Effects of passive and active smoking on induction of anaesthesia

A. Dennis, J. Curran, J. Sherriff and W. Kinnear

Summary

It is said that cigarette smokers suffer stormy induction of anaesthesia; although plausible, this is unsubstantiated. We have studied the incidence of adverse events during induction, together with peripheral oxygen saturation (SpO2), in active and passive smokers, and in non-smokers. During induction, both active and passive smokers had a higher incidence of adverse events than non-smokers (P < 0.01 and P < 0.05, respectively). Irrespective of smoking status, those suffering adverse events had greater concentrations of carboxyhaemoglobin and suffered more oxygen desaturation than those not suffering such events. Although we were unable to demonstrate a direct link between smoking status and oxygen desaturation during induction, our study gives a firmer basis for exhorting patients not only to stop smoking before anaesthesia but also to avoid passive smoking. (Br. J. Anaesth. 1994; 73: 450-452)

Key words


Cigarette smoking causes cough, mucous hypersecretion and airflow obstruction [1]. Smokers have a high incidence of postoperative respiratory complications [1] and although there is evidence of increased upper airway sensitivity [2], the assumption that smokers suffer stormy induction of anaesthesia [3] seems unsubstantiated. Current non-invasive methods of measurement of oxygen saturation (SpO2) overestimate the true value, because they fail to distinguish between oxyhaemoglobin (HbO2) and carboxyhaemoglobin (COHb) [4] which is increased in smokers [5]. We have investigated the effects of smoking, both active and passive, on respiratory problems during induction of anaesthesia.

Patients and methods

After obtaining Ethics Committee approval, we studied 120 patients, ASA I or II, aged 18-75 yr undergoing elective surgery. Those with known respiratory disease, recent upper respiratory tract infections, gastro-oesophageal reflux or those receiving medications affecting lung function were excluded. Before surgery, one investigator (A.D.) obtained a smoking history, including details of passive smoking at home or at work, previous smoking history and, if an active smoker, the number of cigarettes consumed a day and for how many years. Forty of the non-smoking patients were asked retrospectively about exposure to passive smoking. Measurements were obtained of peak expiratory flow (PEFR) using a Wright spirometer, and forced expiratory volume (FEV1) and forced vital capacity (FVC) using a Vitalograph.

Temazepam 10-20 mg was prescribed as premedication. Before induction of anaesthesia, 2 ml of venous blood was obtained and stored on ice before carboxyhaemoglobin estimation using an Instrumentation Laboratory 282 Co-Oximeter, which was calibrated formally every day. SpO2 was measured by a Datex Satlite monitor. When a stable value was present, a second investigator (J.C.), unaware of the patient’s smoking history, induced anaesthesia with thiopentone 4.5 mg kg-1 given over 30-40 s. After loss of the eyelash reflex, 67% nitrous oxide in oxygen was given at a rate of 9 litre min-1 via a Bain circuit. No oral airway was inserted and no patient required manual ventilation of the lungs. When spontaneous breathing recommenced, enflurane was added in 0.5% increments every three breaths until 5% was reached. This concentration was maintained until 5 min had elapsed since injection of thiopentone, marking the end of the study period. During this period, SpO2 was recorded continuously and an independent observer noted any coughing, breath-holding or laryngospasm and recorded these as “adverse events”. Because SpO2 measurement cannot accurately distinguish between COHb and HbO2, we subtracted venous COHb% to obtain a corrected value for SpO2 [6].

Statistical analysis was performed using unpaired t tests, Fisher’s exact test and analysis of variance (ANOVA) using Minitab and SPSS/PC statistical packages. P < 0.05 was taken as significant.

Results

Patients were allocated to one of three groups: active daily smokers (range 3-30/day), passive smokers (those who did not smoke themselves but lived with a smoker or had regular daily contact with smokers at work) and true non-smokers. Of the 16 ex-smokers,
Effects of smoking on induction of anaesthesia

Table 1  Patient data and pulmonary function tests (mean (range or SD)). No significant differences between groups

<table>
<thead>
<tr>
<th></th>
<th>Active smokers (n = 57)</th>
<th>Passive smokers (n = 21)</th>
<th>Non-smokers (n = 42)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>41.4 (18-73)</td>
<td>42.9 (24-67)</td>
<td>41.6 (19-75)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>68.9 (14.7)</td>
<td>67.6 (9.5)</td>
<td>65.5 (11.9)</td>
</tr>
<tr>
<td>PEFR (litre min⁻¹)</td>
<td>391 (98)</td>
<td>405 (43)</td>
<td>401 (62)</td>
</tr>
<tr>
<td>FEV₁ (litre)</td>
<td>2.48 (0.83)</td>
<td>2.48 (0.64)</td>
<td>2.49 (0.71)</td>
</tr>
<tr>
<td>FVC (litre)</td>
<td>2.88 (0.57)</td>
<td>3.26 (1.06)</td>
<td>3.15 (0.14)</td>
</tr>
<tr>
<td>Sex (M:F)</td>
<td>13:44</td>
<td>2:19</td>
<td>5:37</td>
</tr>
</tbody>
</table>

When compared with non-smokers, both active and passive smokers suffered significantly more adverse events during induction, with no difference between the active and passive smokers (table 2). In all three groups, mean COHb values were significantly greater in those suffering adverse events during induction (table 3). In general, the least corrected value of Spo₂ reached by individuals (fig. 1) was independent of category of smoker but was lower for patients who suffered adverse events (P < 0.01, ANOVA).

Discussion

We have shown that during induction of anaesthesia, in ASA I and II patients, smokers, both passive and active, are more likely to suffer adverse events (coughing, breath-holding and laryngospasm). Irrespective of smoking status, for patients suffering such events during induction, mean COHb was significantly greater than for those not suffering such events. As expected, the reduction in 5pO₂ was also greater in those with adverse events during induction.

Although there are other causes of increased COHb in an urban population other than cigarette smoking [5], our data suggest that COHb is a more reliable predictor of adverse events than asking the patient the number of cigarettes they consume per day.

It is interesting to note that passive smokers also had an increased likelihood of suffering adverse events. The proportions of ex-smokers in the passive and non-smoking groups were similar, making it less likely that the risk in passive smokers was caused by retention of airway irritability in ex-smokers. It is possible that a spectrum of risk exists from the heavy active smoker, to passive smokers to the true non-smoker.

Our study has implications for induction of anaesthesia. First, we now have a further reason for exhorting patients to stop smoking before general anaesthesia and to avoid passive smoking as much as possible. Second, hospital policy should be revised so that patients are not exposed to passive smoking before operation, for example in the ward day room. Third, even if routine measurement of COHb% or exhaled CO with a portable meter are not practical propositions, it is a simple enough part of pre-operative assessment to enquire about passive smoking, and forewarned appropriate precautions, including preoxygenation, should be taken.

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References


