The Comparison of Hemodynamic Responses Following Laryngeal Mask Airway Insertion Versus Tracheal Intubation in Hypertensive Patients Scheduled for Elective Ophthalmic Surgery Under General Anesthesia

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Abstract: We compared hemodynamic responses following laryngeal mask airway insertion versus tracheal intubation in hypertensive patients who were scheduled for elective ophthalmic surgery under general anesthesia. We studied 48 controlled hypertensive patients that were randomly divided into two groups (n=24) for insertion of laryngeal mask airway (LMA) and endotracheal intubation (EI). The mean arterial blood pressure (MAP), heart rate, rate pressure product (RPP), and ST-segment changes were recorded preoperatively, immediately preintubation and 1, 3, and 5 minutes after LMA insertion or tracheal intubation in all patients and compared between two groups. There was a reduction in MAP after induction and immediately preintubation in all of patients of both groups (P<0.05). The MAP, heart rate and RPP increased immediately after both LMA insertion and tracheal intubation (P<0.05). The elevation of MAP and RPP were maintained for longer time in intubation group versus LMA group (P<0.05). There was no difference between the groups with respect to ST-segment variation. The incidence of airway injury was similar between two groups. The laryngeal mask airway insertion may be preferable to endotracheal intubation in hypertensive patients where attenuation of hemodynamic stress response is desired.

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Keywords: Laryngeal mask airway; Endotracheal intubation; Hypertension

Introduction

Tracheal intubation after induction of general anesthesia is accompanied by transient rising of blood pressure and heart rate. This response is a sympathetic reflex and is provoked by stimulation of oropharynx. Although, these circulatory responses are transient and with little consequence in healthy subjects, they are unpredictable and more hazardous in hypertensive patients (1). Hypertensive patients may be more vulnerable to mechanical damage and pressure to airway tissue that induces by endotracheal intubation and led to more circulatory responses (2). The sympathetic nervous system has more activity in hypertensive patients, moreover, marked increase in catecholamine concentration in these subjects is more than normotensive cases, and then these patients exhibit exaggerated circulatory responses to the intubation (3-5). In order to decrease the undesirable circulatory responses to intubation especially in hypertensive patients, we can use a different intubation device or attenuate the hemodynamic responses with pharmacological agents. Insertion of the laryngeal mask airway (LMA) identified lower circulatory changes after induction of general anesthesia than tracheal intubation (6,7). Because insertion of LMA similar to that of establishing an oropharyngeal airway does not need direct exposure of larynx and may be less stimulating than laryngoscopy and tracheal intubation (8,9). The hemodynamic responses after tracheal intubation can precipitate life-threatening cardiovascular events such as pulmonary edema, myocardial infarction, and cerebrovascular hemorrhage in hypertensive patients especially with cardiovascular disease (1,10). The rate pressure product (RPP) is an index of myocardial oxygen consumption and was calculated by multiplying systolic blood pressure by heart rate. This value was used for analysis of the LMA insertion and intubation-
induced stress response. It was shown that high RPP value together with ST-segment changes (elevation or depression) might be early sign of myocardial ischemia (11). In the following randomized study, we assessed the hemodynamic responses of LMA insertion compared to endotracheal intubation in hypertensive patients who were scheduled for elective ophthalmic surgery under general anesthesia.

**Materials and Methods**

The study was approved by hospital ethics’ committee, and informed consent was obtained from our subjects. We studied 48 controlled hypertensives (ASA physical status II) patients aged between 45 and 78 years who were scheduled for elective ophthalmic surgery under general anesthesia. According to World Health Organization criteria, hypertension was defined if systolic blood pressure more than 160 mm Hg and/or diastolic blood pressure were more than 95 mmHg. Patients with a history of systolic blood pressure on three occasions less than 180 mm Hg and diastolic less than 105 mmHg during admission were included in this study. Exclusion criteria were age<18 years, history of serious pulmonary, cardiac, central nervous system, or cervical spine disease, and a history of difficult intubation and gastroesophageal reflux. All of our subjects were evaluated by cardiologist to optimize antihypertensive regime before operation. All of hypertensive subjects received their antihypertensive medications such as beta-blockers, calcium channel blocker, diuretic and angiotensin-converting enzyme inhibitors (ACEIs) approximately 3 hours before the induction. Hypertensive patients were randomly divided by opening a sealed envelope to two equal-size groups (n=24) for insertion of LMA (LMA group) and endotracheal intubation (EI group). Premedication was with oral diazepam 5 mg 1.5 hours before operation in all patients. Our standard monitoring in this study was an electrocardiograph capable of ST-segment analysis, a noninvasive blood pressure monitor, capnography and a pulse oximeter in the operating room. Oxygen was administered via face mask for 5 minutes before induction. For reduction of propofol injection pain, lidocaine 0.5 mg/kg was administered in all patients. General anesthesia was induced with 2.5 mg/kg propofol and 1 µg/kg fentanyl and maintained with isoflurane 1% in oxygen. Muscle relaxation was achieved with 0.5 mg/kg atracurium. In the LMA group, LMA was inserted using a single-handed rotational technique. LMA with size 3, 4, and 5 was used for patients< 60 kg, 60-80 kg and > 80 kg in weight respectively. The cuff of LMA 3, 4 and 5 were inflated with air of 20 ml, 30 ml and 40 ml, respectively and an anesthesia circuit was connected. The LMA was sealed until optimal ventilation was obtained. We routinely measured the LMA intracuff pressure by using manometer and by deflating the intracuff pressure to be lower than 44 mmHg. In the endotracheal intubation group, tracheal tube with internal diameter of 8 and 7 mm was established for male and female with a size 3 Macintosh, respectively. Successful intubation was identified with capnography. The mean arterial blood pressure (MAP), heart rate and ST-segment changes (degree of depression or elevation) were recorded preoperatively as baseline value, immediately preintubation and were also measured at 1, 3 and 5 minutes after LMA insertion or tracheal intubation. The systolic blood pressure and heart rate were multiplied to determine the RPP value. All of hemodynamic variables of both groups were stored in the monitor’s memory. For decreasing bias and error, the stored data were verified by two other anesthesiologists. Myocardial ischemia was identified as reversible ST-segment changes lasting at least one minute and described as 1 mm (elevation or depression) shift from baseline. The sample size of our study was based on difference of 20 mm Hg in blood pressure and 20 bpm in heart rate respectively, and a power of 0.8 and error of 0.05. Blood pressure and heart rate were tested using analysis of variance repeated measures. Statistical comparisons were performed by analysis of variance (ANOVA), followed by student’s t-test. All values were expressed as mean±standard deviation. Significance was taken as P<0.05.

**Results**

Baseline hemodynamic values, demographic characteristics and concurrent medications were similar between two groups (Table 1). Face mask ventilation was easy in all patients, and there were no failed LMA insertion and endotracheal intubation. There was a reduction in MAP after induction and immediately preintubation in all of patients of both groups (P<0.05). However, the heart rate increased immediately preintubation in all subjects in both groups. Heart rate also increased more immediately after intubation compared to insertion of LMA and remained elevated for three minutes after LMA insertion or tracheal intubation (P<0.05). The MAP increased immediately after both LMA insertion and tracheal intubation (P<0.05). Moreover, RPP increased in both groups
immediately after LMA insertion and tracheal intubation ($P<0.05$). The elevation of MAP and RPP were maintained for longer time in intubation group versus LMA group ($P<0.05$) (Table 2). A clinically significant ST-segment change was identified in two patients of intubation group, but no significant ST changes were observed in the LMA group. There was no difference between the groups with respect to ST-segment variation. Eight patients in the intubation group and two patients in the LMA group were treated for hypertension immediately after intubation or LMA insertion with intravenous labetalol. Three patients in intubation group and none patient in LMA group appeared transient premature ventricular contractions (PVC) immediately after intubation, and none of them needed treatment. The incidence of airway injury was similar between two groups. Postoperative airway complications had no differences between two groups.

**Table 1. Demographic characteristics and concurrent medications of both groups**

<table>
<thead>
<tr>
<th>Variables</th>
<th>LMA Group</th>
<th>EI Group</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$n$</td>
<td>24</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>Age (yr.)</td>
<td>56±18</td>
<td>58±12</td>
<td>0.23</td>
</tr>
<tr>
<td>Men/Female</td>
<td>14/10</td>
<td>13/11</td>
<td>0.82</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>68±14</td>
<td>70±16</td>
<td>0.64</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>158±10</td>
<td>161±14</td>
<td>0.24</td>
</tr>
<tr>
<td>Antihypertensive medication</td>
<td>Beta blocker</td>
<td>16</td>
<td>18</td>
</tr>
<tr>
<td>Alpha-blocker</td>
<td>2</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Calcium channel blocker</td>
<td>8</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Renin-angiotensin-inhibitor</td>
<td>20</td>
<td>18</td>
<td></td>
</tr>
</tbody>
</table>

All variables are expressed as mean ± SD. LMA= laryngeal mask airway, EI= endotracheal intubation

**Table 2. Hemodynamic variables in the LMA group and endotracheal intubation group**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group</th>
<th>Baseline</th>
<th>Before LMA insertion/EI intubation</th>
<th>Immediately after LMA insertion/EI intubation</th>
<th>After LMA insertion/EI intubation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1 min</td>
</tr>
<tr>
<td>Mean arterial pressure (MAP)(mmHg)</td>
<td>LMA</td>
<td>99±14</td>
<td>85±8*</td>
<td>112±16*</td>
<td>110±8*</td>
</tr>
<tr>
<td></td>
<td>EI</td>
<td>100±12</td>
<td>86±9*</td>
<td>124±18*</td>
<td>122±12*</td>
</tr>
<tr>
<td>Heart rate (HR) (bpm)</td>
<td>LMA</td>
<td>82±14</td>
<td>84±16*</td>
<td>98±18*</td>
<td>96±10*</td>
</tr>
<tr>
<td></td>
<td>EI</td>
<td>84±10</td>
<td>86±18*</td>
<td>108±16*</td>
<td>102±16*</td>
</tr>
<tr>
<td>RPP value</td>
<td>LMA</td>
<td>10356±2781</td>
<td>10066±1078</td>
<td>16566±3477*</td>
<td>14877±2388*</td>
</tr>
<tr>
<td></td>
<td>EI</td>
<td>11445±1678</td>
<td>10887±2178</td>
<td>19234±3408*</td>
<td>16456±1864*</td>
</tr>
</tbody>
</table>

All variables are expressed as mean ± SD. LMA = laryngeal mask airway, EI= endotracheal intubation, RPP=rate pressure product, *: $P<0.05$ (Immediately after LMA insertion or EI intubation versus baseline), #: $P<0.05$ (LMA group versus EI group)

**Discussion**

Our study demonstrated that heart rate, blood pressure, and RPP increased after LMA insertion and tracheal intubation in hypertensive patients. Moreover, these hemodynamic changes after tracheal intubation were greater than LMA insertion. Our results identified that oropharyngeal and tracheal stimulation by intubation induces greater responses than LMA insertion in hypertensive patients.

LMA significantly attenuates hemodynamic responses when compared with tracheal intubation (12,13). Moreover, previous studies showed that hemodynamic stress responses were exaggerated in hypertensive subjects compared with normotensive cases (3,14) and these findings may be related to an increased level of serum catecholamine and also, increased peripheral vessel sensitivity to catecholamine (5,15). The circulatory responses after tracheal intubation are more serious in hypertensive patients compared to normotensive cases and lead to significant increase in blood pressure and may cause myocardial ischemia, left ventricular failure and cerebral hemorrhage (11). Some studies concluded that the hemodynamic responses that induced by LMA insertion are less than tracheal intubation (3,6,7). In a trial by Kihara et al., concluded that stress responses with intubating LMA were less compared to tracheal intubation...
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intubation (12). However, some trials found that intubating LMA failed to attenuate the hemodynamic responses such as blood pressure and RPP compared to tracheal intubation (11,12,16). It is likely that these contrasting results are related to number of attempts of intubation, duration of intubation and force during laryngoscopy. Shribman et al., showed that the serum level of catecholamine and circulatory values of 10 seconds’ laryngoscopy were equal to laryngoscopy and then intubation (17) and another study revealed that level of catecholamine and hemodynamic responses after three seconds’ laryngoscopy were lower than laryngoscopy and then intubation (18). Therefore, the hemodynamic responses and serum level of catecholamine have a direct relationship with duration of laryngoscopy. Laryngoscopy for tracheal intubation leads to elevation of epiglottis and exposure of the glottis and cause enhancing sympathetic activity and lead to increased blood pressure and heart rate especially in hypertensive patients. However, insertion of LMA prevents the need for laryngoscopy and does not directly stimulate the receptors in the larynx; therefore, produces less adverse cardiovascular stress responses. Although, these responses are transient, but may lead to myocardial ischemia or infarction, pulmonary edema, and intracranial hemorrhage in hypertensive patients (1,19-22).

RPP normally is less than 12000, and it is shown that RPP> 20000 is more commonly associated with angina and myocardial ischemia (23,24). Kanaide et al., compared the hemodynamic variables such as RPP and ST-segment changes of two groups of patients that underwent intubation with Lightwand device and a laryngoscope in octogenarian hypertensive patients and concluded that the major cause of hemodynamic responses to tracheal intubation was stimulation of the trachea by the tube. The disturbance of myocardial oxygenation with increased systolic blood pressure along with increased heart rate may be more than increased blood pressure alone (25). In our study, we found that the level of RPP is more than 20000 in some cases in intubation group and by use of LMA device, this critical increase of RPP value was prevented. Moreover, we found that increased RPP in both groups in our study was due to increased systolic blood pressure and increased heart rate and RPP was higher during 3 minutes following tracheal intubation compared to LMA insertion.

It was shown that 1 mm ST-segment changes as depression or elevation on the ECG led to single 1 min episode of myocardial ischemia and increased the risk of cardiovascular events 10-fold (26,27). In our study, only two patients observed significant ST-segment changes that were part of the tracheal intubation group. It should be noted that the ST software of our monitoring has moderate sensitivity and specificity compared to Holter monitoring and this can be a reason for the low variations of ST changes in our study. Moreover, Theodoraki et al., found that ST-segment monitoring on the ECG could not assess the myocardial ischemia due to circulatory responses of laryngoscopy and tracheal intubation (28,29). Also, patients with left ventricular hypertrophy due to hypertension may present a typical pattern ST depression and asymmetrical T wave inversion because of disturbance of the repolarization processes. Therefore, the cause of ST-segment changes during anesthesia may be related to left ventricular hypertrophy.

In our study, the pharyngolaryngeal complications were not different between the two groups. We used manometer for evaluation of cuff pressure of endotracheal tube and LMA during operation. It was shown that by control of cuff pressure after LMA insertion, the airway complications were reduced by 70% (30-32).

Our study has some possible limitations. First, we evaluated the subjects with normal airway, and we know that in patients with a difficult airway, the intubation time is longer and lead to different results between endotracheal intubation group and LMA group. Therefore, our finding may not be applicable to subjects with difficult airway. Second, collection of data in this study was not blinded to the device applied. But, the hemodynamic data verified by two other anesthesiologists and this strategy may reduce the bias and error of this study. Third, our results were related to anesthesia with usual, and routine anesthetic technique and use of formulary anesthesia agents and may not be applicable to other anesthesia regimes, such as the use of large dose of narcotics. Fourth, our hypertensive patients did not undergo routine stress test, echocardiography, and coronary angiography prior surgery for evaluation of end-organ damage secondary to hypertension. However, all of our patients were evaluated by cardiologist prior to operation and had controlled hypertension with no target-organ damage.

We concluded that intense tracheal stimulation by the use of endotracheal tube induced greater hemodynamic responses than stimulation following LMA insertion in hypertensive patients. The LMA insertion may be preferable to endotracheal intubation in hypertensive patients where attenuation of
hemodynamic stress response is desired.

References

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