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 (SpaO2) 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), SpaO2, PETCO2 and IOP in patients undergoing cataract surgery during general anesthesia.

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, SpaO2, 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 SpaO2 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, SpaO2, 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.[1] 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. [2-4]

Stimulation of the tracheal mucosa by endotracheal tube displacement induces bradycardia, arrhythmias, hypotension, laryngospasm, bronchospasm, coughing, and apnoea will occur in light anesthesia.[5-8] Inflation of the endotracheal cuff also induces above signs.[8] Changes in depth of anaesthesia can modify the laryngeal and respiratory responses to tracheal irritation. [9]

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 .[10] The mechanism is not clear, but it probably relates to sympathetic cardiovascular responses to tracheal intubation .[11-12] 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 (SpaO2). 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), SpaO2, 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 prolonged duration of direct laryngoscopy may affect the magnitude of circulatory stimulation associated with tracheal intubation.[13]

After establishing intravenous access and routine monitors, patients were ventilated with 100% oxygen. Anaesthesia 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 pressure tracheal tube (PVC, SUPA, Tehran, Iran) and cuff was inflated with a volume of air to a maximum pressure of 25 cm H2O. After tracheal intubation, periorbital 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 maintain normocarbia. Anaesthesia was maintained using isoflurane oxygen and 50% nitrous oxide. Neuromuscular 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, SpaO2, PETCO2, and 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 minutes. 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 emergence of anaesthesia. Presence or absence of cough was recorded as either “yes” or “no.” If cough was present, it was graded using a three-category scale (Table 1).[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 significance. Statistical analyses were performed using the SPSS 11.0 for Windows software application.

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

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

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 significantly after head flexion compared with baseline values and returned to normal at 5 minutes (Table 3, Fig 1). PETCO2 and SpaO2 were decreased significantly at 1 and 2 minutes after head flexion compared with baseline values and returned to normal at 5 minutes (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

Variable	**Mean ± SD or Proportion**
Age (years)	62.2 ± 12.6
Sex (Male/Female)	52/48
ASA (I/II)	82/18
Surgical time (min)	62.0 ± 14.0
Intraoperative fentanyl dosage (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.

Variables	Baseline	After ETI	1min After Head Flexion	2 min After Head Flexion	5 min After Head Flexion
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.

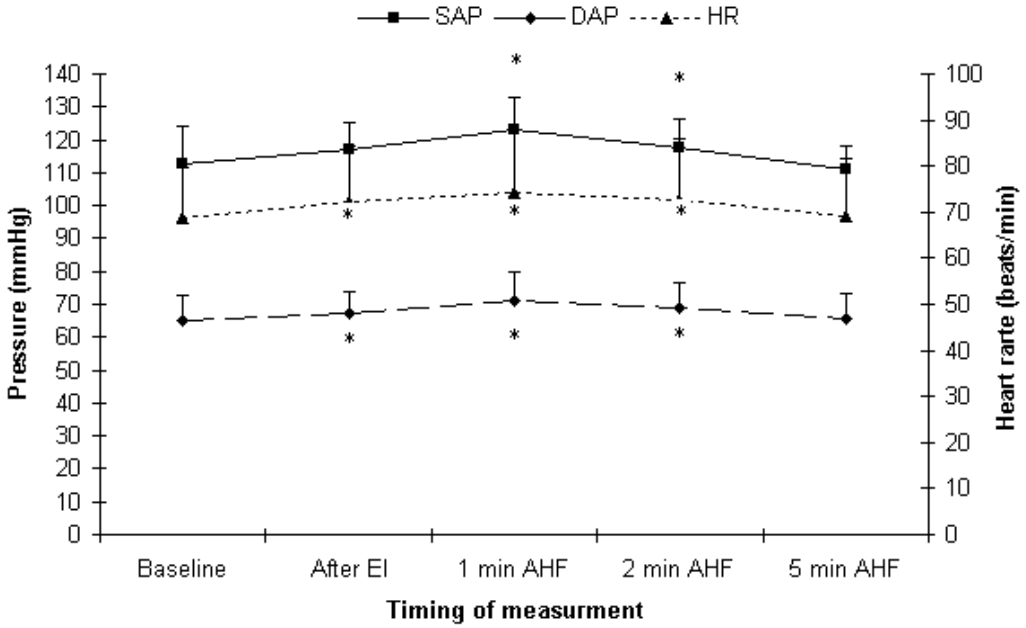

Figure 1 The cardiovascular changes during intubation period and after head flexion in study patients. Data are expressed as mean ± SD. SAP= systolic arterial pressure; DAP=diastolic arterial pressure; HR=heart rate; EI= endotracheal intubation. AHF = after head flexion. * P <0.05 vs. baseline.

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

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

Values are expressed as percentages

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 intubatin and/or head flexion. Laryngoscopy and intubation are associated with tachycardia and a rise in

blood pressure.[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] In one study, neck flexion caused 5.5 m inward movement of the endotracheal tube.[2] In another study, the mean extent of endotracheal tube displacement was 3.1 mm with neck flexion in low birth weight neonates.[3] 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.[4]

Stimulation of the tracheal mucosa by endotracheal tube displacement 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.[5] 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.[16] In contrast, parasympathetic response is dominant following stimulation of tracheal mucosa by endotracheal tube movement.[5] 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 apposed sympathetic response following direct laryngoscopy.

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 .[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. 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.[2, 20-21] 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.[8, 22]

Bronchospasm is triggered by mechanical stimulation, especially of the laryngotracheal 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 .[23] 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 anesthetic practice for inciting cough or bucking.[24] 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.[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. Inoue and colleagues[6] 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[7], over-inflating the sealing cuff had caused a significant prolongation of the expiratory time during the first challenged breath in dogs.[7]

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 sympathetic cardiovascular responses to head flexion[11] or blockage of aqueous outflow by acute venous congestion.[12] 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.[12]

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

1. McGoldrick KE: Anesthesia and the eye. In: Barash PG, Cullen BF, Stoelting RK. Clinical Anesthesia. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 500-504.
2. Yap SJ, Morris RW, Pybus DA. Alterations in endotracheal tube position during general anaesthesia. *Anaesth Intensive Care* 1994;22(5):586-8.
3. Rost JR, Frush DP, Auten RL. Effect of neck position on endotracheal tube location in low birth weight infants. *Pediatr Pulmonol*. 1999; 27(3):199-202.
4. Sugiyama K, Yokoyama K. Displacement of the endotracheal tube caused by change of head position in pediatric anesthesia: evaluation by fiberoptic bronchoscopy. *Anesth Analg* 1996; 82(2):251-3.
5. Pott M, Habler O, Meininger D. Unexpected hemodynamic depression after induction of anaesthesia. *Anasthesiol Intensivmed Notfallmed Schmerzther* 2006; 41(10):636-8.
6. Inoue S, Takauchi Y, Kuro M, Ninaga H. Effects of changes in head and neck position on a tracheal tube cuff. *Masui* 1998; 47(9):1069-72.
7. Rao SV, Sant'Ambrogio FB, Sant'Ambrogio G. Respiratory reflexes evoked by tracheal distension. *J Appl Physiol* 1981; 50(2):421-7.
8. Collins VJ.Autonomic reflexes during anesthesia and surgery .In: Principales of Anesthesiology General and Regional Anesthesia, Vincent J .Collins , 3rd ed. Philadelphia: Lea and Febiger; 1993. p. 1186-7
9. Nishino T, Hiraga K, Yokokawa N. Laryngeal and respiratory responses to tracheal irritation at different depths of enflurane anesthesia in humans. *Anesthesiology* 1990 ; 73(1):46-51.
10. Donlon JV . Anesthesia for ophthalmic surgery. In: Barash P (ed): ASA Refresher Course Lectures. Philadelphia: JB Lippincott; 1998. p. 81.
11. Donlon JV. Anaesthesia for Eye, Ear, Nose, and Throat Surgery In: Miller RD. Anesthesia. 5th ed. Philadelphia: Churchill Livingstone; 2000. p. 2178.
12. Stead SW, Beatie CD, Keyes MA. Anesthesia for Ophthalmic Surgery. In: Longnecker DE, Tinker JH, Morgan GE. Principles and Practice of Anesthesiology. 2nd ed. Mosby; 1998. p. 2184
13. Stoeling RK . Ischemic heart disease. In: Dierdorf SF, Anesthesia and co-existing disease 4 th edition . Philadelphia, Pennsylvania: Churchill Livingstone; 2002. p. 15
14. Donlon JV. Anesthesia for Eye, Ear, Nose, and Throat Surgery. In: Miller RD. Anesthesia. 5th ed. Philadelphia: Churchill Livingstone; 2000. p. 2176.
15. Minogue, Sean C. FCARCSI; Ralph, James FRCA; Lampa, Martin J. FRCPC. Laryngotracheal topicalization with lidocaine before intubation decreases the incidence of coughing on emergence from general anesthesia. *Anesth Analg* 2004; 99(4):1253-7.
16. Choi SU, Lim CH, Lee SH, et al. Thoracic epidural clonidine attenuates haemodynamic responses induced by endobronchial intubation. *J Int Med Res* 2006; 34(6):565-72.
17. Kavhan Z, Aldemir D, Mutlu H. Which is responsible for the haemodynamic response due to laryngoscopy and endotracheal intubation? Catecholamines, vasopressin or angiotensin? *Eur J Anaesthesiol* 2005; 22(10):780-5.
18. S. Kihara, J. Brimacombe, Y. Yaguchi et al., Hemodynamic responses among three tracheal intubation devices in normotensive and hypertensive patients. *Anesth Analg* 2003; 96: 890–895.
19. Kahl M, Eberhart LH, Behnke H, et al. Stress response to tracheal intubation in patients undergoing coronary artery surgery: direct laryngoscopy versus an intubating laryngeal mask airway. *J Cardiothorac Vasc Anesth* 2004 Jun;18(3):275-80.
20. Detach bag from endotracheal tube and insert sterile tip of suction. A sudden drop in EtCO2 reading could indicate displacement of the endotracheal tube. Procedure Protocols Airway Management: General Principles, Section III. http://www.health.state.ok.us/program/ems/omtp/Section%20 III.doc
21. Marvin A. Wayne. End Tidal CO2: From Airway to Cardiac Output. Spring. 2003; May 15. p.3 http://www.itaccs.com/traumacare/archive/spring_03/program.pdf
22. Collins VJ. .Autonomic reflexes during anesthesia and surgery .In: Principales of Anesthesiology General and Regional Anesthesia, Vincent J .Collins , 3rd ed. Philadelphia: Lea and Febiger; 1993. p. 1185
23. Collins VJ. .Autonomic reflexes during anesthesia and surgery .In: Principales of Anesthesiology General and Regional Anesthesia , Vincent J .Collins , 3rd ed. Philadelphia: Lea and Febiger; 1993. p. 1188.
24. Russell WJ, Morris RG, Frewin DB, Drew SE. Changes in plasma cathecolamine concentrations during endotracheal intubation. *Br J Anesth* 1981; 53:837-9
25. Sant'Ambrogio G, Widdicombe J. Reflexes from airway rapidly adapting receptors. *Respir Physiol* 2001;125:33–45.