Smoking and the surgical patient

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Key points

- Smoking adversely affects the surgical patient before, during and after surgery
- During the peri-operative period, the main problem is a reduction in the delivery of usable oxygen to the tissues
- Children of smokers are affected as well as their parents
- Pre-operative abstinence should improve outcome
- Smoking cessation at specific times has specific benefit

In 1944, 20 years before the American Surgeon General identified cigarette smoking as a hazard to health, Morton described it as a cause of post-operative pulmonary complications. This was the start of the accumulating evidence that anyone scheduled to undergo surgery should, as a matter of urgency, be helped to stop smoking. From before diagnosis of the surgical condition to many years after operation, smoking may adversely affect a surgical patient. This review sets out to summarise the current level of understanding of these risks (Table 1).

Smoking and surgical outcome

Firstly, smoking is partly responsible for many of the diseases that require surgery. These include most thoracic surgery, coronary artery by-pass grafting (CABG), gastrectomy, bladder cancer and even femoral neck fracture. Crohn's disease is commoner in smokers and continuing smoking increases their risk of requiring surgery by at lest 50%. Secondly, smoking can affect surgical outcome. For example, smokers fare worse after vascular surgery, CABG and colorectal anastomoses (which are more prone to leak). Plastic surgical flaps are more likely to necrose in smokers and patients surviving a subarachnoid haemorrhage are more likely to re-bleed if they continue to smoke.

Peri-operative effects of smoking

The peri-operative course of both active and passive smokers is compromised because smoking undermines fundamental aims of safe anaesthesia and surgery, *i.e.* a safe start, passage through surgery and rapid uncomplicated recovery. Through all these, the adequate delivery to the tissues of usable oxygen (O_2) is the recurrent theme. The threat is because of smoking-induced: (i) changes in O_2 carriage in the blood; (ii) damage to the lung and airway; and (iii) changes in the heart and circulation.

Table 1 The effects of smoking

Effect	Implications
Respiratory	
Decreased oxygen carriage	Hypoxaemia
Irritable upper airways	Laryngospasm
Irritable lower airways	Bronchospasm
Depressed ciliary function	Retained secretions and infection
Decreased FEV_1	
Increased closing capacity	
Cardiovascular	
Hypertension	Peri-operative ischaemia
Ischaemic heart disease	Myocardial infarction
Hypercoagulation	Thrombosis
	Worst outcome after coronary artery bypass graft surgery and vascular surgery
Gut	
Crohn's disease	
Gastro-oesophageal reflux	
Increased anastomotic leaks	
Peptic ulcerations	

After surgery, smokers spend longer before discharge from theatre recovery rooms and have a higher rate of unplanned admission to higher dependency care areas. This is, in part, related to untoward incidents associated with O_2 desaturation that occur both in smoking adults and their children.

Oxygen carriage in blood

Oxygen and haemoglobin (Hb) interact in a characteristic fashion to give the well-known oxygen–haemoglobin dissociation curve. Under normal circumstances, after blood has passed through the lungs, Hb is almost fully saturated with O_2 even when the concentration of O_2 in the lungs (and, therefore, in arterial blood) is slightly reduced. In tissues, where pH is lower and CO_2 concentration and temperature are greater, the characteristics of Hb favour O_2 release.

In smokers, several changes occur. Blood concentration of carbon monoxide (CO) is increased to as much as 10% (normal 2%). This has two

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effects on O_2 carriage. First, Hb has 250 times more affinity for CO than for O_2, so the total amount of Hb available for O_2 carriage is markedly decreased. Secondly, CO shifts the dissociation curve to the left, which reduces the ability of Hb to release O_2. To make matters worse, CO also inhibits cytochrome oxidase, the enzyme that is needed for the final oxygen dependent synthesis of ATP in the mitochondria. This compromise of O_2 delivery makes the events that ordinarily tend to reduce O_2 saturation (*i.e.* induction of anaesthesia, intra- and peri-operative critical incidents, postoperative hypoventilation and atelectasis) more threatening for smokers. With values of carboxyhaemoglobin (COHb) approaching 10–15%, the smoker may already be near the point on the dissociation curve where any further reduction in P_aO_2 will lead to rapid desaturation.

The light absorbance of COHb is nearly the same as that of oxygenated Hb at the wavelengths used by bedside pulse oximeters. Thus, they cannot differentiate COHb from oxyhaemoglobin and over-estimate O_2 saturation. Just as reliance on oximeters in other situations of severe CO excess (*e.g.* house fires) is dangerous, an apparently normal reading can lead to a false sense of security in heavy smokers.

Airways

Smokers have 'irritable' upper airways and this increases the tendency for breath-holding and laryngospasm at induction of anaesthesia. Although usually a minor inconvenience, these can be life threatening. The commonly used volatile anaesthetics are bronchodilators, although some such as desflurane have a direct irritant effect on the airways. Any bronchodilating effects of volatile anaesthetics are obviously lost when anaesthesia ends.

Lower airway reactivity is also increased in smokers and mucociliary transport impaired. The forced expiratory volume at 1 s (FEV_1) declines in smokers at a rate of around 60 ml yr^{-1} compared with 20 ml yr^{-1} in non-smokers. Also, the closing capacity (*i.e.* lung volume at which airways collapse and trap air) is increased. Smokers have a greater degree of shunt under anaesthesia, even when changes in functional residual capacity and closing capacity are accounted for. This is presumably due to altered regional pulmonary mechanics. These changes, in addition to the changes in respiratory mechanics that occur with both surgery and anaesthesia itself, make hypoxia more likely both during and after operation. The relative risk of postoperative pulmonary complications in smokers versus non-smokers is around six. The absolute values vary between studies (5–25% non-smokers, 22–57% current smokers). Passive smoking has recently been identified as a strong predictor of critical incidents in children with presumed upper respiratory tract infection undergoing anaesthesia.

Cardiovascular effects

Smokers are prone to hypertension, ischaemic heart disease, cerebrovascular disease and heart failure. All of these are risk factors for postoperative cardiovascular morbidity and mortality. Smokers also have higher resting plasma catecholamine concentrations than non-smokers and an exaggerated sympathetic response to desflurane anaesthesia. The electrocardiograms of smokers are more likely to show ST segment depression during general anaesthesia, implying impaired coronary perfusion that, together with hypoxaemic effects of COHb, reduces myocardial O_2 supply during the peri-operative period, especially when the demand is increased. This phenomenon is reflected in the reduced time of onset of exercise-induced angina and the increased incidence of ventricular dysrhythmia and dysfunction in the awake subject with COHb values of as little as 4.5–6%. Myocardial ischaemia itself promotes carboxymyoglobin formation reducing myocardial O_2 supply still further, which, together with the effect of CO on cytochrome oxidase, may explain the known negative inotropic effect of CO.

Miscellaneous adverse effects

Smokers may suffer polycythaemia secondary to chronic hypoxaemia, worsened by a degree of plasma volume reduction secondary to carbon monoxide (CO) exposure. This leads to blood hypercoagulability but of more significance is the enhancement of platelet function and increase in plasma fibrinogen concentrations seen in smokers. These effects are associated with increased risk of both central and peripheral arterial thrombosis, especially when associated with the use of oral contraceptives.

Smoking affects the gut with an increased risk of peptic ulcer disease, gastro-oesophageal reflux and Crohn's disease. Patients with ulcerative colitis are more likely to present for the first time after stopping smoking, though smoking does not improve the condition. There may be a small increase in residual stomach contents, but the gastric pH of smokers and non-smokers appears to be much the same. Overall, there is no evidence of increased pulmonary aspiration risk. The risk of postoperative nausea and vomiting is reduced in smokers, possibly because of induction of the cytochrome P450 enzyme system. Nicotine, a potent neurotransmitter found in cigarette smoke, alters responses to muscle relaxants used in anaesthetic practice, but these are of little, if any, clinical significance. Smoking does not alter barbiturate pharmacodynamics and pharmacokinetics but end-organ responsiveness to benzodiazepines is reduced.

Stopping smoking before surgery

A recent national press article asked, '*why anyone under the age of 50 years should expect to be treated free on the (UK) NHS for a smoking related disease is beyond rational explanation*'. Fortunately, logic assures us that this argument can be dismissed by *reductio ad absurdum*. It is a short step to asserting that nobody who has contracted an illness attributable to a habit, addiction or life-style of which some of us might well disapprove, should expect so called 'free' treatment, which they or the community may have funded in the first place. Support, encouragement and 'chipping away' at the problem are better weapons than threats.

In hospitals, the atmosphere of restricted smoking has been achieved by common-sense and our experience is that patients do try to use this as an environment in which to stop smoking. The 'life-event' of an operation can capitalise on this and we can tell patients that, as well as the obvious benefits of permanent cessation, stopping or reducing smoking before their operation makes sense. This can be done at any time from when patients present to their primary health team to just before anaesthesia starts. The specific benefits of stopping smoking and the health workers who can act as 'catalysts' for this are summarised in Table 2.

The haemodynamic effects of acute exposure to cigarette smoke lasts about 1 h. Abstinence from cigarettes for 12 h increases physical work capacity by 10–20%, a similar change to that required for successful pre-optimisation in high-risk surgical patients.

The effects of smoking cessation on respiratory function are complex and not understood completely. The rate of decline of FEV_1 falls from approximately 60 ml yr^{-1} to 20 ml yr^{-1} within a year of cessation. It takes 6 months' cessation to bring the postoperative pulmonary complication rate down to that of those who have never smoked, and after 1–2 months, the rate may be worse than those who persist in smoking in some situations. These observations are in keeping with evidence that improvements in ciliary activity, small airway function and sputum production take some weeks to establish themselves, and also with the suggestion that there may be a beneficial irritant and presumably expectorant effect of cigarette smoke, perverse though this observation seems. Studies suggesting the latter should be interpreted with caution due to the inevitable methodological problems in determining smoking status and reasons for stopping. They do not support the approach that it is not worth stopping within 2 months of surgery. Improvements in upper airway reactivity can be anticipated after only 48 h cessation, although peak benefit is only reached after 10 days.

Finally, a late, but important, opportunity presents the night before surgery. Clearance of COHb is improved by overnight abstinence because its half-life is approximately 4–6 h. Therefore, O_2 carriage and delivery are significantly improved. Of interest, is the observation that CO clearance is affected by exercise, sleep and sex. The elimination half-life of CO is longer in men, approaches 10 h during sleep but falls to 1 h during exercise. Therefore, those most at risk are inactive men who smoke heavily before retiring to bed on the night before, and also rise to smoke on the morning of their operation.

Table 2 Stopping smoking – benefits and catalysts

Time frame	Effect	Possible catalysts
Years	Reduction in lung cancer, chronic obstructive pulmonary disease, ischaemic heart disease, cerebrovascular disease	GP, society, government
5–6 months	Reduction of postoperative complications	GP, surgeon
1 month	Possible increased risk of postoperative pulmonary complications	GP, surgeon
2–10 days	Improvements in upper airway reactivity	Anaesthetist, surgeon, nurses in pre-admission clinics
12–24 h	Clearance of carbon monoxide	Anaesthetist, surgeon, nurses

The psychological and physiological effects of smoking are sometimes given as reasons not to stop smoking peri-operatively and withdrawal symptoms may be more severe in partial rather than complete quitters. Nicotine replacement may alleviate much of the psychological component. 'Physiological' effects include decreases in plasma epinephrine and norepinephrine concentrations and a symptom complex described as a state of 'less arousal'.

If the surgical team is to influence smoking, it clearly needs to do so early. Smoking cessation clinics are relatively cheap, often nurse-led and are effective both in primary care and in association with surgical preclerking clinics. Very brief smoking advice is less effective but still worthwhile and can be accommodated into surgical outpatient practice and anaesthetic visits or clinics. Current best practice advocates the use of nicotine replacement therapy that doubles the chances of cure. Bupropion (Zyban®) has been recently introduced in the UK, but its place in early postoperative cessation has not been evaluated. Postoperative, predischarge intervention to promote smoking cessation also doubles the chance of long-term abstinence.

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See multiple choice questions 76–78.