Original Article

Influence of head flexion on intraocular pressure, cardiovascular, and respiratory responses in patients undergoing cataract surgery after endotracheal intubation

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Background: In cataract surgery, the periorbital area is prepared and draped after induction of general anesthesia and endotracheal intubation (ETI). 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. Stimulation of the tracheal mucosa may cause bucking, increased intraocular pressure (IOP), laryngospasm and/or bronchospasm, during light anesthesia. Laryngeal constriction and all components of the tracheal response may affect end-tidal carbon dioxide pressure (PETCO2) and peripheral arterial hemoglobin oxygen saturation (SpaO2). Thus, in the current study, we investigated the influence of head and neck flexion on heart rate (HR), systolic and diastolic blood pressure (SAP and DAP), SpaO2, PETCO2, and IOP in patients undergoing cataract surgery with endotracheal intubation during general anesthesia.

Patients and Methods: The present prospective study comprised patients aged from 40 to 80 year with 106 American Society of Anesthesia (ASA) physical status I and II. Anesthesia was induced with thiopental sodium, lidocaine and fentanyl. Atracurium 0.5 mg/kg was administered to facilitate tracheal intubation. HR, SAP, DAP, SpaO2, PETCO2, and IOP were measured at 1, 2, and 5 minutes after head flexion.

Results: Mean SAP, DAP, IOP, and HR was increased after ETI and head flexion compared with baseline values. PETCO2 and SpaO2 were decreased after ETI and at 1, 2 minutes after head flexion compared with baseline values.

Conclusion: In patients undergoing cataract surgery during general anesthesia, endotracheal tube movement caused changes in head and neck position resulting in significant effects on heart rate, systolic and diastolic blood pressures, laryngeal reflexes, SpaO2, PETCO2, and intraocular pressure.

Keywords: 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 surgery is usually performed by extracapsular or intracapsular extraction technique under regional eye block or general anesthesia[1].

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After induction of general anesthesia and endotracheal intubation (ETI), 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. In one study, inward movement of the endotracheal tube (ETT) that resulted from neck flexion, shortened the distance (mean = 5.5 mm) between the ETT tip and the carina[2]. In another study, the mean ETT displacement

was 3.1 mm (SD±1.7 mm) caused by neck flexion in low birth weight (LBW) neonates[3]. Displacement of ETT 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 ETT tip moved a mean distance of 0.9 cm toward the carina with flexion of the neck[4]. Bradycardia ,arrhythmia and possibly hypotension occur as a result of stimulation of the tracheal mucosa by ETT displacement[5]. ETT movement by changes in head and neck position has caused significant increase in intracuff pressure and excessive pressure (> 25 cm H2O) developed in some patients with head and neck flexion[6]. Over-inflating the sealing cuff caused a significant prolongation of the expiratory period of the first challenged breath in dogs[7]. Stimulation of the tracheal mucosa caused laryngospasm and/or bronchospasm as well as unproductive cough (bucking) in light anesthesia. Ineffective coughing movements called bucking are also elicited. Apnea is usual if the patient is breathing spontaneously. This is frequently caused by tracheal intubation, but it is also seen on inflation of the endotracheal cuff[8]. Changes to depth of anesthesia can modify the laryngeal and respiratory responses to tracheal irritation. The close association of laryngeal and respiratory responses may be an inte gral part of the defensive reflex synergism[9]. Based on the above studies, the protective reflex responses to airway irritation seemed to be exaggerated immediately after endotracheal intubation and before initiation of surgical stimulation due to the inadequate depth of anesthesia.

Laryngoscopy and endotracheal intubation are the anesthesia-related procedures which most likely lead to significant increase in intraocular pressure (IOP) at least 10 to 20 mm Hg[10]. The mechanism is not clear, but it probably relates to sympathetic cardiovascular responses to tracheal intubation[11]. The most severe increase in IOP are usually caused by blockage of aqueous outflow by acute venous congestion. Any straining, bucking, breath holding or obstructed airway during the induction, maintenance, or emergence of general anesthesia will increase venous congestion in the ophthalmic veins and therefore raise IOP[12]. It is concluded that IOP may be increased by ETT movement during head and neck positioning due to inadequate depth of anesthesia immediately after endotracheal intubation and before initiation of surgical stimulation.

Laryngeal constriction and all components of the tracheal response, such as apnea, expiration reflex, cough reflex, and spasmodic panting caused by tracheal stimulation following ETT movement may affect end-tidal carbon dioxide pressure (PETCO2) and peripheral arterial hemoglobin oxygen saturation (SpaO2).

In the current study, we investigated the influence of head and neck flexion after endotracheal intubation on heart rate (HR), systolic and diastolic blood pressure (SAP and DAP), peripheral arterial hemoglobin oxygen saturation (SpaO2), PETCO2, wheezing, coughing, stridor, and IOP in patients undergoing cataract surgery under general anesthesia.

Patients and Methods

The present prospective study comprised 106 ASA physical status I and II patients aged from 40 to 80 years and selected by convenience sampling method. Having obtained the

approval of Institutional Ethics Committee and informed consent from each case, the patients underwent elective cataract surgery with duration of anesthesia between 40 and 70 minutes. Exclusion criteria were anticipated difficulty in tracheal intubation, direct laryngoscopy of more than 15 seconds[13], cigarette smoking, a history of respiratory disease or recent respiratory tract infection, cardiovascular disease, or IOP of more than 20 mmHg[14]. After establishing IV access, routine monitors and recording vital signs, patients were breathed with 100% oxygen. Anesthesia 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 followed after approximately 2 min by direct laryngoscopy. During maximum 15 seconds laryngoscopy, the trachea was intubated using an unlubricated 7.5-8.5 mm internal diameter high volume/low pressure ETT (PVC, SUPA, Tehran, Iran) and the cuff was inflated with a volume of air to maximum intracuff pressure 25 cm H2O. The lungs were mechanically ventilated using a tidal volume of 8–10 mL/kg and the respiratory rate was adjusted to maintain normocarbia. Anesthesia was maintained using isoflurane oxygen and 50% nitrous oxide. Neuromuscular blockade was sustained using increments of atracurium (0.15 mg/kg) as required. Three-electrode electrocardiogram (ECG) monitoring system was used for the detection of arrhythmias during anesthesia. Any arrhythmias occurring after head and neck flexion, was recorded and the patients were then closely monitored for 5 minutes. Head flexion was quantitated by goniometer and recorded, following head elevation. Any arrhythmias occurring after head and neck flexion, was recorded and the patients were then closely monitored for 5 minutes. Head flexion was quantitated by goniometer and recorded, following head elevation. HR, SAP, DAP, SpaO2, PETCO2, and IOP were measured at 1, 2, and 5 minutes after head flexion. Before induction of general anesthesia, intraocular pressure was measured using a Schiotz Tonometry in both eyes in supine position. After ETI, IOP was measured at 1, 2, and 5 minutes after head flexion by an ophthalmologist. Ineffective coughing movements (bucking) and wheezing were also noted after head flexion. Upon completion of the procedure, the oropharynx was suctioned, isoflurane was stopped, and the patient was administered 100% oxygen. Residual neuromuscular block was reversed using neostigmine and atropine. Patients were then placed in the recovery position, mechanical ventilation was discontinued, and ventilation was assisted until spontaneous ventilation resumed. The trachea was extubated when patients demonstrated the ability to follow verbal commands or demonstrated purposeful movement in addition to resumption of regular spontaneous respiration. A blinded observer noted the presence or absence of cough and stridor during emergence before and after extubation and before transfer to the postanesthetic care unit. Cough was recorded as either

Table 1 Three- Category Scale for Scoring Cough on Emergence

Severity	Definition
Mild	Single Cough
Moderate	More than one episode of unsustained (≤ 5 s) coughing
Severe	Sustained (> 5 s) bouts of coughing

Table 2. Characteristic of patients

Factor	Mean ±SD
Age (years)	62.2 ± 12.6
Surgical time (min)	62.0 ± 14.0
Intraoperative fentanyl dosage (μg)	137.0 ± 9.0
Sex (Male/Female)	52 (52%)/ 48 (48%)
ASA (I/II)	82 (82%)/ 18 (18%)

Discrete data are expressed as absolute numbers and percentages; Continuous data are expressed as mean ± SD.

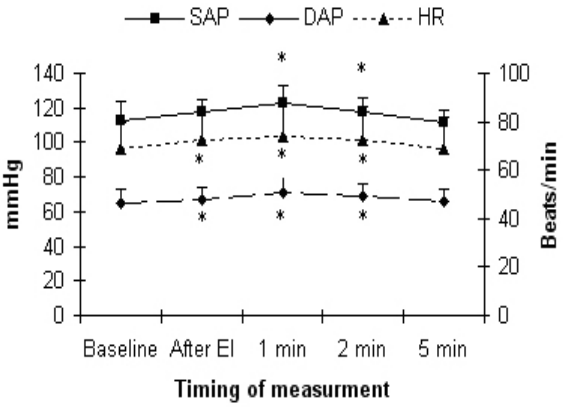

Figure 1 The hemodynamic changes during intubation period in study patients. Data are mean ± SD. SAP= systolic arterial pressure; DAP=diastolic arterial pressure; HR=heart rate; EI=endotracheal intubation. *P <0.05 vs. baseline.

"yes" or "no." If cough was present, it was graded using a three-category scale (Table 1)[15]. Data were summarized as means and standard deviations and analyzed using appropriate tests including Chi-Square and Kruskall-Wallis tests using SPSS 11.0 statistical analysis software. A P-value <0.05 was considered statistically significant.

Results

A total of 106 patients participated in the study and underwent elective cataract surgery. Of these, 6 patients were excluded: One patient had unpredictable difficult intubations requiring multiple laryngoscopies before successful tracheal intubation, two patients received an intraoperative opioid infusion, which violated the study protocol, two patients received anesthetics between 90 and 120 min in duration, and one case was lost because of cancellation of surgery. The patients' demographic data, case duration and total opioid usage were summarized in Table 2. Mean SAP, DAP, IOP, and HR was increased significantly after ETI and head flexion compared with baseline values (P < 0.05) (see Table 3, Fig 1). PETCO2 and SpaO2 were decreased significantly after ETI and at 1, 2 minutes after head flexion compared with baseline values (P < 0.05) (Table 3). Incidence of arrhythmias,

Table 3 Cardiovascular, respiratory, and intraocular pressure changes after endotracheal intubation and head flexion.

Variables	Baseline	After ETI	After Head Flexion		
			1 min	2 min	5 min
SAP (mmHg)	112.8 ± 11.1	117.2 ± 7.8	123.1 ± 9.7 *	117.8 ± 8.3 *	111.1± 7.1
DAP (mmHg)	65.2 ± 7.8	67.4 ± 6.7 *	71.0 ± 9.1 *	69.0 ± 7.4	65.6 ± 7.9
HR (beats/min)	68.8 ± 10.8	72.2 ± 12 *	74.2 ±13.7*	72.6 ±13.3*	69.1 ±12.6
PETCO2 (mmHg)	40.0 ± 2.0	36.3 ± 1.2 *	33.6 ± 3.0 *	34.3 ± 2.0	38.0 ± 6.5
SpO2 (%)	99.2 ± 4.7	97.3 ± 9.7	93.4 ± 6.3 *	95.3 ± 4 *	98.8 ± 1.2
IOP (mmHg)	14.3 ± 1.3	16.3 ± 1.4 **	14.9 ± 3.6 * *	16.0 ± 2.9 **	14.5 ± 2.4

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.

Table 4 Incidence of arrhythmias, bucking, and wheezing at 1, 2 and 5 minutes after head flexion

Variables	After head flexion		
	1 min	2 min	5 min
Arrhythmias	5	2	0
Bucking	4	2	0
Wheezing	5	3	0

Values are expressed as percentages

bucking and wheezing after head flexion were summarized in Table 4. The incidence of coughing and stridor observed in patients emerging from general anesthesia (or recovery) was 5% and 2% respectively.

Discussion

Our study showed that in patients underwent 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 and ETI and/or head flexion. Laryngoscopy and ETI are associated with tachycardia and hypertension[16]. These changes have been observed to be associated with rise in plasma noradrenaline levels, confirming a predominantly sympathetic response to it[17].

Head flexion causes displacement of the endotracheal tube tip toward the carina[2]. From stimulation of the tracheal mucosa by ETT displacement, bradycardia and arrythmias occur, and hypotension may result. Bradycardia and arrhythmias are independent responses that occur rapidly and are not related to developing hypoxia or dependent on the respiratory responses[5].

Predominant response after laryngoscopy and ETI is sympathetic[16]. In contrast, parasympathetic response is predominant following stimulation of tracheal mucosa by endotracheal tube movement[5]. As fig 1 shows, increase in HR was more sustained than increase in SAP. Increase in DAP was almost slight. So, it is hypothesized that head flexion after ETI probably causes biphasic cardiac response.

In a recent trial by Kihara et al[18], both systolic and diastolic pressure increased after intubation for 2 minutes with highest values in the hypertensive group receiving direct laryngoscopy[19]. 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. So it is concluded that head flexion after ETI has both sympathomimetic and parasympathomimetic effect.

PETCO2 decreased significantly at 1, 2 minutes after head flexion compared with baseline values. Neck flexion causes displacement of the endotracheal tube tip toward the carina[2]. A sudden drop in PETCO2 reading could indicate displacement of the ETT toward a main bronchus[20, 21].

SpaO2 also decreased significantly at 1, 2 minutes after head flexion compared with baseline values. Wheezing and bucking occurred after

head flexion till 2 min presumably due to stimulation of the tracheal mucosa[8, 22].

Bronchospasm is triggered by mechanical stimulation, especially of the laryngotacheal area. Laryngeal and glottic stimulation may not only evoke varying degrees of laryngospasm, but if the stimulus is of sufficient intenseity, bronchospasm may be induced[23].

The incidence of coughing and stridor observed in patients emerging from general anesthesia (or recovery) 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 anesthetic practice for inciting of cough or bucking[24].

As patients emerge from general anesthesia, the stimulating effect of positive pressure ventilation on the mechanosensitive receptors of the trachea and larger bronchi may provoke coughing[25].

The occurrence of coughing and stridor on emergence from anesthesia could probably be due to stimuli applied to the mucosa of the tracheaobronchial tree by tracheal tube or cuff after head flexion. But it needs documentation in future studies.

The increase of mean IOP at 1, 2 minutes after head flexion compared with baseline may be due to stimulation of tracheal mucosa after head flexion. The mechanism is not clear, but it probably relates to sympathetic cardiovascular responses to head flexion[11] or blockage of aqueous outflow by acute venous congestion[12]. Due to inadequate depth of anesthesia immediately after endotracheal intubation and before initiation of surgical stimulation, it is concluded that IOP may increase by ETT movement during head and neck positioning.

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 underwent cataract surgery during general anesthesia. So, head flexion after ETI could be hazardous in situations where minimal changes in homodynamic and intraocular pressure are desirable like patients with coronary artery disease, glaucoma and possibly in patients with perforating eye injuries.

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