INFLUENCE OF HEAD FLEXION AFTER ENDOTRACHEAL INTUBATION ON INTRAOCULAR PRESSURE AND CARDIO-RESPIRATORY RESPONSE IN PATIENTS UNDERGOING CATARACT SURGERY

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ABSTRACT

Background: During preparation and draping of periorbital area, neck flexion causes displacement of the endotracheal tube tip toward the carina. Stimulation of the tracheal mucosa may cause bucking, increased intraocular pressure (IOP), laryngospasm, bronchospasm, change in end-tidal carbon dioxide pressure (PETCO2) or peripheral arterial haemoglobin oxygen saturation (SpO2) during light anaesthesia.

Objective: To investigate the influence of head and neck flexion after endotracheal intubation on heart rate (HR), systolic and diastolic blood pressure (SAP and DAP), SpO2, PETCO2 and IOP in patients undergoing cataract surgery during general anaesthesia.

Method: In this prospective observational study, 106 ASA physical status I and II patients scheduled for elective cataract surgery under general anaesthesia were studied. Anaesthesia was induced with thiopental sodium, lidocaine and fentanyl. Atracurium 0.5 mg/kg was given to facilitate tracheal intubation. HR, SAP, DAP, SpO2, PETCO2, and IOP were measured at 1, 2, and 5 minutes after head flexion.

Results: Mean SAP, DAP, IOP, and HR were significantly increased after head flexion compared with baseline values (P < 0.05). PETCO2 and SpO2 were significantly decreased at 1 and 2 minutes after head flexion compared with baseline values (P < 0.001).

Conclusion: It is concluded that endotracheal tube movement by changes in head and neck position has significant effects on heart rate, systolic and diastolic blood pressures, laryngeal reflexes, SpO2, PETCO2, and intraocular pressure in patients undergoing cataract surgery under general anaesthesia.

Key Words: endotracheal intubation, intraocular pressure, head and neck positioning, pressor responses, respiratory responses

INTRODUCTION

Cataract is a common cause of visual impairment in older individuals. Cataract extraction is usually performed under regional eye block or general anesthesia. After induction of general anesthesia and endotracheal intubation, the periorbital area is prepared and draped. For this purpose, the patient's head and neck is usually flexed 30 to 45 degrees. Neck flexion causes displacement of the endotracheal tube tip toward the carina for about 3.1-5.5 mm.

Stimulation of the tracheal mucosa by endotracheal tube displacement induces bradycardia, arrhythmias, hypotension, laryngospasm, bronchospasm, coughing, and apnoea will occur in light anesthesia. Inflation of the endotracheal cuff also induces above signs. Changes in depth of anaesthesia can modify the laryngeal and respiratory responses to tracheal irritation.

Laryngoscopy and endotracheal intubation are the anesthesia-related practices most likely to increase intraocular pressure (IOP) significantly, that is, by at least 10 to 20 mm Hg. The mechanism is not clear, but it probably relates to sympathetic cardiovascular responses to tracheal intubation. Laryngeal constriction and all components of the tracheal response caused by tracheal stimulation following endotracheal tube movement may impair end-tidal carbon dioxide pressure (PETCO2) and peripheral arterial haemoglobin oxygen saturation (SpO2). This study investigated the influence of head and neck flexion after endotracheal intubation on heart rate (HR), systolic and diastolic blood pressure (SAP and DAP), SpO2, PETCO2, wheezing, coughing, stridor, and IOP in patients undergoing cataract surgery.

MATERIALS AND METHODS

After obtaining approval from the our institutional ethics committee and written informed consent, in a prospective observational study, 100 ASA physical status I and II patients, aged 40-80 years, were scheduled for elective cataract surgery under general anaesthesia with duration between 40 and 70 minutes. Patients with anticipated difficult tracheal intubation, direct laryngoscopy lasted more than 15 seconds, respiratory disease or recent respiratory tract infection, cardiovascular disease, IOP more than 20 mmHg, and cigarette smokers were excluded from the study. Patients with more than...
15 seconds laryngoscopy were excluded because pro-
longed duration of direct laryngoscopy may affect the mag-
nitude of circulatory stimulation associated with
tracheal intubation.\textsuperscript{13}

After establishing intravenous access and routine moni-
tors, patients were ventilated with 100% oxygen. An-
aesthesia was induced with thiopental sodium 5 mg/kg,
lidocaine 1.5mg/kg and fentanyl 1.5 µg/kg. Atracurium
0.5 mg/kg was given to facilitate tracheal intubation.
Approximately 2 minutes after drug administration,
direct laryngoscopy was performed and trachea was
intubated within 15 seconds by using an un-lubricated
7.5-8.5 mm internal diameter high volume/low pres-
sure tracheal tube (PVC, SUPA, Tehran, Iran) and cuff
was inflated with a volume of air to a maximum pres-
sure of 25 cm H2O. After tracheal intubation, periorbi-
tal area was prepared and draped. During this period,
head was elevated and the degree of head flexion was
estimated by goniometry and recorded. The lungs were
mechanically ventilated using a tidal volume of 8–10
mL/kg and the respiratory rate was adjusted to main-
tain normocarbia. Anaesthesia was maintained using
isoflurane oxygen and 50% nitrous oxide. Neuromus-
cular blockade was maintained using increments of
atracurium 0.15 mg/kg as required. Three-electrode
electrocardiogram (ECG) monitoring system was used
for the detection of arrhythmias during anaesthesia.
HR, SAP, DAP, IOP were measured at 1, 2, and 5 minutes after head flexion. If patients had arrhythmias after head and neck flexion, it was recorded and patient closely monitored for 5 min-
utes. Ineffective coughing movements (bucking) and wheezing were also noted after head flexion. Before
induction of general anaesthesia, intraocular pressure
was measured using a Schiotz Tonometry in both eyes in
supine position.

After completion of surgery, residual neuromuscular
block was reversed using neostigmine 0.04 mg/kg and
atropine 0.02 mg/kg. A blinded observer noted the
presence or absence of cough and stridor during emer-
gence of anaesthesia. Presence or absence of cough
was recorded as either “yes” or “no.” If cough was pre-
sent, it was graded using a three-category scale (Table
1).\textsuperscript{15}

Data are expressed as mean ± standard deviation (SD)
of the mean. Results were analyzed using one-sample
T-test, paired sample T-test, the Chi squared test and
repeated measure analysis of variance. A p value of
<0.05 was considered to represent statistical signifi-
cance. Statistical analyses were performed using the
SPSS 11.0 for Windows software application.

Table 1: Three- Category Scale for Scoring Cough on
Emergence

<table>
<thead>
<tr>
<th>Severity of Cough</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>Single Cough</td>
</tr>
</tbody>
</table>
| Moderate          | More than one episode of unsust-
                    |ained (≤ 5 s) coughing            |
| Severe            | Sustained (> 5 s) bouts of cough-
                    |ing                               |

RESULTS

The patients' demographic data, duration of surgery
and total opioid administration were summarized in
Table 2. Mean (SD) degree of head flexion was 36.4 ±
4.4. Mean SAP, DAP, IOP, and HR was increased sig-
nificantly after head flexion compared with baseline
values and returned to normal at 5 minutes (Table 3,
Fig 1). PETCO2 and SpaO2 were decreased signifi-
cantly at 1 and 2 minutes after head flexion compared
with baseline values and returned to normal at 5 min-
utes (Table 3). Incidence of arrhythmias, bucking and
wheezing after head flexion were summarized in Table
4. The incidence of coughing and stridor observed in
patients emerging from general anaesthesia was 5% and
2% respectively.

Table 2: Characteristic of patients, duration of surgery
and intraoperative fentanyl administration

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD or Proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>62.2 ± 12.6</td>
</tr>
<tr>
<td>Sex (Male/Female)</td>
<td>52/48</td>
</tr>
<tr>
<td>ASA (I/II)</td>
<td>82/18</td>
</tr>
<tr>
<td>Surgical time (min)</td>
<td>62.0 ± 14.0</td>
</tr>
</tbody>
</table>
| Intraoperative fentanyl dos-
  age (µg)                     | 137.0 ± 9.0             |

Discrete data are expressed as absolute numbers; Continuous
data are expressed as Mean ± SD.
Table 3: Cardiovascular, respiratory, and intraocular pressure changes after endotracheal intubation and head flexion.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Baseline</th>
<th>After ETI</th>
<th>1 min After Head Flexion</th>
<th>2 min After Head Flexion</th>
<th>5 min After Head Flexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAP (mmHg)</td>
<td>112.8 ± 11.1</td>
<td>117.2 ± 7.8</td>
<td>123.1 ± 9.7 *</td>
<td>117.8 ± 8.3 *</td>
<td>111.1 ± 7.1</td>
</tr>
<tr>
<td>DAP (mmHg)</td>
<td>65.2 ± 7.8</td>
<td>67.4 ± 6.7 *</td>
<td>71.0 ± 9.1 *</td>
<td>69.0 ± 7.4 *</td>
<td>65.6 ± 7.9</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>68.8 ± 10.8</td>
<td>72.2 ± 12.0 *</td>
<td>74.2 ± 13.7 *</td>
<td>72.6 ± 13.3 *</td>
<td>69.1 ± 12.6</td>
</tr>
<tr>
<td>PETCO2 (mmHg)</td>
<td>40.0 ± 2.0</td>
<td>36.3 ± 1.2 *</td>
<td>33.6 ± 3.0 *</td>
<td>34.3 ± 2.0 *</td>
<td>38.0 ± 6.5</td>
</tr>
<tr>
<td>SpO2 (%)</td>
<td>99.2 ± 4.7</td>
<td>97.3 ± 9.7</td>
<td>93.4 ± 6.3 *</td>
<td>95.3 ± 4 *</td>
<td>98.8 ± 1.2</td>
</tr>
<tr>
<td>IOP (mmHg)</td>
<td>14.3 ± 1.3</td>
<td>16.3 ± 1.4 **</td>
<td>14.9 ± 3.6 * *</td>
<td>16.0 ± 2.9 **</td>
<td>14.5 ± 2.4</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± SD. * Significant (P<0.001) vs. baseline. ** Significant (P<0.05) vs. baseline. HR: Heart rate, SAP: Systolic arterial pressure; DAP: Diastolic arterial pressure; IOP: Intraocular pressure; ETI: Endotracheal Intubation.

DISCUSSION

Our study showed that in patients undergoing cataract surgery during general anesthesia, endotracheal tube movement by changes in head and neck position had significant effect on heart rate, systolic and diastolic blood pressures, laryngeal reflexes, SpaO2, PETCO2, and intraocular pressure.

SAP, DAP, and HR increased after head flexion compared with baseline values. Incidence of arrhythmias after head flexion was 2-5%. These changes could be due to stimulation caused by laryngoscopy, endotracheal intubation and/or head flexion. Laryngoscopy and intubation are associated with tachycardia and a rise in...
Head flexion causes displacement of the endotracheal tube tip toward the carina. In one study, neck flexion caused 5.5 mm inward movement of the endotracheal tube. In another study, the mean extent of endotracheal tube displacement was 3.1 mm with neck flexion in low birth weight neonates. Displacement of the endotracheal tube caused by flexion of the neck was also investigated in 10 small children between the ages of 16 and 19 months by means of a fiberoptic bronchoscope. The endotracheal tube tip moved a mean distance of 0.9 cm toward the carina with flexion of the neck. Stimulation of the tracheal mucosa by endotracheal tube movement induces bradycardia, arrhythmias and hypotension. Bradycardia and arrhythmias are independent responses that occur rapidly and are not related to developing hypoxia or dependent on the respiratory responses. In our study, head flexion stimulated the tracheal mucosa and caused bradycardia and arrhythmias at 1 and 2 minutes after it.

Predominant response after laryngoscopy and intubation is sympathetic. In contrast, parasympathetic response is dominant following stimulation of tracheal mucosa by endotracheal tube movement. As fig 1 shows, increase in HR was more sustained than the increase in SAP. Increase in DAP was almost slight. So, it is hypothesized that head flexion after intubation probably causes biphasic cardiac response. At the first phase, direct laryngoscopy caused increased sympathetic response so that HR, SAP and DAP were increased above the baseline. At the second phase, head flexion caused parasympathetic stimulation and opposed sympathetic response following direct laryngoscopy.

In a recent trial by Kihara et al, both systolic and diastolic pressure increased after intubation for 2 minutes with highest values in the hypertensive group. In our study, head flexion was done about 6 min after endotracheal intubation. At this time, it is probable that sympathetic response to laryngoscopy has been attenuated. So, it seems that increase in SAP and DAP after head flexion was more due to stimulation of tracheal mucosa by endotracheal tube movement. Therefore, it is concluded that head flexion after intubation have both sympathomimetic and parasympathomimetic effect.

In this study, PETCO2 decreased significantly at 1 and 2 minutes after head flexion compared with baseline values since neck flexion caused displacement of the endotracheal tube tip toward the carina. SpaO2 also decreased significantly at 1 and 2 minutes after head flexion compared with baseline values. Wheezing and bucking after head flexion was presumably due to stimulation of the tracheal mucosa. Bronchospasm is triggered by mechanical stimulation, especially of the laryngotraceal area. Laryngeal and glottic stimulation may not only evoke varying degrees of laryngospasm, but if the stimulus is of sufficient intensity, bronchospasm may be induced as well. Bronchospasm and laryngospasm after stimulation of the tracheal mucosa by neck flexion caused decreased SpaO2.

The incidence of coughing and stridor observed in patients emerging from general anaesthesia was 5% and 2% respectively. The cough reflex is initiated chiefly by stimuli applied to the mucosa of the tracheobronchial tree. Endotracheal intubation is a common cause in anaesthetic practice for inciting cough or bucking. As patients emerge from general anaesthesia, the stimulating effect of positive pressure ventilation on the mechanosensitive receptors of the trachea and larger bronchi may provoke coughing. The occurrence of coughing and stridor on emergence from anesthesia could probably be due to stimuli applied to the mucosa of the tracheobronchial tree by tracheal tube or cuff after head flexion. Inoue and colleagues showed that endotracheal tube movement after head flexion causes significant increase in intracuff pressure (> 25 cm H2O) in some patients. In another study done by Rao et al, over-inflating the sealing cuff had caused a significant prolongation of the expiratory time during the first challenged breath in dogs.

The increased IOP after head flexion might be due to stimulation of tracheal mucosa by head flexion. The mechanism is not clear, but it probably relates to sympathethetic cardiovascular responses to head flexion or blockage of aqueous outflow by acute venous congestion. Any straining, bucking, breath holding or obstructed airway during the induction, maintenance, or emergence from general anesthesia will increase venous congestion in the ophthalmic veins and therefore raise IOP.

CONCLUSION
Our study showed that endotracheal tube movement by changes in head and neck position has significant effects on heart rate, systolic and diastolic blood pressures, laryngeal reflexes, SpaO2, PETCO2, and intraocular pressure in patients undergoing cataract sur-
gery during general anaesthesia. So, head flexion after endotracheal intubation can be hazardous in patients with coronary artery disease, glaucoma and perforating eye injuries where minimal changes in hemodynamic and intraocular pressure are detrimental.

REFERENCES