Hemodynamic Response of Young Smokers to Induction and Intubation

Department of Anesthesiology and Pain Medicine, Yeungnam University College of Medicine, Daegu, Korea

Daelim Jee, M.D., and Ui-Kyun Park, M.D.

**Background:** To test whether smokers have exaggerated hemodynamic responses to induction and intubation, we investigated the changes in blood pressure (BP) and heart rate (HR) on induction and intubation in smokers and nonsmokers.

**Methods:** Healthy male patients (25 smokers and 25 nonsmokers, aged 20 to 29 yrs) for elective surgery were studied. Anesthesia was induced with thiopental 3 mg/kg, fentanyl 1.5μg/kg, vecuronium 0.1 mg/kg and maintained with enflurane 1% in N2O and O2 for the period of observation. After three minutes, orotracheal intubation was performed. Noninvasive BP and HR were recorded one minute before induction (baseline), immediately before intubation, and then every minute until five minutes after intubation.

**Results:** Systolic BP of smokers decreased significantly immediately before intubation, and at 4 and 5 min after intubation as compared to that of nonsmokers (Mean ± SEM, 120.8 ± 3.0 vs. 109.9 ± 2.8, 116.7 ± 2.4 vs. 108.9 ± 2.3, and 114.8 ± 2.2 vs. 106.7 ± 2.3 mmHg, respectively, P < 0.05). Diastolic BP of smokers decreased only immediately before intubation compared with that of nonsmokers (Mean ± SEM, nonsmoker 64.6 ± 2.4 vs. smoker 58.6 ± 1.8 mmHg, P < 0.05). However, there was no significant difference of HR between smokers and nonsmokers during the observational period.

**Conclusions:** Hemodynamic response is different in smokers compared with nonsmokers. Our results indicate that smoking affects the hemodynamic response to induction and intubation. *(Korean J Anesthesiol 2006; 50: S 14~8)*

**Key Words:** anesthesia, blood pressure, heart rate, smoking.

**INTRODUCTION**

Chronic cigarette smoking is well known to cause a consistent and widespread vascular endothelial injury in large and small arteries and arterioles with smooth muscle cell proliferation. These structural changes may impair the buffering function of the arterial vasculature. Baroreflex sensitivity is also reported to be depressed in chronic smokers. These changes might result in exaggerated rises or falls of blood pressure when sympathetic system is stimulated or inhibited as in the case of anesthetic induction and tracheal intubation.

In contrast to these observations, Laxton et al. reported that the transient increase in blood pressure after tracheal intubation did not appear to be affected by chronic exposure to cigarette smoke whereas the heart rates of smokers were significantly greater than those of nonsmokers. They speculated that the increase in heart rate might be related to an increased upper airway reflex. However in their study, smokers had a significantly lower baseline heart rate and blood pressure than nonsmokers. The ages of the patients were relatively heterogeneous. As a result, age-dependent changes of the cardiovascular function might not reflect the difference of hemodynamic responses between smokers and nonsmokers in their study. Moreover, because the study was undertaken in women, in whom cardiovascular regulatory function including baroreflex sensitivity and sensitivity to catecholamines might be different from men, the hemodynamic response to induction and intubation in male smokers might be different and the results may not be applicable to men or postmenopausal women.

We hypothesized that current smokers might have exaggerated hemodynamic responses to induction and intubation. To test this hypothesis, we investigated the differences in blood...
pressure and heart rate during anesthetic induction and intubation between young healthy male smokers and nonsmokers.

**MATERIALS AND METHODS**

With institutional approval and written informed consent, 50 consenting ASA physical status 1 healthy male patients (twenty-five smokers and twenty-five nonsmokers, aged 20 to 29 years) undergoing elective surgery under general anesthesia participated in this study. Patients with the following criteria were excluded from the study: patients in whom difficulty with laryngoscopy was expected or those with abnormal or absent dentition; patients with coexisting systemic illness and those taking cardiovascular medications; history of bronchial asthma; history of laryngeal or tracheal surgery or pathology; or patients in whom tracheal intubation was not completed within 20 seconds (defined as the time from insertion to removal of the laryngoscope). For the smokers, we selected, arbitrarily, the patients with a history of smoking ≥ six months.

Each patient was premedicated with fentanyl 1µg/kg by intramuscular injection 1 hour prior to surgery. In the operating room, the selected patient characteristics (age, weight, height, and number of cigarette smoked) were recorded and a recording of arterial blood pressure and heart rate was obtained after a period of stabilization. For the smokers, smoking was not allowed for 12 hours prior to surgery. All patients received a standardized anesthetic protocol. The patients were not allowed to eat or drink for 8 h and had no intravenous administration of fluid prior to anesthesia. Anesthesia was induced with thiopental (3 mg/kg) and fentanyl (1.5µg/kg) IV. Vecuronium (0.1 mg/kg IV) was used for tracheal intubation with a 7.5 mm diameter endotracheal tube using a Macintosh blade. Patients were ventilated to a PaO2 of 33–37 mmHg with enflurane 1% (end-tidal concentration 0.6–0.7%) in nitrous oxide (4 L/min) - oxygen (2 L/min) for the whole period of observation. Monitoring included automated noninvasive blood pressure, electrocardiogram, oxygen saturation (SpO2), end-tidal enflurane concentration, and PaO2. Three minutes after initiation of induction, direct laryngoscopy and intubation was performed. Systolic and diastolic blood pressure, and heart rate were recorded 1 min before induction and served as the baseline values. Subsequent measurements were taken immediately before and every one-minute until five minutes after tracheal intubation. Nurses who were blinded to whether or not the patients were smokers recorded the data. The same experienced anesthesiologist, who was blinded to the smoking history, performed anesthetic induction and intubation in every case.

Data were presented as mean ± SEM. Two-way repeated measures ANOVA and independent t-test were used for between group comparisons. One-way repeated measures ANOVA was performed to analyze within group differences from baseline. A P value less than 0.05 was considered statistically significant. SPSS 10.0 software (SPSS, Inc., Chicago, IL) was used for the statistical analysis.

**RESULTS**

All patients enrolled in the study completed it. No significant differences between smokers and nonsmokers were found regarding age, weight, and height (Table 1). Baseline measurements of systolic blood pressure, diastolic blood pressure, and heart rate were comparable between the two groups (Fig 1).

Systolic blood pressures of smokers decreased significantly immediately before intubation, at 4 and 5 min after intubation as compared with that of nonsmokers (Mean ± SEM, 120.8 ± 3.0 vs. 109.9 ± 2.8, 116.7 ± 2.4 vs. 108.9 ± 2.3, and 114.8 ± 2.2 vs. 106.7 ± 2.3 mmHg, respectively, P < 0.05). Systolic blood pressure increased only at 1 min after intubation both in nonsmokers (Mean ± SEM, 121.6 ± 1.5 to 140.0 ± 3.0 mmHg, P < 0.001) and in smokers (Mean ± SEM, 123.2 ± 1.6 to 131.2 ± 3.5, mmHg, P < 0.05) when compared with the baseline, with no significant difference between smokers and nonsmokers (Mean ± SEM, 140.0

**Table 1. Patient Characteristics**

<table>
<thead>
<tr>
<th></th>
<th>Age (yr)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>Pack-year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoker</td>
<td>23.2 ± 0.7</td>
<td>67.9 ± 2.1</td>
<td>173.8 ± 1.0</td>
<td>5.9 ± 0.9</td>
</tr>
<tr>
<td>Nonsmoker</td>
<td>24.5 ± 0.6</td>
<td>69.9 ± 2.0</td>
<td>174.3 ± 1.0</td>
<td>0</td>
</tr>
</tbody>
</table>

Values are mean ± SEM. No significant difference was seen between smokers and nonsmokers.
± 3.0 vs. 131.2 ± 3.5, mmHg, P = 0.06). Systolic blood pressure decreased immediately before intubation, at 3, 4, and 5 min after intubation in smokers (Mean ± SEM, 123.2 ± 1.6 to 109.9 ± 2.8, 113.0 ± 3.2, 108.9 ± 2.4, and 106.7 ± 2.3 mmHg, respectively, P < 0.05) but decreased only at 5 min in nonsmokers (Mean ± SEM, 121.6 ± 1.5 to 114.8 ± 2.2, P < 0.05) compared with the baseline. Diastolic blood pressure of smokers decreased only immediately before intubation compared with that of nonsmokers (Mean ± SEM, nonsmoker 64.6 ± 2.4 vs. smoker 58.6 ± 1.8, P < 0.05).

Diastolic blood pressure changed significantly at all time points except 1 min after intubation compared with the baseline in both smokers and nonsmokers during the observational period (P < 0.05). Heart rate increased only at 1 min in smokers (Mean ± SEM, 76.2 ± 1.4 to 87.2 ± 3.1 beat/min, P < 0.01) while it increased at 1 and 2 min in nonsmokers after intubation (Mean ± SEM, 73.9 ± 1.2 to 91.0 ± 3.7 or 82.8 ± 3.8 beat/min, respectively, P < 0.01) compared to the baseline, with no significant difference between smokers and nonsmokers during the observational period (Fig. 1).

**DISCUSSION**

Although some authors reported detrimental effects of smoking concerning anesthesia and surgery, the hemodynamic responses of smokers during anesthesia induction and intubation are not well defined. To determine whether current smokers might result in exaggerated hemodynamic responses during induction and intubation, we examined the differences in systolic blood pressure, diastolic blood pressure, and heart rate between young male healthy smokers and nonsmokers. In this study, we found that the fall of systolic and diastolic blood pressure during induction, and systolic blood pressure during last minutes of observational period are of greater magnitude in smokers than in nonsmokers. We also found that the increase in heart rate was prolonged in nonsmokers compared with smokers after intubation. As result, the hemodynamic response is different in smokers compared with nonsmokers, indicating that current smokers affect the hemodynamic response to induction and intubation.

Our results suggest that smokers might be more sensitive to anesthetic cardiovascular depression (during induction and last minutes of the observational period). Pathophysiologic changes resulted from chronic smoking are vascular endothelial injury, smooth muscle cell proliferation, atherosclerosis initiation and progression, all these increasing vascular stiffness. McVeigh et al. suggested that modifications in wall structure associated with cigarette smoking might impair the buffering function of the arterial vasculature since cigarette smoking is associated with consistent and wide spread changes in small arteries and arterioles. In combination of these changes, reduced baroreceptor sensitivity would produce an exaggerated fall of blood pressure in response to the cardiovascular depressant effect of anesthetics in smokers. These might explain that the greater fall of systolic blood pressure immediately before intubation, at 4 and 5 min after intubation in smokers. Hemodynamic responses following tracheal intubation were expected to be exaggerated in smokers due to the decreased buffering function. However we could not find any exaggerated rise of blood pressure in smokers compared with nonsmokers after intubation. We speculate that this might be due to blunted vasoconstrictor responses to norepinephrine in smokers. The increase of heart rate from the baseline was prolonged in nonsmokers compared with smokers after intubation with no difference between the two groups in our study. However, Laxton et al. reported that heart rate of smokers was significantly greater than that of nonsmokers immediately after intubation in young and middle-aged women. We speculate that this discrepancy might result from the difference of age distribution and gender between the two studies.

Bennett and Richardson reported that chronic tobacco
smoke exposure in experimental animals for periods as short as 4-6 wk altered the reflex regulation of the cardiovascular system. For human beings, a marked decline in endothelium-dependent vasomotor response was observed over a six-month period in current smokers. These reports suggest that alterations in cardiovascular regulation might occur after smoking of shorter duration. In this regard, we arbitrarily selected the patients with history of smoking ≥ six months.

The clinical impact of this study per se is minor because the young healthy patients in the present study could accommodate the hemodynamic changes during anesthesia induction. However, because advancing age, hypertension, or gender is a well-known patient characteristic to influence the hemodynamic response to induction and intubation, smokers in these conditions might be more sensitive to anesthetic cardiovascular depression than nonsmokers. Therefore, care of smokers with impaired cardiovascular function should be more cautious during induction and intubation.

The benefits of short-term cessation of smoking before surgery are confined to the cardiovascular system. However, our study indicates that the exaggerated fall of blood pressure during induction may be expected in smokers. This hemodynamic response could increase the risk of cardiovascular complication in spite of the well-recognized benefits of short-term cessation of smoking. Thus giving up smoking 12 hrs before surgery may not be enough for cardiovascular benefits.

In summary, hemodynamic response is different in smokers compared with nonsmokers during induction and intubation. The difference of the response might result from the depressed baroreflex sensitivity, impaired arterial vascular buffering function, and/or impaired vasoconstrictor response in smokers. Smoking might be another patient characteristic that might affect the hemodynamic response to induction and intubation.

REFERENCES

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