Control of haemodynamic response to tracheal intubation in cigarette smokers compared with non-smokers

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Abstract. – Some authors have demonstrated that a bolus dose of 1 µg/kg followed by an infusion rate of 0.5 µg/kg/min is adequate to attenuate the haemodynamic response to laringoscopy and tracheal intubation. In this study we have evaluated the efficacy of Remifentanil in controlling haemodynamic and some neuroendocrine responses to tracheal intubation in smokers compared with non-smokers.

We studied 126 patients, ASA I-II, aged 20-49 yr, submitted laparoscopic cholecystectomy (66 male, 60 female); sixty-three patients were non-smokers and 63 patients smoked 10 or more cigarettes per day. Anaesthesia was induced with thiopental 3-5 mg/kg and remifentanil 1 µg/kg. Vecuronium 0.1 mg/kg was administrated to facilitate tracheal intubation. Immediately after intubation heart rate of smokers (mean 101.2 ± 17 beat/min) was significantly higher (p < 0.001) than non-smokers (mean 90.2 ± 14 beat/min) and also the neuroendocrine responses of smokers (epinephrine value 155 ± 173 pg/ml; norepinephrine value 276 ± 164 pg/ml) was significantly higher (p < 0.01) than non-smokers (epinephrine 95 ± 75, norepinephrine 154 ± 76).

These findings may be clinically important to evaluate the risk of ischaemic heart diseases.

Key Words: Intubation tracheal, Remifentanil, Anaesthesia, Cardiovascular system, Effects, Smokers, Complications.

Introduction

Remifentanil, a new esterase metabolized opioid, provides analgesia of rapid onset and short duration. Some authors have demonstrated that a bolus dose of 1 µg/kg followed by an infusion rate of 0.5 µg/kg/min is adequate to attenuate the haemodynamic response to laringoscopy and tracheal intubation. In this study we have evaluated the efficacy of Remifentanil in controlling haemodynamic and some neuroendocrine responses to tracheal intubation in smokers compared with non-smokers.

Methods

The study was approved by the clinical research ethics committee. Written informed consent was obtained from all patients.

We studied 126 patients, ASA I-II, aged 20-49 yr, submitted laparoscopic cholecystectomy (66 male, 60 female). Sixty-three patients were non-smokers and 63 patients smoked 10 or more cigarettes per day. Preoperative electrocardiogram, full blood counts and renal and liver function test were performed. The patients were premedicated with diazepam (0.02 mg/kg) and from midnight before surgery smoking was not allowed. In the operating room, routine monitoring was applied. Arterial pressure and heart rate were recorded noninvasively every 2 minutes and at standard times (baseline, pre-induction and post-intubation) venous blood samples were taken for epinephrine and norepinephrine determination by spectrophotometry.

Anaesthesia was induced with remifentanil 1 µg/kg and thiopental 3-5 mg/kg until the eyelash reflex has been abolished, when vecuronium 0.1 mg/kg was given to facilitate the intubation. The patient’s lungs were ventilated with a mixture air/oxygen (FiO₂ 50%) and end-tidal 1.1% sevoflurane. The anaesthesia was also maintained with a remifentanil mean
infusion of 0.5 \( \mu \text{g/kg/min} \). Perioperative analgesia was provided by IV ketorolac 60 mg and antiemetic therapy was made by ondansetron 8 mg.

Haemodynamic (measurements of systolic, diastolic and mean arterial pressure, heart rate and oxygen saturation) and neuroendocrine responses (epinephrine and norepinephrine) were determined at standard times: baseline (T1), before intubation (T2), post intubation (T3). The haemodynamic and neuroendocrine measurements were recorded by a second investigator who was unaware of the patient’s smoking status.

### Data Analysis

Statistical analysis over results were performed by ANOVA and Student’s t test. Statistical significance was defined as \( p < 0.05 \).

### Results

Haemodynamic (blood pressure and heart rate) and neuroendocrine (epinephrine and norepinephrine) data are shown in Figures 1 and 2.

Immediately after intubation heart rate of smokers (mean 101.2 ± 17 beat/min) was significantly greater (\( p < 0.001 \)) than that of non-smokers (mean 90.2 ± 14 beat/min). Heart rate in smokers was significantly greater than that in non smokers both at intubation (\( p < 0.01 \)) and at 5 minute after intubation (\( p < 0.05 \)), but smokers had significantly lower heart rates before induction (\( p < 0.01 \)). In addition, smokers had significantly lower systolic (mean 112.8 ± 10.9 mmHg) and mean arterial pressures (83.5 ± 8.9 mmHg) before induction than non-smokers (123.1 ± 12.5 mmHg, and 90 ± 9.9 mmHg respectively). Mean systolic arterial pressures in smokers and non-smokers before intubation were 101.8 ± 1.7 mmHg and 104.9 ± 9.1 mmHg, respectively, increasing to 130 ± 19 mmHg and 124.1 ± 18.5 mmHg on intubation; there was no significant difference between groups. Similarly, diastolic and mean arterial pressures increased in both groups on intubation (ns).

Immediately after intubation the neuroendocrine responses of smokers (epinephrine value mean 155 ± 173; norepinephrine value mean 276 ± 164) was significantly greater (\( p < 0.01 \)) than that of non-smokers (epinephrine value 95 ± 75, norepinephrine value mean 154 ± 76).

There were no significant differences between the two groups in age, weight, intubation condition score or doses of drugs used, and there was no movement or cough in any patient on intubation.

### Discussion

King in 1951\(^3\) described the reflex circulatory responses to laringoscopy and tracheal intubation. These comprise a transient increase in arterial pressure and heart rate. In this study, we have demonstrated a heightened haemodynamic and neuroendocrine response to laryngoscopy and intubation in smokers. Nicotine is known to act through the sympathoadrenergic system, causing increases in heart rate and vasoconstriction. The half-time of nicotine is variably reported from 30 min to 2.5 h; acute abstinence is followed by a reduction in heart rate and arterial pressure and decreased cathecolamine concentration\(^1-3\).

All of our smokers abstained from cigarettes for at least 6 hours before induction. This may explain why our smoking group had significantly lower heart rates, and systolic and mean arterial pressures than the non-smoking group, immediately before induction. However, it would also reduce the likelihood of nicotine playing a significant role in the greater increase in heart rate seen in the smoking group.

It has been suggested that because smoking induces chronic changes in the characteristic of the upper airway epithelium, there is greater exposure of subepithelial upper airway receptors to stimuli\(^1-2\). Mechanical stimulation of the upper airway during laryngoscopy and/or intubation may, by a similar mechanism, cause greater haemodynamic changes in smokers. Our study have shown that cigarette smokers exhibited a greater tachycardic response to intubation compared with non-smokers. Some studies\(^4-5\) have
Figure 1. Heart rate and blood pressure variations of smokers and non smokers. T1 = baseline; T2 = before intubation; T3 = after intubation.
demonstrated that most myocardial ischaemic episodes during anaesthesia are associated with intubation and surgical stimulation, especially if tachycardia occurs. Although the difference in heart rate between the groups is unlikely to be clinically relevant, smokers also have acute unfavorable effects on cardiopulmonary function caused by carbon monoxide- and nicotine-mediated changes in oxygen delivery and myocardial oxygen balance. A's smokers are at a greater risk of ischaemic heart disease, they may form a group in whom attempts to blunt the cardiovascular response at intubation may be particularly beneficial. Although this difference with O'Hare series may be related to the smoker status of his population. At last, it is likely that a more dose of Remifentanil is required at the induction of smokers patients if we will completely blunt the neuroendocrine responses (epinephrine and norepinephrine). Furthermore it is reasonable to consider the smoking habits of the patients enrolled in studies evaluating the haemodynamic responses to intubation.

References


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