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REVIEW

Anaesthesia in smokers

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SUMMARY

Smoking increases the risk of postoperative complications. The potentially most serious smoking-related complications are cardiovascular and respiratory events and delayed wound healing and infection. Smokers should have these risks explained in the preoperative visit and lifetime exposure should be calculated. High exposure should signal the need to refer patients for expert management whether or not clinical manifestations of respiratory disease are found. Preoperative abstinence from smoking must be strongly recommended in all situations because it has been shown to reduce postoperative morbidity. The parents of children about to undergo surgery should be warned of the consequences of passive smoking.

1. Introduction

Smoking-related disease is expected to become the leading cause of death worldwide by 2030, causing about 10 million deaths annually, three times more than in 1995.[1] Although the rate of smoking in Western countries is continuously declining, mainly due to persistent public health policies, between a quarter and a third of patients undergoing surgery are still active smokers.[2] As smoking is associated with adverse surgical outcomes and is a recognized risk factor for postoperative complications,[3] patients who smoke continue to be a major concern for anaesthesiologists planning perioperative care strategies. Meanwhile, the number of former smokers is increasing, and ex-smokers may still be at higher risk of postoperative complications, depending on when they quit. Children are another group that concerns us, as those who are exposed to passive smoking also share the risk of perioperative complications. [4] Recent reviews and recommendations suggest that these risks should be disclosed during patient interviews and that the possible advantages of quitting preoperatively should be emphasized.[5–10] Anaesthesiologists should bear in mind that preparation for surgery is a good moment to promote long-term smoking cessation.[11]

2. Effects of smoking

2.1. Cardiovascular system

Nicotine has an impact on haemodynamics by directly acting to increase the sympathetic tone of peripheral blood vessels and indirectly raising the level of circulating catecholamines. Consequently heart rate, blood pressure, peripheral vasoconstriction and myocardial contractility and work all increase. In addition, oxygen delivery is impaired because carbon monoxide increases carboxyhaemoglobin levels, which can exceed 10% in smokers.[8] The ability of the blood to carry oxygen decreases and the oxyhaemoglobin dissociation curve shifts to the left, impeding the release of oxygen.[12] In smokers with coronary artery disease these effects contribute to exercise-induced angina and increase the frequency of ventricular arrhythmias. Nicotine and other substances in cigarette smoke may affect mitochondrial respiration because they inhibit haeme-containing proteins, such as cytochrome C oxidase.[13] Smoking also produces pro-inflammatory effects, endothelial damage, and oxidant injury, while enhancing thrombosis and affecting blood lipids adversely.[8] Together, all these effects promote atherosclerosis.

2.2. Respiratory system

Changes in lung morphology and immune function occur as smoking induces an inflammatory state in the lung, increasing the number of macrophages and neutrophils, whose function is also altered.[14] These cells become less capable of releasing inflammatory mediators or mounting an effective response to infection.

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Airway epithelial structure and function change and small airways become narrower. Because smoking produces goblet cell hyperplasia, the volume and composition of mucous is affected and mucociliary clearance decreased. Other changes lead to increased smooth muscle and epithelial permeability, loss of pulmonary surfactant tension and fibrosis.[15] Overall, these changes accelerate age-related decline in forced expiratory volume in one second (FEV_1).[16] Airway reactivity to methacholine also increases,[17] although the ability of inhaled irritants such as capsaicin and citric acid aerosols to trigger cough diminishes.[18] Fifteen percent of smokers, depending on individual susceptibility in ways that have not yet been well established, develop a lung disease[19] and an additional 50% will eventually develop chronic bronchitis.[20]

2.3. Wound and bone healing

The higher incidence of complications related to tissue healing in smokers is probably due to a combination of effects on tissue oxygenation, the vascular system, the healing process and the immune system.[7] Nicotine inhibits proliferation of fibroblasts and macrophages and increases platelet adhesion, which causes microclots and reduces microperfusion. Smoking has significant effects on bone metabolism and is a major risk for osteoporosis,[21] which may itself impair the healing of fractures.

2.4. Nervous system

Nicotine activates several subtypes of nicotinic acetylcholine receptors. These receptors are located throughout the body, but their main role is in the central nervous system.[8] The psychoactive effects of nicotine seem to be mediated in part through the activation of dopaminergic neurons.[22] Tolerance to nicotine arises rapidly through desensitization with continued exposure, and prolonged exposure can also induce long lasting plastic changes in the central nervous system. These effects explain smokers' withdrawal symptoms on eliminating or reducing nicotine intake. Although the activation of acetylcholine receptors on peripheral nerves produces pain, the application of nicotinic agonists to different sites in the central nervous system can have either pronociceptive or anti-nociceptive effects. Smokers exhibit a lower tolerance for pain and therefore require more analgesics irrespective of the effect of smoking on the analgesic.[23]

2.5. Other effects

Smoking leads to gastro-oesophageal sphincter incompetence, which allows reflux. However, this incompetence abates within 8 min of stopping smoking.[24] Smoking induces liver microsomal enzymes, thereby increasing the rate of metabolism of some drugs[25] and may require higher dosages than never-smokers or ex-smokers.[26]

3. Perioperative risk

3.1. Cardiovascular complications

Because smoking contributes to cardiac disease, it increases perioperative cardiac risk. However, it is not clear whether active smoker status itself increases this risk. We think intuitively that the acute effects of nicotine and carbon monoxide must have deleterious effects, however, so stopping smoking 12–48 h before a procedure should improve oxygen availability and reverse negative inotropic and arrhythmic effects.[27] One study, in fact, found a correlation between electrocardiographic signs of ischaemia in anaesthetized patients with high carbon monoxide levels.[28] Refraining from smoking after a procedure should also be beneficial. The risk of death after coronary artery bypass grafting is lower in patients who quit smoking after surgery.[29] Persistent smokers have more risk of angina after myocardial infarction[30] and are more likely to undergo a repeat coronary artery bypass.[31] Continued smoking also increases the risk of restenosis after carotid endarterectomy[32] and of graft failure after lower extremity bypass surgery[33] and after renal transplantation.[34] Smoking may also increase the risk of cardiovascular complications after non-cardiovascular surgery, such as hip or knee replacement.[9] Nonetheless, in spite of smoking's measurable effects on the cardiovascular system, this factor has not been selected in indices developed to predict cardiovascular complications.[35,36] Smoking remains an undeniable, demonstrated risk factor for coronary and cerebrovascular disease, however.[37]

3.2. Respiratory complications

Smoking is associated with a higher incidence of perioperative respiratory complications.[3] Because smoking can affect the severity of pulmonary disease, it is difficult to separate the risk posed by smoking itself or by smoking-related pulmonary disease. The overall relative risk among smokers is 1.8, and this risk is higher in young and obese smokers.[38] Several mechanisms may contribute to perioperative risk. Excessive mucous secretion itself seems to be a risk factor. In addition, the sensitivity of upper airway reflexes to chemical stimulants is increased and airway irritation on contact with desflurane is greater in smokers.[39] However, pulmonary resistance after tracheal intubation and the frequency and amplitude of coughing during emergence from isoflurane anaesthesia are not affected by smoking status. Smokers' risk for developing postoperative pulmonary complications has been seen to be 2–6 times higher than the risk of never-smokers in different studies, which define complications variously.[2,40] The population-based ARISCAT study, designed to assess the postoperative complications associated with smoking and surgery in a randomly selected general surgical population,[41] found postoperative pulmonary complications in significantly more former smokers (8.9%) than current (2.4%) or never-smokers (3.7%). The mean age of current smokers was much lower than the other subpopulations (by 17 yr) and these young smokers had accumulated significantly less lifetime exposure. For this reason, smoking status by itself is not as good a predictor of risk as packet-years, independently of the current status. One packet-year is equivalent to consumption of twenty cigarettes a day for one year. Smokers or former smokers with a lifetime exposure greater than 40 packet-years had three times more postoperative respiratory complications (13.1%) than those with less lifetime exposure (4.1%) or never-smokers (3.7%) in the ARISCAT study, an observation consistent with others in the literature.[42] Men who smoke suffer a loss of 7.4 ml in FEV_1 for each packet-year of exposure; for female smokers the loss is of 4.4 ml.[43] A direct relationship between the number of packet-years and the progression of severity of chronic obstructive pulmonary disease (COPD) has also been demonstrated.[44]

3.3. Wound complications

Suture dehiscence is a common problem in abdominal surgery, appearing after around 3.4% of laparotomies in a large study of 17,044 patients in hospitals that serve military veterans in the United States.[45] In the predictive model developed in that study, the presence of COPD was related to surgical wound dehiscence. Surgical wound infection represents a very frequent nosocomial infection and 4.3% of patients in another large study (163,624 patients) had wound infections.[46] One of the fourteen factors