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Haemodynamic changes after tracheal intubation in cigarette smokers compared with non-smokers

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We have studied the haemodynamic changes in response to tracheal intubation in 60 ASA I women undergoing elective gynaecological surgery; 30 women smoked more than 10 cigarettes a day, while the other 30 were non-smokers. Immediately after intubation, heart rate of smokers (mean 102.0 (SD 17) beat min^{-1}) was significantly greater ($P<0.01$) than that of non-smokers (mean 91.0 (12.3) beat min^{-1}). This may be clinically important in smokers who are at risk of ischaemic heart disease and also relevant for future studies investigating haemodynamic responses to intubation.

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Smokers have been shown to exhibit heightened upper airway reflex responses to chemical stimulation.[1] However, it is not known if smokers have an accentuated response to mechanical stimulation of the upper airway.

The haemodynamic changes associated with laryngoscopy and intubation have been studied extensively and many pharmacological methods have been used to obtund them, but to our knowledge, the effect of smoking *per se* has not been considered. Few, if any, investigators comment on the smoking status of their subjects. Although in the majority of patients undergoing anaesthesia these responses are probably of little consequence, they may be relevant in patients with cardiovascular and cerebrovascular disease. Thus the aim of this study was to ascertain if the cardiovascular changes associated with tracheal intubation were different in smokers compared with non-smokers.

Methods and results

After obtaining approval from the Local Ethics Committee, we studied 60 ASA I patients, aged 20–49 yr, who were normotensive and receiving no medications and were undergoing elective gynaecological surgery. Thirty patients were non-smokers and 30 patients smoked 10 or more cigarettes per day. Patients with a history of acid reflux and those whose body weight exceeded 15% of predicted values were excluded.

Two hours before operation, patients were premedicated with temazepam 20 mg orally; after premedication, smoking was not allowed. On arrival in the anaesthetic room, a 20-gauge i.v. cannula was inserted, and monitoring, in the form of ECG, pulse oximetry and non-invasive arterial pressure (Dinamap 1846SX), was commenced. After a 4-min stabilization period, baseline, pre-induction measurements of arterial pressure and heart rate were recorded.

Anaesthesia was induced with fentanyl 1 μg kg^{-1} and thiopental 4–6 mg kg^{-1} until the eyelash reflex had been abolished, when vecuronium 0.08–0.09 mg kg^{-1} was given. The patient's lungs were ventilated with 1% isoflurane and 66% nitrous oxide in oxygen via a Bain circuit and a face mask, with a fresh gas flow of 100 ml kg^{-1} min^{-1} and a ventilatory frequency of 12–15 bpm. The patient's arterial pressure was measured every minute, and after the reading at 3 min after induction, the trachea was intubated. The interval between the onset of laryngoscopy and completion of tracheal intubation was timed, and the intubating conditions graded according to an intubating condition score.

Measurements of systolic, diastolic and mean arterial pressure, heart rate and oxygen saturation were recorded at intubation, 45 s after intubation, and at 30-s intervals thereafter for a period of 3 min; any patient in whom the non-invasive arterial pressure monitor had failed to cycle within the specified time interval was omitted from the study. These measurements were recorded by a second investigator; neither the person administering anaesthesia nor the second investigator was aware of the patient's smoking status.

Patient characteristics were compared using the Student's *t* test, and measurements of heart rate, systolic and diastolic pressures were analysed using repeated measures analysis of variance. Statistical significance was defined as $P<0.05$.

Heart rate in smokers was significantly greater than that in non smokers both at intubation ($P<0.01$) and at 45 s after intubation ($P<0.05$), but smokers had significantly

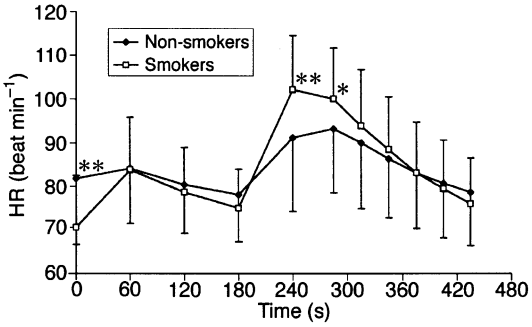

Fig 1 Mean (SD) heart rate in smokers and non-smokers at time intervals from induction of anaesthesia. Intubation was completed by 240 s. *$P<0.05$, **$P<0.01$.

lower heart rates before induction ($P<0.01$) (Fig. 1). In addition, smokers had significantly lower systolic (mean 112.8 (SD 10.6) mm Hg) and mean arterial pressures (82.5 (9.6) mm Hg) before induction than non-smokers (120.6 (12.7) mm Hg, and 90.8 (9.5) mm Hg, respectively). Mean systolic arterial pressures in smokers and non-smokers before intubation were 102.5 (1.9) mm Hg and 105.3 (9.2) mm Hg, respectively, increasing to 129.2 (19.3) mm Hg and 123.9 (19.8) mm Hg on intubation; there was no significant difference between groups. Similarly, diastolic and mean arterial pressures increased in both groups on intubation (ns).

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

Comment

Reflex circulatory responses to laryngoscopy and tracheal intubation were described originally several years ago.[2] These comprise a transient increase in arterial pressure and heart rate. In this study, we have demonstrated a heightened tachycardic response to laryngoscopy and intubation in smokers. Nicotine is known to act through the sympatho-adrenergic system, causing increases in heart rate and vasoconstriction. The half-life 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 catecholamine concentrations.[3] All of our smokers abstained from cigarettes for at least 2 h 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 characteristics of the upper airway epithelium, there is greater exposure of subepithelial upper airway receptors to stimuli.[1] Mechanical stimulation of the upper airway during laryngoscopy and/or intubation may, by a similar mechanism, cause greater haemodynamic changes in smokers. It may also be argued that smokers who have chronic lung disease may be at a lighter plane of anaesthesia at the time of intubation; however, as all patients in our study were ASA I, with no symptoms or signs of lung disease, this is unlikely to be the explanation for our findings.

We have shown that the transient increase in arterial pressure after tracheal intubation did not appear to be affected by chronic exposure to cigarette smoke. However, the peak pressor response, using continuous monitoring, has been shown to occur within 35 s of the start of laryngoscopy[4]; it is possible that, because we used non-invasive arterial pressure measurements, we may have missed the peak pressor response in some patients, and this may have influenced our results.

In summary, we have shown that cigarette smokers exhibited a greater tachycardic response to intubation compared with non-smokers. Most myocardial ischaemic episodes during anaesthesia are associated with intubation and surgical stimulation, especially if tachycardia occurs.[5] Although the difference in heart rate between the groups is unlikely to be clinically relevant, smokers also have acute unfavourable effects on cardiopulmonary function caused by carbon monoxide- and nicotine-mediated changes in oxygen delivery and myocardial oxygen balance.[6] As 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. Finally, as we have shown that smoking alters this cardiovascular response in healthy ASA I women of less than 50 yr of age, we would advise that the smoking habits of subjects should be considered, or that smokers should be excluded from future studies evaluating the haemodynamic responses to intubation.

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