, atropine, glycopyrrolate, dopamine, dobutamine and Fluid preloading with colloid and crystalloid have been administered in various studies to prevent this hypotension, with variable results [1]-[6]. Ephedrine is a non-catecholamine sympathomimetic alkaloid with potent alpha and beta agonist and acts by both direct as well as indirect mechanism. The indirect agonist properties of ephedrine may be due to peripheral postsynaptic norepinephrine release, or by inhibition of norepinephrine reuptake. Its cardiovascular effects include increase in blood pressure, heart rate, contractility, and cardiac output [7]. Ephedrine has been used to prevent the hypotensive effects of induction of anesthesia with these drugs [5] [6]. Ephedrine is not a potent arterial vasoconstrictor; it maintains BP mainly by increasing cardiac output and heart rate. This may be the reason that high doses of prophylactic intravenous ephedrine are associated with side effects such as reactive hypertension, which is usually considered as systolic BP > 140 mmHg [8]. The purpose of this study was undertaken to compare the response of different doses of ephedrine to investigate an optimal dose of ephedrine for attenuation of the hemodynamic changes following anesthetic induction with propofol and fentanyl without causing any adverse effects.

2. Patients and Methods

After obtaining approval from the hospital ethics committee and informed consent we studied 120 patients, ASA I or II, scheduled for elective surgical procedures under general anesthesia. Patients with history of any cardiac, cerebrovascular, respiratory, hepatic, renal or endocrinal 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...
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(BMI > 35) and pregnant females were also excluded. Patients were allocated using sealed envelope technique into four groups, no drug or (normal saline) Control group (group-A), 0.05 ml/kg of ephedrine (group-B), 0.1 mg/kg of ephedrine (group-C) or 0.2 mg/kg of ephedrine (group-D). The patients received no premedication. In the anesthetic room, intravenous access was established using a 18 gauge cannula. On shifting the patient to the operating room, routine monitoring 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. Non-invasive blood pressure was measured. Patients received normal saline, ephedrine 0.05, 0.1, 0.2 mg/kg just 1 min. prior to induction diluted in 10cc H2O by another person. Anesthesia was induced with fentanyl 2 μg/kg followed by propofol 2.5 mg/kg injected over 30 sec. Patients were given atracurium besylate 0.5 mg/kg as muscle relaxant. We measured the heart rate, arterial blood pressure (systolic, diastolic and mean) and oxygen saturation every minute, starting 1 min after induction till 5 min 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 four groups was analyzed by using one way analysis of variance (ANOVA). All tests were referred for P values 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 using comprehensive statistical software i.e. statistical package for social sciences (SPSS).

3. Results

120 patients were recruited to the study.

All the groups were comparable with respect to age and body weight. The four groups were comparable with regard to baseline hemodynamic variables (Table 1).

Systolic blood pressure (SBP) decreased in all the four 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 96.38 ± 6.55 at 5 min (drop 25% from the baseline), in group-B SBP decreased to 97.22 ± 8.73 mmHg (drop 22% from the baseline) and in group-C systolic blood pressure decreased to 103.72 ± 5.44 mmHg (drop16% from the baseline) and in group-D systolic blood pressure decreased to113.72 ± 4.44 mmHg (drop 10% from the baseline). The decrease in systolic blood pressure was highest in group-A and the lowest in group-D as illustrated in Table 2 & Figure 1.

Diastolic blood pressure (DBP) decreased in all the four groups after the in-
duction of anesthesia. Decrease in diastolic blood pressure (DBP) was similar in group-A and group-B and more than group-C and group-D. There was no

Table 1. Demographic data and baseline hemodynamic parameters.

<table>
<thead>
<tr>
<th></th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
<th>Group D</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (year)</strong></td>
<td>38.47 ± 10.84</td>
<td>39.77 ± 9.61</td>
<td>39.76 ± 10.25</td>
<td>39.77 ± 11.25</td>
<td>0.80</td>
</tr>
<tr>
<td><strong>ASA status</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>27 (90%)</td>
<td>26 (86.7%)</td>
<td>28 (93.3%)</td>
<td>28 (100%)</td>
<td>0.86</td>
</tr>
<tr>
<td>II</td>
<td>3 (10%)</td>
<td>4 (13.3%)</td>
<td>2 (6.7)</td>
<td>3 (10%)</td>
<td></td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F</td>
<td>13 (43.3%)</td>
<td>14 (46.7%)</td>
<td>12 (40%)</td>
<td>13 (46.7%)</td>
<td>0.45</td>
</tr>
<tr>
<td>M</td>
<td>17 (56.7%)</td>
<td>16 (53.3%)</td>
<td>18 (60%)</td>
<td>16 (53.3%)</td>
<td></td>
</tr>
<tr>
<td><strong>Weight (kg)</strong></td>
<td>62.18 ± 8.12</td>
<td>64.08 ± 6.67</td>
<td>62.80 ± 7.87</td>
<td>62.80 ± 7.87</td>
<td>0.13</td>
</tr>
<tr>
<td><strong>SBP (mm.Hg)</strong></td>
<td>127.36 ± 5.13</td>
<td>125.08 ± 8.51</td>
<td>124.30 ± 8.58</td>
<td>125.30 ± 8.58</td>
<td>0.15</td>
</tr>
<tr>
<td><strong>DBP (mm.Hg)</strong></td>
<td>78.68 ± 5.98</td>
<td>76.98 ± 7.06</td>
<td>77.46 ± 7.84</td>
<td>78.46 ± 7.74</td>
<td>0.13</td>
</tr>
<tr>
<td><strong>MAP (mm.Hg)</strong></td>
<td>94.18 ± 3.74</td>
<td>95.58 ± 7.26</td>
<td>93.86 ± 7.93</td>
<td>92.86 ± 7.85</td>
<td>0.11</td>
</tr>
<tr>
<td><strong>HR (b./min)</strong></td>
<td>88.06 ± 9.69</td>
<td>89.26 ± 12.30</td>
<td>87.70 ± 12.42</td>
<td>85.70 ± 12.42</td>
<td>0.33</td>
</tr>
</tbody>
</table>

Data are presented as mean ± Sd. *P-value < 0.05.

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

<table>
<thead>
<tr>
<th>Time</th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
<th>Group D</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>128.36 ± 5.35</td>
<td>129.08 ± 8.51</td>
<td>125.30 ± 8.58</td>
<td>126.30 ± 8.58</td>
<td>0.15</td>
</tr>
<tr>
<td>1 min.</td>
<td>102.36 ± 10.10</td>
<td>101.38 ± 8.91</td>
<td>105.56 ± 12.76</td>
<td>108.56 ± 12.76</td>
<td>0.01*</td>
</tr>
<tr>
<td>2 min.</td>
<td>94.28 ± 7.67</td>
<td>95.58 ± 8.72</td>
<td>99.94 ± 13.39</td>
<td>109.94 ± 8.39</td>
<td>0.01*</td>
</tr>
<tr>
<td>3 min.</td>
<td>94.12 ± 7.50</td>
<td>94.72 ± 14.26</td>
<td>100.28 ± 8.30</td>
<td>110.28 ± 7.30</td>
<td>0.03*</td>
</tr>
<tr>
<td>4 min.</td>
<td>95.38 ± 8.67</td>
<td>96.68 ± 12.65</td>
<td>101.40 ± 6.31</td>
<td>112.40 ± 6.31</td>
<td>0.04*</td>
</tr>
<tr>
<td>5 min.</td>
<td>96.38 ± 6.55</td>
<td>97.22 ± 8.73</td>
<td>103.72 ± 5.44</td>
<td>113.72 ± 4.44</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Data are presented as mean ± Sd. *P-value < 0.05.

Figure 1. Comparison of systolic blood pressure during the study period.
significant differences in DBP between group-A and group-B. In group-A DBP decreased to 53.30 ± 4.37 (drop 33% from the baseline) at 5 min, in group-B DBP decreased to 52.48 ± 8.05 (drop 32% from the baseline) mmHg and in group-C diastolic blood pressure decreased to 57.66 ± 5.32 mmHg (drop 25% from the baseline) and in group-D diastolic blood pressure decreased to 60.66 ± 6.32 mmHg (drop 16% from the baseline). The decrease in diastolic blood pressure was highest in group-B and the lowest in group-D as illustrated in Table 3 & Figure 2.

MAP decreased in all the four groups after the induction of anesthesia. The decrease was similar in group-A and group-B and no significant differences in MAP between group-A and group-B. The decrease in MAP in group-A and group-B was similar and more than group-C and group-D. In group-A MAP decreased to 67.52 ± 4.92 (drop 29% from the baseline) at 5min, in group-B MAP decreased to 66.78 ± 7.16 mmHg (drop 30% from the baseline) and in group-C mean blood pressure decreased to 69.78 ± 4.75 mmHg (drop 25% from the baseline) and in group-D mean blood pressure decreased to 72.78 ± 5.75 mmHg (drop 16% from the baseline). The decrease in mean blood pressure was highest in group-A and the lowest in group-D The decrease in MAP in group-C and

**Table 3.** Comparison of diastolic blood pressure during the study period.

<table>
<thead>
<tr>
<th>Time</th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
<th>Group D</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>78.69 ± 5.89</td>
<td>76.98 ± 7.36</td>
<td>76.46 ± 7.95</td>
<td>78.46 ± 7.93</td>
<td>0.13</td>
</tr>
<tr>
<td>1 min.</td>
<td>57.22 ± 7.97</td>
<td>56.72 ± 7.03</td>
<td>58.14 ± 9.82</td>
<td>62.14 ± 9.82</td>
<td>&lt;0.05*</td>
</tr>
<tr>
<td>2 min.</td>
<td>48.64 ± 10.40</td>
<td>49.38 ± 6.54</td>
<td>54.58 ± 8.33</td>
<td>63.58 ± 9.33</td>
<td>&lt;0.05*</td>
</tr>
<tr>
<td>3 min.</td>
<td>47.68 ± 6.07</td>
<td>48.10 ± 8.52</td>
<td>53.34 ± 4.30</td>
<td>66.34 ± 5.30</td>
<td>&lt;0.05*</td>
</tr>
<tr>
<td>4 min.</td>
<td>47.94 ± 4.58</td>
<td>48.60 ± 10.69</td>
<td>54.76 ± 5.40</td>
<td>64.76 ± 6.40</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>5 min.</td>
<td>53.30 ± 4.37</td>
<td>52.48 ± 8.05</td>
<td>57.66 ± 5.32</td>
<td>60.66 ± 6.32</td>
<td>0.08</td>
</tr>
</tbody>
</table>

Data are presented as mean ± Sd. *P-value < 0.05.

**Figure 2.** Comparison of diastolic blood pressure during the study period.
group-D was significantly less when compared to group-A and group-B as illustrated in Table 4 & Figure 3.

Baseline heart rate (HR) was comparable in the four groups. In group-A and group-B it decreased following anesthetic induction. In group-C and group-D it increased from baseline following anesthetic induction. In group-A H.R decreased to 72.84 ± 11.59 (drop 20% from the baseline) at 5 min, in group-B H.R decreased to 78.88 ± 11.71 (drop 11% from the baseline) and in group-C H.R increased to 88.46 ± 8.67 (increase 2% from the baseline) and in group-D H.R increased to 90.66 ± 7.57 (increase 5% from the baseline). The decrease in H.R was in group-A 20% and B11% and increase in group-C 2% and group-D 5% was insignificantly in all groups as illustrated in Table 5 & Figure 4.

The incidence of hypotension in the four groups during the study period was also compared. The number of patients developing hypotension at 1 min was not significant when compared among the four groups (P > 0.05). The incidence of hypotension was significant at 2 min, 3 min, 4 min and 5 min (P < 0.05). The incidence of hypotension during the study period was highest in group-A followed by group-B and group-C and group-D as illustrated in Table 6 & Figure 5.

<table>
<thead>
<tr>
<th>Time</th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
<th>Group D</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>94.18 ± 3.64</td>
<td>94.12 ± 7.16</td>
<td>93.86 ± 7.85</td>
<td>93.86 ± 7.85</td>
<td>0.11</td>
</tr>
<tr>
<td>1 min.</td>
<td>72.36 ± 6.04</td>
<td>72.98 ± 6.45</td>
<td>73.8 ± 10.23</td>
<td>77.80 ± 11.23</td>
<td>0.01*</td>
</tr>
<tr>
<td>2 min.</td>
<td>62.56 ± 7.92</td>
<td>62.88 ± 7.32</td>
<td>67.74 ± 9.60</td>
<td>74.74 ± 10.60</td>
<td>0.02*</td>
</tr>
<tr>
<td>3 min.</td>
<td>62.86 ± 4.18</td>
<td>62.70 ± 9.03</td>
<td>68.92 ± 4.23</td>
<td>73.92 ± 6.23</td>
<td>0.04*</td>
</tr>
<tr>
<td>4 min.</td>
<td>63.64 ± 4.43</td>
<td>64.24 ± 10.89</td>
<td>69.62 ± 4.11</td>
<td>76.62 ± 4.11</td>
<td>0.02*</td>
</tr>
<tr>
<td>5 min.</td>
<td>67.52 ± 4.92</td>
<td>66.78 ± 7.16</td>
<td>69.78 ± 4.75</td>
<td>79.78 ± 5.75</td>
<td>0.01*</td>
</tr>
</tbody>
</table>

Data are presented as mean ± Sd. *P-value < 0.05.

![Mean arterial pressure](RETRACTED)

**Figure 3.** Comparison of mean arterial pressure during the study period.
Table 5. Comparison of heart rate during the study period.

<table>
<thead>
<tr>
<th>Time</th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
<th>Group D</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>89.16 ± 9.59</td>
<td>87.26 ± 13.32</td>
<td>86.70 ± 11.40</td>
<td>85.70 ± 12.40</td>
<td>0.33</td>
</tr>
<tr>
<td>1 min.</td>
<td>90.46 ± 12.58</td>
<td>89.72 ± 16.98</td>
<td>87.16 ± 9.91</td>
<td>88.26 ± 9.91</td>
<td>0.48</td>
</tr>
<tr>
<td>2 min.</td>
<td>78.38 ± 11.94</td>
<td>79.58 ± 13.68</td>
<td>89.74 ± 7.29</td>
<td>90.74 ± 6.29</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>3 min.</td>
<td>76.98 ± 13.92</td>
<td>79.88 ± 13.25</td>
<td>85.06 ± 7.36</td>
<td>87.26 ± 8.36</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>4 min.</td>
<td>73.92 ± 12.29</td>
<td>77.26 ± 11.42</td>
<td>86.48 ± 7.55</td>
<td>88.58 ± 7.65</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>5 min.</td>
<td>72.84 ± 11.59</td>
<td>78.88 ± 11.71</td>
<td>88.46 ± 8.67</td>
<td>90.66 ± 7.75</td>
<td>&lt;0.001*</td>
</tr>
</tbody>
</table>

Data are presented as mean ± Sd. *P-value < 0.05.

Figure 4. Comparison of heart rate during the study period.

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

<table>
<thead>
<tr>
<th>Time</th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
<th>Group D</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 min.</td>
<td>7 (23%)</td>
<td>6 (20%)</td>
<td>6 (20%)</td>
<td>4 (15%)</td>
<td>0.13</td>
</tr>
<tr>
<td>2 min.</td>
<td>26 (85%)</td>
<td>19 (62%)</td>
<td>12 (40%)</td>
<td>9 (30)</td>
<td>0.05*</td>
</tr>
<tr>
<td>3 min.</td>
<td>26 (85%)</td>
<td>19 (62%)</td>
<td>13 (42%)</td>
<td>7 (25%)</td>
<td>0.05*</td>
</tr>
<tr>
<td>4 min.</td>
<td>24 (80%)</td>
<td>20 (65%)</td>
<td>11 (36%)</td>
<td>6 (20%)</td>
<td>0.05*</td>
</tr>
<tr>
<td>5 min.</td>
<td>22 (75%)</td>
<td>6 (20%)</td>
<td>16 (54%)</td>
<td>22 (75%)</td>
<td>0.05*</td>
</tr>
</tbody>
</table>

Data are presented as mean ± Sd. *P-value < 0.05.

4. Discussion

Hypotension after induction of anesthesia with propofol is well known [9]. The cause of this hypotension has been found to be a depression of myocardial contractility and a reduced systemic vascular resistance [10]. Fentanyl was used as adjuvant induction of anesthesia with propofol. Fentanyl in small doses has minimal cardiovascular effects [11]. However, when used with propofol for induction of anesthesia, it may accentuate the bradycardia and hypotensive effects of propofol [12]. 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. Preinduction IV injection of ephedrine 0.1 mg/kg significantly attenuated, but did not fully abolish the decrease in blood pressure, but 0.2 mg/kg much better, where the drop in systolic blood pressure from base line after 5 minute was 25% and 22% in group A and B but 16% in group C and only 11% drop in Group D. Also significant decrease in systolic blood pressure from the baseline was observed in all the groups after propofol administration in our study dropped 21%, 20%, 16%, 10% from the base line respectively A, B, C, D groups after 1 min. Gamlin et al. [13] found that 15 or 20 mg of ephedrine premixed with 20 ml of 1% propofol maintained blood pressure at preinduction values, whereas ephedrine 10 mg was insufficient. The difference in observations could be correlated with higher dose of ephedrine (15 mg, 20 mg and 25 mg) in other studies than in ours (0.2 mg/kg, mean dose 10 mg). In our study, we observed that prophylactic IV ephedrine was effective in preventing the hypotension during propofol induction in doses 0.1 mg/kg and 0.2 mg/kg. 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. [14]. They found that prophylactic IV ephedrine 0.2 mg/kg significantly attenuated, but did not abolish the decrease in blood pressure during propofol and fentanyl induction. Similarly, El-Beheiry et al. [15] found that ephedrine 0.07 mg/kg given just before propofol induction and subsequent tracheal intubation maintained blood pressure at preinduction values for up to 6 min 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 [16], whereas propofol, under conditions similar to those in the present study, causes arterial hypotension by reducing peripheral vascular resistance [2] [17]. Gopalakrishna and col-

Figure 5. Number of patients developing hypotension and time of onset of hypotension.
leagues [18] have reported ephedrine to be ineffective in preventing hypotension after induction of anesthesia with propofol and rocuronium during rapid tracheal intubation. However, Gamlin et al. [19] have reported full effectiveness in obtinguishing hypotensive effects of propofol when ephedrine was mixed with propofol. But, marked tachycardia was observed in majority of patients in their study. In our study, we observed decrease in heart rate in control group and increase in the ephedrine group, but it was less than 20% of the baseline and statistically insignificant. Gamlin et al. [20] 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 25 mg) in other studies than in ours (0.2 mg/kg). Dhungana et al. [18] also reported insignificant increases in heart rate in patients receiving ephedrine. In conclusion, we found that the prophylactic intravenous injection of ephedrine 0.1m g/kg significantly attenuated, but did not abolish, the decrease in systolic blood pressure associated with induction of anesthesia with propofol and fentanyl, but 0.2 mg/kg was much better without causing any adverse effects. We recommended that ephedrine reduced the incidence of hypotension in significant number of our ASA I and II grade patients, and their safety and efficacy needed to be used during routine clinical practice and in high risk groups and critical ill patients, especially ephedrine 0.2 mg/kg.

References


