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Influence of hypercapnia on cardiovascular responses to tracheal intubation

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Abstract

Background: Laryngoscopy and tracheal intubation are often associated with tachycardia, hypertension, and arrhythmias. There is a risk of hypercapnia in the case of difficult mask ventilation. The circulatory response to hypercapnia is increases in arterial pressure and heart rate. We evaluated the difference of cardiovascular responses to tracheal intubation between normocapnia and hypercapnia during mask ventilation before tracheal intubation.

Methods: We studied 40 ASA physical status I to II patients under general anesthesia. Induction of anesthesia was achieved with midazolam 0.05 mg/kg, propofol 1.5 mg/kg, alfentanil 10 μg/kg, and rocuronium 0.6 mg/kg IV. The lungs were mechanically ventilated with a tidal volume of 10 mL/kg and 6 to 10 bpm in the hypercapnia group (n = 20) or 12 to 15 bpm in the normocapnia group (n = 20) during the induction period. Intubation was performed 3 minutes after the induction, and anesthesia was maintained using 1.5% sevoflurane (inspired) and 75% N_2O in oxygen. Heart rate, systolic arterial pressure (SAP), and diastolic arterial pressure were recorded every minute throughout the study.

Results: The proportion of the patients whose increase of SAP between just before intubation and 1 minute after intubation was more than 30 mm Hg in the hypercapnia group (40%) was greater than that in the normocapnia group (9.5%) (P = .0325). There were no differences in heart rate and diastolic arterial pressure between hypercapnia and normocapnia groups. For the SAP of the patients, the trend of changes was increased (P = .024).

Conclusions: Hypercapnia during mask ventilation before tracheal intubation may exaggerate the increase of SAP during intubation compared to normocapnia. Ventilation was important in minimizing hemodynamic responses during induction regardless of using drugs.

1. Introduction

Laryngoscopy and tracheal intubation are often associated with tachycardia, hypertension, and arrhythmias [1]. The hemodynamic responses to tracheal intubation are known to be caused, in part, by a reflex sympathetic

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discharge [2,3]. Any factors that can exacerbate these responses should be prohibited if possible. The incidence of difficult mask ventilation has been reported from 1.4% to 5% [4-6]. There is a risk of hypercapnia in the case of difficult mask ventilation. The circulatory response to hypercapnia is also increases in heart rate (HR) and cardiac output through stimulation of sympathetic nervous system [7,8]. However, changes in systolic and diastolic arterial blood pressure were not well known.

We evaluated the difference of cardiovascular responses to tracheal intubation between normocapnia and hypercapnia groups during mask ventilation before tracheal intubation.

2. Methods

The study was approved by the Institutional Review Board of the Kyung Hee University Hospital.

We studied 40 American Society of Anesthesiologists (ASA) physical status I to II patients aged 20 to 65 years presenting for routine elective surgery under general anesthesia. Patients were excluded if they had severe ischemic heart disease, congestive heart failure, cerebrovascular disease, diabetes mellitus, chronic obstructive pulmonary disease, or other disorders known to affect autonomic function. None of the patients were taking medications that affect cardiovascular function including antihypertensive agents. Patients were randomized to 2 groups using the sealed-envelope technique: normocapnia and hypercapnia. After obtaining written and informed consent, all patients had an intravenous catheter placed and were hydrated with 5 mL/kg crystalloid. Routine monitors were used. Each patient lay supine on the operating table and breathed oxygen for 3 minutes. Induction of anesthesia was achieved with midazolam 0.05 mg/kg, propofol 1.5 mg/kg, and alfentanil 10 μg/kg IV. Muscle relaxation was obtained with rocuronium 0.6 mg/kg IV. After breathing 100% oxygen, patients were ventilated with 100% oxygen through an anesthesia face mask connected to a semiclosed anesthesia circuit. The lungs were mechanically ventilated using an anesthesia machine (Primus, Dräger Medizintechnik GmbH, Lubeck, Germany) with a tidal volume of 10 mL/kg and 6 to 10 bpm in the hypercapnia group ($EtCO_2$ = 45 mm Hg, expecting $PaCO_2$ around 50 mm Hg) or 12 to 15 bpm in the normocapnia group ($EtCO_2$ = 35 mm Hg, expecting $PaCO_2$ around 40 mm Hg) [9]. Three minutes after the induction, when neuromuscular block was achieved, intubation was performed, and anesthesia was maintained using 1.5% sevoflurane (inspired) and 75% N_2O in oxygen. The fresh gas flow rate was adjusted to 3 L/min before and after the intubation. The lungs were mechanically ventilated to maintain an $EtCO_2$ around 35 mm Hg in both groups after intubation. Electrocardiograms and oxygen saturation were monitored continuously. Heart rate, systolic arterial pressure (SAP), and diastolic arterial pressure (DAP) were recorded every minute throughout the study. The baseline values were determined 1 minute before the induction of anesthesia. The number of the patients whose difference of systolic arterial pressures between just before intubation and 1 minute after intubation (ΔSAP) was more than 30 mm Hg (arbitrarily defined) in each group was checked. Arterial blood samples were taken just before the intubation and analyzed immediately (Rapidlab 1265, Bayer Diagnostics, Sudbury, UK). Time to intubation was measured from the insertion of blade of laryngoscope to the connection of intubated tracheal tube with anesthesia circuit. External tracheal manipulation, posterior displacement of the larynx by putting backward, upward, and lateral (slightly to the right) pressure on the thyroid or cricoid cartilage [10], was performed. However, it was done in minimal fashion in order not to aggravate cardiovascular response by the manipulation itself. The number of patients who were given external laryngeal manipulation for intubation in each group was checked.

The face mask fixing, intubation, and evaluation of Cormack grade were independently conducted by an anesthesiologist who was not fully informed about the purpose and process of this study and blinded to the anesthesia machine and the monitor, and modulating the anesthesia machine and the data recording were done by an investigator.

Escape medication (ephedrine 3-mg increments) was prepared to be administered for hypotension (SAP <80 mm Hg, or a decrease of >30% of baseline for >60 seconds); atropine, in 300-μg increments, for bradycardia (HR <45 bpm); and esmolol, in 30-mg increments, for hypertension (SAP >200 mm Hg, or an increase of >30% above baseline values, for >60 seconds) or tachycardia (HR >130 bpm for >60 seconds).

Power analysis, based on the past literature [11], suggested that 20 patients per group would be sufficient to detect a difference between groups of 15 mm Hg with respect to the cardiovascular response to intubation for a type I error of 0.05 and a power of 0.8.

All results are expressed as mean ± SD. Categorical variables were analyzed using Fisher exact test and continuous variables using *t* test or Wilcoxon rank sum test. Heart rate,

Table 1 Baseline characteristics

	Normocapnia (n = 20)	Hypercapnia (n = 20)
Sex (M/F)	10/10	11/9
Age (y)	43 (12)	42 (13)
Height (cm)	163 (8)	165 (10)
Weight (kg)	63 (10)	65 (11)
Time to intubation (s)	5.3 (1.3)	6.0 (2.1)
No of external laryngeal manipulation	3	4
Cormack grade (1/2/3/4)	9/7/4/0	8/6/4/2
pH	7.41 (0.03)	7.37 (0.02)
$PaCO_2$ (mm Hg)	40 (3)	49 (3)
PaO_2 (mm Hg)	484 (90)	475 (98)

Data are expressed as mean (SD) or numbers of patients.