Changes in arterial oxygen saturation in cigarette smokers following general anaesthesia

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The effect of cigarette smoking on postoperative arterial oxygen saturation was evaluated in 45 adult patients using pulse oximetry. Patients were divided into a smoking group (n = 20) and a non-smoking group (n = 25) based on current smoking habits up until the time of surgery. The two groups were similar with respect to sex, ASA physical status, surgical procedure, duration of anaesthesia, narcotic and anaesthetic use and recovery characteristics. The non-smoking group was, however, significantly (P < 0.05) older than the smoking group. Postoperative oxygen saturation (SaO_2) decreased (P < 0.001) during transport of both groups of patients from the Operating Room to the Recovery Room; a decrease which was significantly greater in the smoking group. The severity of hypoxaemia was also significantly greater in the smoking group than in the non-smoking group. This study suggests that cigarette smoking contributes to postoperative arterial oxygen desaturation following general anaesthesia and that supplemental oxygen should be administered to these patients during postoperative transport.

A l'aide d'un saturomètre digital, nous avons évalué l'effet du tabagisme sur la saturation artérielle en oxygène (SaO_2) post-opératoire. Vingt-cinq fumeurs de cigarette constituaient un groupe tandis que l'autre groupe était composé de vingt non-fumeurs. Les deux groupes étaient comparables quant au sexe, la classe ASA, le type d'intervention, la durée et le type d'anesthésie, les morphiniques utilisés et la récupération en salle de réveil. Toutefois, les non-fumeurs étaient plus agés que les fumeurs (P < 0,05). Chez les deux groupes, la SaO_2 diminuait (P < 0,001) pendant le transport entre la salle d'opération et la salle de réveil quoique de façon significativement plus marquée chez les fumeurs. Le degré d'hypoxémie atteint était aussi plus sérieux chez les fumeurs. Il semble donc que la consommation de cigarettes contribue à l'hypoxémie post-opératoire observé après une anesthésie générale et que les fumeurs devraient respirer un mélange enrichi d'oxygène pendant leur transport vers la salle de réveil.

Key Words:
ANAESTHETIC TECHNIQUES: general;
COMPLICATIONS: smoking;
HYPOXIA: postoperative;
OXYGEN: monitoring.

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Cigarette smoking has been described as the single most important preventable cause of morbidity and mortality today. Although the prevalence of current cigarette smoking in the United States is at its lowest level, cigarette smoking is responsible for an estimated 320,000 premature deaths per year and the development of chronic illness in another ten million americans.[1]

For the patient presenting for anaesthesia and surgery, the cigarette smoker is at an increased risk for the development of perioperative complications.[2,3] One complication that has been frequently observed in otherwise healthy patients is arterial oxygen desaturation during transport of the patient from the Operating Room (OR) to the Post-Anaesthesia Care Unit (PACU).[4-7] Although a transient decrease in oxygen saturation following anaesthesia may be easily tolerated in the healthy patient, there exists a potential for severe hypoxaemia in patients with compromised lung function. The respiratory effects of smoking, such as increased carboxyhaemoglobin levels, and decreased pulmonary function, suggest a potential for impaired postoperative oxygen saturation in patients who smoke cigarettes. This study, therefore, was designed to examine, by pulse oximetry, the degree of postoperative arterial oxygen desaturation in a group of self-identified smokers without evidence of symptomatic lung disease.

Methods

Informed consent and approval from the Institutional Review Board was obtained. The study sample consisted

of 45 consecutive ASA physical status I-III adult patients who were free from overt respiratory disease and who underwent a number of elective intra-abdominal procedures.

Information regarding smoking habits and medical history was solicited from each patient in interviews before surgery. Inclusion of patients into the study required that they had no history of chronic obstructive pulmonary disease (COPD), asthma or neoplastic lung disease. Patients were assigned to a smoking group (n = 20) or a non-smoking group (n = 25) based on current smoking habits up until the time of surgery. Preoperative arterial oxygen saturation (SaO_2) was measured before the induction of anaesthesia with the patient breathing room air using a Nellcor N-100 pulse oximeter (Nellcor, Inc, Hayward CA). Details of pulse oximetry as a measure of SaO_2 have been described elsewhere.[8] All patients received a general anaesthetic and all had their tracheas intubated. Details of any pre- and intraoperative medication, narcotic use and muscle relaxant use were recorded. The adequacy of muscle relaxant reversal was tested clinically and by peripheral nerve stimulation. During surgery a 1 ml heparinized blood sample was taken from a random sample of patients and analyzed for carboxyhaemoglobin (COHb) levels using a Radiometer OSM3 Hemoximeter (Radiometer America Inc., Cleveland Ohio).

At the end of surgery, prior to transfer to the PACU, the tracheas were extubated (three were extubated in PACU) and the patients were allowed to breathe 100 per cent oxygen (O_2) until the anaesthetist deemed them suitable for transfer. Measurements of SaO_2 were obtained prior to transfer and after the patient had breathed 100 per cent O_2 for a minimum of three minutes.[5,6] In addition, end-tidal concentrations of carbon dioxide ($ETCO_2$), anaesthetic agent (ETA_g), and nitrous oxide (ETN_2O) were measured at this time using an anaesthetic circuit evaluator (Traverse Medical, Saline, MI). The patient was then transferred to the PACU. No supplemental O_2 was given en route to the recovery unit.

Immediately on arrival in the PACU, oxygen saturation was measured together with measurements of $ETCO_2$, ETA_g, and ETN_2O with the patient breathing room air. The patient's temperature and respiratory rate were recorded and the level of consciousness scored using a standard recovery scale (0 = unresponsive, 1 = arousable, 2 = awake). Immediately following these measurements the patient was allowed to breathe 30–40 per cent O_2 by mask as needed.

The study observers were not directly involved in the anaesthetic care of the patient and were blinded to the patient's smoking status. Comparison of the incidence rates for hypoxaemia was by chi-square analysis. Continuous variables were analyzed using analysis of variance. Significance, unless stated otherwise, was achieved at the five per cent level ($P < 0.05$). All data are reported as mean ± SD.

Results

The demographics of the smoking and non-smoking groups with respect to age, sex, anaesthetic use and surgical procedure are described in Table I. Approximately 86 per cent of the patients were female and this was similar for both groups. The preponderance of female patients was a result of the choice of OR suite, based on the frequency of intra-abdominal procedures performed there. The majority of procedures were gynaecological; however, there was no difference in the distribution of the surgical procedures between the two groups. The mean age of the overall population was 43 yr (range 19–84 yr); however, the non-smoking group was significantly older than the smoking group. Comparison of the distribution of patients within the three ASA classes (I–III) showed no differences between the two groups.

Overall, midazolam was the premedicant of choice (67 per cent of patients) and vecuronium the relaxant of choice (53 per cent). Only two volatile agents were used, isoflurane (76 per cent) and enflurane (24 per cent). In 76 per cent of patients, fentanyl was the narcotic of choice with morphine being used in 14 per cent. The balance of narcotic use was either a combination of fentanyl and morphine or other narcotic agents such as demerol. Table I describes the similarities in the proportional use of the anaesthetic and narcotic agents between the smoking and non-smoking groups.

There were no differences between smokers and non-smokers with respect to duration of surgery (140.8 ± 52.4 vs 150.4 ± 81.7 min), duration of anaesthesia (168.3 ± 68.2 vs 193.6 ± 90.4 min), or the time taken to transport the patient from the OR to the PACU (7.6 ± 4.5 vs 8.5 ± 4.2 min). Cigarette use in the smoking group was measured in terms of pack years of use which ranged from 5 to 55 pack years (mean 15.3 yr.).

Overall, there were significant decreases in ETAg and ETN_2O concentrations during transport of the patient from the OR to the PACU. The ET isoflurane concentrations in the non-smoking and smoking groups, respectively, decreased from 0.25 and 0.22 per cent in the OR to 0.07 and 0.08 per cent in the PACU. Similarly, the corresponding ETN_2O concentrations decreased from 6.63 and 7.51 per cent to 1.36 and 1.92 per cent in the non-smoking and smoking groups respectively. These expected decreases in end-tidal concentrations were also similar between the two groups when enflurane was used as the anaesthetic agent. There was no change in $ETCO_2$ levels during transport of the patient from the OR (5.5 ±

TABLE I Group demographics

	Non-smokers (n = 25)	*Smokers (n = 20)*
Age (yr)	48.4 ± 20.8*	37.0 ± 9.9
Sex: M/F (%)	17.0/83.0	11.0/89.0
Distribution of anaesthetic/drug use (%):		
Enflurane	20.8	26.3
Isoflurane	79.2	73.7
Fentanyl	83.3	78.9
Morphine	12.0	15.0
Midazolam	64.0	70.0
Distribution of surgical procedures (%):		
Exploratory laparotomy	20.0	30.0
TAH/BSO†	48.0	50.0
Upper Abdominal (incl. cholecystectomies)	28.0	20.0
Other	4.0	0.0

*$P < 0.05$ vs smokers. Values are mean ± SD.
†TAH/BSO: Total abdominal hysterectomy/Bilateral salpingo-oophorectomy.

1.5 KPa, 41.9 ± 11.4 mmHg) to the PACU (5.3 ± 1.0 KPa, 40.7 ± 7.7 mmHg) and this finding was consistent for both groups.

The recovery characteristics of the patient population in the PACU were similar for both groups. The respective recovery characteristics for the smoking and non-smoking groups were measured in terms of temperature (35.3 ± 0.5 vs 35.7 ± 0.3° C), respiratory rate (18.6 ± 4.5 vs 18.1 ± 4.5 breaths · min^{-1}) and level of consciousness. The distribution of patients within the three levels of consciousness (i.e., asleep, arousable, and awake) was similar for both groups. The majority of patients in both groups (69 per cent) arrived in the PACU in the arousable state.

Arterial oxygen saturation data are shown in Table II. There was a highly significant ($P < 0.001$) decrease in SaO_2 during transport of the patient from the OR to the PACU which was reflected in both smoking and non-smoking groups. The magnitude of the decrease in SaO_2 was, however, significantly greater in the smoking group. The severity of postoperative hypoxaemia is presented in Table III. The incidence of severe hypoxaemia (SaO_2 < 85 per cent) was significantly higher in the smoking group. Although there was evidence of moderate hypoxaemia (SaO_2 85–90 per cent) among the non-smokers, arterial saturation did not decrease below 85 per cent. Two patients were determined by the attending anaesthetist to be obese. One patient was in the non-smoking group (wt 102 kg, ht 1.64 m) and the other in the smoking group (wt 118 kg., ht 1.69 m). Oxygen saturation levels measured in the PACU were 95 and 83 per cent for the obese non-smoker and smoker respectively. There was no observed morbidity or mortality resulting from any of the hypoxaemic episodes and all patients responded rapidly and appropriately to O_2 administration in the PACU.

Blood carboxyhaemoglobin levels were significantly higher in the smoking group (mean 3.1 per cent; range 2.1–5.9) than the non-smoking group (mean 1.2 per cent; range 0.8–1.7). The tracheas of three patients were extubated in the PACU; two of the patients were non-smokers and one a smoker. There was no difference in the SaO_2 levels or recovery characteristics of this group compared with the patients extubated in the OR.

TABLE II SaO_2 (%) on induction of anaesthesia and during transport from OR to PACU

	Non-smokers (n = 24)	*Smokers (n = 19)*
Induction	96.3 ± 2.1	96.4 ± 2.4
OR	99.5 ± 0.9	98.8 ± 1.8
PACU	93.8 ± 3.7*	90.5 ± 6.5*†

Values are mean ± SD.
*$P < 0.001$ PACU vs OR.
†$P < 0.05$ PACU smokers vs non-smokers.

Discussion

The two study groups were similar with respect to patient

TABLE III Postoperative hypoxaemia: smokers vs non-smokers

SaO_2 %	*% Non-smokers (n = 24)*	*% Smokers (n = 19)*
≤84	0	15.8*
85–89	16.6	15.8
90–94	41.7	36.8
≥95	41.7	31.6

*$P < 0.05$ vs non-smokers.

demographics and clinical management, differing only in smoking habits and age. Although all patients in the study were free of overt respiratory disease such as COPD there is evidence to suggest that apparently healthy cigarette smokers may have varying degrees of undetected pulmonary disease. Niewoehner *et al.* showed that in a group of young otherwise healthy cigarette smokers there was evidence of bronchiolitis and pathologic changes in the small distal airways consistent with early prodromal COPD.[9] Pulmonary function tests also reveal that smokers have smaller forced expiratory volumes, reduced functional residual capacities (FRC) and lower diffusion capacities compared to non-smokers. In addition, cigarette smokers have increased airway resistance and greater closing capacities (CC).[10–12] Cigarette smokers also demonstrate elevated levels of carboxyhaemoglobin in the blood which can result in a decrease in the amount of haemoglobin available for combination with oxygen and a shift in the oxygen dissociation curve to the left.[13,14]

That the pre-induction saturation levels were similar in the two groups suggests that any compromise of pulmonary function in the smoking group was not reflected in SaO_2 levels with the patient in the awake state. Carboxyhaemoglobin levels, although significantly elevated in the smoking group, represented the lower end of the range for that group. In non-smokers COHb levels usually peak at approximately 2.5 per cent whereas smokers have levels between 3 and 15 per cent.[14] The relatively low COHb levels found in our smoking group suggest minimal influence of carbon-monoxide on the oxygen dissociation curve and SaO_2. The measurement of COHb was pertinent for another reason. A recent report by Barker *et al.* suggests that in the presence of high levels of COHb pulse oximetry is inaccurate resulting in an over-estimation of SaO_2.[15] Based on the findings from Barker's study, the levels of COHb observed in our study would not have affected the measurement of SaO_2 by the pulse oximeter.

Oxygen saturation levels taken during 100 per cent O_2 administration at the end of surgery were similar and adequate in the two groups. However, during transport of the patient from the OR to the PACU, there was a significant decrease in SaO_2 which was exacerbated in the smoking group. Hypoxaemia in the immediate postoperative period is a common sequel to general anaesthesia.[16] A number of recent studies using pulse oximetry has demonstrated significant oxygen desaturation during transport of the patient from the OR to the PACU and in the immediate postoperative period.[4–7] In a study of 95 adult patients, Tyler *et al.* showed that the incidence of postoperative hypoxaemia ($SaO_2 < 90$ per cent) was 35 per cent and the incidence of severe hypoxaemia ($SaO_2 < 85$ per cent) was 12 per cent.[5] Using Tyler's definitions of hypoxaemia and severe hypoxaemia the overall incidence rates in our study were 22 and seven per cent respectively. The incidence of severe hypoxaemia in our study, however, was limited entirely to the smoking group.

A number of mechanisms has been implicated in the genesis of postoperative hypoxaemia including nitrous oxide diffusion hypoxia, the abolition of the hypoxic pulmonary vasoconstrictor response, and changes in pulmonary mechanics.[16,17] The most consistent explanation for postoperative hypoxaemia is a decrease in FRC which is shown to occur during general anaesthesia.[18,19] Various studies have demonstrated decreases in FRC of between 10–25 per cent in both spontaneously breathing patients and during controlled ventilation.[20,21] As FRC is reduced from total lung capacity, the volume at which the airways start to close, (closing volume) is reached. Non-ventilated or collapsed alveoli distal to the closed airways result in an increase in venous admixture and an increase in the alveolar component of the physiologic shunt.[16] Patients who smoke tobacco or who have COPD or who are obese may have closing capacities during anaesthesia which approach or exceed FRC, resulting in ventilation/perfusion mismatching, an increase in the alveolar-arterial oxygen difference and hypoxaemia.[22,23] These changes in pulmonary mechanics continue into the postoperative period[16,24] and may explain the greater degree of hypoxaemia observed in the cigarette smokers compared with non-smokers.

The similarity between the two groups in this study was important in reducing the influence of variables which may affect or confound the apparent relationship between cigarette smoking and postoperative SaO_2. The potential confounding factors in this study are those that individually are known to affect SaO_2. These include respiratory depression due to narcotic use or inadequate muscle relaxant reversal, temperature, age, surgical site, duration of anaesthesia, and obesity.[16,25–28] Respiratory depression does not appear to have been a factor in this study, given that the smoking and non-smoking groups were similar in terms of their pharmacological milieu, their respiratory rates, and their recovery scores, and that there was no evidence of CO_2 retention postoperatively. Postoperative temperature and duration of anaesthesia were also similar for each group. Individual ASA physical status was similar for both groups and had no apparent effect on SaO_2. The ages of the two groups were different in that the non-smoking group was significantly older than the smoking group. It is known that closing volume increases with age and approaches FRC at age 40 with patients in the supine position.[25,28] One would expect, therefore, that the older non-smoker would have worse postoperative SaO_2s than the younger smokers. Also, for

those patients that exhibited severe hypoxaemia postoperatively, there was no apparent age-dependence observed in this study.

The site of incision and the surgical procedure have been shown to affect postoperative SaO_2. A greater number of hypoxaemic episodes are associated with surgery of the thorax and upper abdomen.[24,27] Although several of the patients in our study underwent upper abdominal procedures the distribution of these cases were similar between the two study groups.

Although no formal attempt was made to evaluate obesity in terms of a height and weight index, two patients were noted by the anaesthetist as being obese. Obesity is a risk factor for postoperative hypoxaemia due to the reduction in FRC and the increase in CC that occurs in these patients.[22] Although desaturation did not occur in the one obese patient in the non-smoking group the obese cigarette smoker was severely hypoxaemic postoperatively ($SaO_2 < 85$ per cent). Although it is not possible in this one patient to determine the proportional contributions of cigarette smoking and obesity to the observed desaturation, it is reasonable to assume that since the physiologic changes that occur in the two states are similar (i.e., reduced FRC and increased CC), the patient who is both obese and who smokes would be at an even greater risk of desaturating postoperatively. Removal of the two obese patients from the analysis showed that the increased incidence of severe hypoxaemia in the smoking group was maintained and thus reduced the confounding effect of the obesity.

In the absence of a dose–response relationship between cigarette consumption and oxygen desaturation it was not possible in this study to establish cause and effect. However, given that the two study groups were so similar with respect to all the potential confounding variables we present evidence that postoperative arterial oxygen desaturation may be exacerbated in patients who smoke cigarettes and that episodes of severe hypoxaemia may occur in these patients following general anaesthesia. Although a decrease in postoperative SaO_2 among smokers is predictable given the individual physiological effects of cigarette smoke and of general anaesthesia, the fact that in our study the incidence of severe hypoxaemia was significantly greater among smokers than non-smokers should be of considerable concern to the anaesthetist.

Although there was no morbidity or mortality associated with any of the hypoxaemic episodes, and all the patients responded promptly to the administration of oxygen in the PACU, it is obviously desirable to avoid hypoxaemia postoperatively. It would appear, therefore, that the safest and most conservative approach would be to administer supplemental O_2 routinely during transport of these patients from the OR to the PACU.

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