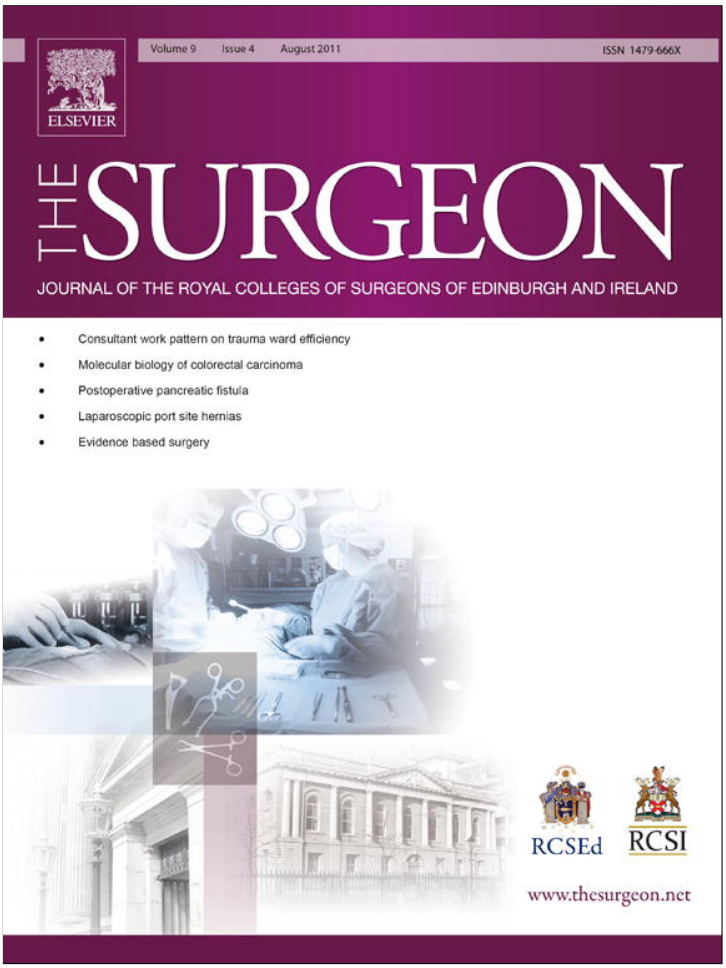

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Review

The effects of tobacco smoking on the incidence and risk of intraoperative and postoperative complications in adults

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ABSTRACT

Introduction: Despite the warnings of health hazards of cigarette smoking, still one third of the population in industrial countries smoke. This review was conducted with the aim of exploring the effects of preoperative tobacco smoking on the risk of intra- and postoperative complications and to identify the value of preoperative smoking cessation.
Methods: The databases that were searched included The Cochrane Library Database, Medline, and EMBASE. Articles were also identified through a general internet search using the Google search engine. The incidence or risk of different types of intra- and postoperative complications were used as outcome measures.
Results: Tobacco smoking has a negative effect on surgical outcome, as has been found to be a risk factor for the development of complications during and after many types of surgery, even in the absence of chronic lung disease. Furthermore, the long-term health hazards of smoking reduce health-related quality of life and premature death.
Conclusion: It is widely documented that stopping smoking before surgery has substantial health benefits in the longer term and should be recommended to every smoker in order for them to gain maximum benefit from their treatment. However, identification of the optimal period of preoperative smoking cessation on postoperative complications cannot be determined.

Introduction

Despite the warnings of health hazards of cigarette smoking, still one third of the population in industrial countries smoke.[1] In 2006, 20.1% of adults in the United States were smoking cigarettes.[2] Cigarettes contain more than 4000 chemicals some of which are pharmacologically active (antigenic, cytotoxic, mutagenic or carcinogenic).[3]

Smoking has a negative effect on surgical outcome, as the carbon monoxide (CO) and nicotine, inhaled from smoking a cigarette, increase heart rate and blood pressure and the body's demand for oxygen. Nicotine also causes vasoconstriction reducing the blood flow to certain parts of the body.[4] Smoking has been found to be a risk factor for the development of complications during and after many types of surgery, even in the absence of chronic lung disease. This happens

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because smoking causes the small-airways in the lungs to narrow, making them more prone to collapse and leading to increased susceptibility of infection, coughing, pulmonary complications, and prolonged mechanical ventilation in smokers.[5]

The relative risk of complications after surgery for smokers compared to non-smokers has been reported to increase from 1.2-fold to 5.5-fold.[6,7] Daily smoking increases the development of postoperative complications by two to four times.[8,9] Smoking inhibits immune responses involved in wound healing,[10] increases the risk of blood clotting,[11] and remains serious risk factor in anesthesia. It has been found that smokers often require larger doses of muscle relaxants and other anesthetics.[12,13] Smokers also have a higher prevalence of chronic bronchitis than non-smokers (23.3% vs. 4.8%),[14] while the incidence of perioperative respiratory events occurs in 5.5% for smokers and in 3.1% for non-smokers.[14] Postoperative complications cause suffering and are costly for society.[15] Added to this, the long-term health hazards of smoking reduce health-related quality of life and premature death.[16,17]

However, evidence suggests that nicotine and CO levels are usually reduced after 120 min and 12 h respectively.[18,19] Therefore, there may be benefits to health from short-term smoking cessation, such as improved oxygen delivery to the patient. It is well documented that preoperative smoking cessation programs of 4–8 weeks duration, significantly reduce the increased risk of complications after surgery.[20,21]

This review was conducted with the aim of exploring the effects of preoperative tobacco smoking on the risk of intra- and postoperative complications and to identify the value of preoperative smoking cessation.

Study design

We defined the term 'complication' as a secondary disease or a negative reaction occurring during, or after a surgical procedure. The incidence or risk of different types of intra- and postoperative complications such as respiratory, cardiovascular, gastrointestinal, wound infection, mortality, and duration of hospital stay were used as outcome measures. Studies that did not explore the risk of these complications in current and past tobacco smokers were excluded from this review.

Search strategy

The databases that were searched included The Cochrane Library Database, Medline, and EMBASE. Articles were also identified through a general internet search using the Google search engine. The search terms that were used included, smoking, smoking cessation, tobacco use, tobacco-abstinence, cigarettes, postoperative complications, surgery, surgical procedures and postoperative complications. Search terms were selected based on common key terms identified in the initial search of the literature and related reviews conducted in the area. Reference lists were checked to ensure all relevant articles had been identified.

Effects on cardiovascular system

Nicotine stimulates the adrenal medulla to secrete adrenaline, resets the carotid body and aortic receptors to maintain a higher blood pressure, and stimulates autonomic ganglia increasing sympathetic tone. The result is an increase in systolic and diastolic blood pressure, in heart rate, and also in peripheral vascular resistance. These increase the myocardial contractility leading to an increase in oxygen consumption by the cardiac muscle.

Increase in excitability leads to more frequent contractions and again an increase in oxygen consumption. Thus, the demand for oxygen is increased. An increase in coronary vascular resistance leads to a decrease in the coronary blood flow resulting in a decrease in oxygen supply.[22] Nicotine also increases intracellular calcium during ischemia. This may exacerbate myocardial cell damage.[22]

The other constituent of smoke that affects the cardiovascular system is CO. In the blood, CO combines with hemoglobin (Hb) to form carboxyhemoglobin (COHb). In smokers, the amount of COHb in the blood ranges from 5 to 15%. In non-smokers, it is only about 0.3–1.6%.[2] The amount of COHb presents in the blood of smokers depends on the type of cigarette smoked, the frequency, and the method of smoking. The affinity of CO for Hb is 200 times that of oxygen.[2] Thus, the amount of Hb available for combining with oxygen is drastically reduced. It also shifts the oxyhemoglobin (HbO_2) curve to the left due to the fact that makes it difficult for tissues to extract oxygen from the Hb. The result is a decrease in the oxygen available to the tissues. CO also binds with cytochrome oxidase and myoglobin and inactivates mitochondrial enzymes in the cardiac muscle.[22] The result is a decrease in the intracellular oxygen transport and usage and a negative inotropic effect. These mechanisms lead to chronic tissue hypoxia. The body compensates with an increase in red blood cells. The result is an improvement of the oxygen availability at the expense of increased plasma viscosity.

Smoking increases the production of Hb, red blood cells, white blood cells, fibrinogen, platelets, and platelets reactivity.[22] These result in an increase in the hematocrit and the blood viscosity leading to an increased thrombotic tendency. The result is an increased incidence of arterial thromboembolic disease in smokers with a decreased in the incidence of deep vein thrombosis in comparison with non-smokers as has been reported.[2] Chronic hypoxia to the cardiac muscle and the increase in incidence of thromboembolic disease causes smokers to be at a 70% greater risk of coronary artery disease compared with non-smokers, and the postoperative mortality in smokers is higher than in non-smokers.[22] In 38% of vascular patients with cardiovascular problems, 80% are smokers.[23] However, one study reported that the risk of myocardial infarction was lower in past smokers of more than six months than current smokers, although the risk of severe angina was similar for current, past and non-smokers.[24]

Effects on respiratory system

Smoking-related pulmonary dysfunction is a major cause of pulmonary complications after surgical procedures. Chronic

cigarette smoking damages the ciliated epithelium and the tracheobronchial tree. This can lead to obstruction in the bronchioles and increases the chances of infection, which progressively leads to chronic obstructive pulmonary disease. Smoking also affects oxygen transport and delivery.[14] Irritants in smoke increase mucus secretions. The mucus becomes hyperviscous, with altered elasticity. Cilia become inactive and are destroyed by ciliotoxins. The result is impaired tracheobronchial clearance. Laryngeal and bronchial reactivity is increased. Cigarette smoke is known to disrupt the epithelial lining of the lung, causing an increase in pulmonary epithelial permeability. This loss of epithelial integrity allows irritants to penetrate the epithelium more easily and stimulate the subepithelial irritant receptors resulting in increased reactivity. Smoking leads to small-airway narrowing, causing an increased closing volume. Pulmonary surfactant is also decreased. These lead to small-airway disease. An increase in pulmonary proteolytic enzymes or elastolytic enzymes causes loss of elastic lung recoil and emphysema. Lung infections are increased.[14]

Furthermore, the half life of COHb depends chiefly on pulmonary ventilation. At rest, the half life is about 4–6 h. With strenuous exercise, due to rapid breathing, it is decreased to 1 h. During sleep, when the breathing is slow, its half life is prolonged to about 10–12 h. If one breathes 100% oxygen, its half life is reduced to 40–80 min and with hyperbaric oxygen therapy is further reduced to 23 min. Thus, on advising patients before anesthesia, these variations should be noted. During the day time, abstinence for 12 h is sufficient to get rid of CO. If an operation is scheduled for the next morning, the patient should not smoke the previous evening.[14]

Twenty-five percent of smokers suffer from chronic bronchitis occurring five times more often than in non-smokers.[14] The incidence of chronic obstructive airway disease is also higher than in non-smokers. When pulmonary function tests are done, chronic smokers with chronic obstructive airway disease show an obstructive pattern. In asymptomatic smokers, the spirometric pulmonary function tests are normal. However, in asymptomatic smokers, closing volumes are significantly increased, exhibiting small-airway disease.[14]

Chalon et al.[25] looked at the correlation of cytological damage from smoking and postoperative pulmonary complication. He found a steady rise in the percentage of complications after operations as cigarette smoking increased, from 7.9% in non-smokers to 43% in heavy smokers. In a large survey, the authors compared specific respiratory events such as reintubation, laryngospasm, bronchospasm, aspiration, hypoventilation and hypoxemia during anesthesia in smokers and non-smokers.[14] The incidence was found to be 5.5% in smokers, compared with 3.3% in non-smokers. The authors calculated the relative risk of these events occurring during anesthesia and found that the risk in all smokers was 1.8 times than in non-smokers. In young smokers it was 2.3 times, and in obese smokers it was 6.3 times the normal. It was also reported that smokers have a significantly high risk of having bronchospasm during anesthesia. The risk was higher in female smokers and it also was 25.7 times normal in young smokers with chronic bronchitis.[14]

Fletcher[26] also found that the difference between the partial pressure of arterial carbon dioxide (CO_2) and end tidal carbon dioxide ($PaCO_2$–$EtCO_2$) in non-smokers was 0.3 kPa while in smokers it was 0.9 kPa. He also found that this difference increased with age in smokers but not in non-smokers. It increased by 0.2 kPa per decade. Thus, a 60-year-old smoker will need 25% more minute ventilation than a non-smoker to maintain a given partial pressure of arterial CO_2. Furthermore, postoperative pulmonary complications were reported by Wellman and Smith[27] to be two times higher in smokers compared with non-smokers following abdominal and thoracic surgery while Bluman et al.[6] found it to be four times that of non-smokers.

Effects on gastrointestinal system

Cigarette smoking does not increase either the volume or the acidity of gastric juices.[28] Smoking make the gastroesophageal sphincter incompetent, which allows reflux, with accompanying risks of pulmonary aspiration. The short-lived reduction in the tone of the lower oesophageal sphincter is reversible within minutes after termination of smoking. While the emptying of liquid gastric juices is not influenced by smoking, there is a certain delay in the propulgation of solid food.

Effects on renal system and immune system

Smoking causes and worsens renal damage in those with medical problems affecting kidneys, e.g. diabetes mellitus or hypertension. Urinary tract problems are more common among smokers, because tobacco components in urine irritate urinary tract, causing frequent micturation and nocturnal dysuria. Tobacco smoking results in an increased secretion of antidiuretic hormone (ADH) leading to dilutional hyponatremia.[29]

Smoking impairs the immune response increasing the risk of infections and neoplasia.[1] Anesthesia further impairs the immune response, leading to a compounded detrimental effect on the immune system. Smoking interferes with cell mediated immunity and makes smokers prone to gum infections and periodontal diseases.[30]

Effects on postoperative pain, analgesic drugs, nausea, and vomiting

The incidence of postoperative nausea and vomiting is less in smokers compared with non-smokers.[31] It is suggested that this may be due to an antiemetic in the constituents of smoke.[31]

Cigarette smoke, which serves as a nicotine delivery vehicle in humans, produces profound changes in physiology. Experimental studies suggest that nicotine has analgesic properties. However, epidemiological evidence shows that tobacco smoking is a risk factor for chronic pain.[32] The complex relationship between smoking and pain has clinical relevance in the practice of anesthesiology and pain medicine. Thus, independently of the action on the analgesic drugs, chronic smokers require more analgesics for pain control. Fentanyl and pentazocine are metabolized quicker in smokers.[32]

Morphine and meperidine have been metabolized quicker in rats in the presence of nicotine.[32] However, in humans, mean total meperidine clearance has been shown to be not significantly different between smokers and non-smokers. There is no effect on codeine or paracetamol while phenylbutazone metabolism is increased.[32]

Epidemiological studies have suggested that tobacco smoking may be associated with painful musculoskeletal disorders.[33] Smokers are also more likely to use analgesic medications than people who have never smoked.[34] In a recent study by Weingarten et al.,[35] it was reported that current tobacco use among survey respondents was associated with greater pain intensity as measured by all brief pain inventory (BPI) intensity scales. Studies of different patient populations had also demonstrated that smoking was associated with greater pain intensity. Population-based studies in Great Britain, Norway, and Germany also found that tobacco use was associated with more severe symptoms from musculoskeletal disorders while several epidemiological studies suggest that there is a higher prevalence of chronic pain in former smokers, who were more likely to use analgesic medications than never smokers.[33,36] Several mechanisms have been suggested to explain this association between the intensity of pain in chronic pain states and smoking status. Smoking has been associated with alterations of the levels of neuropeptides that play a role in chronic pain states.[37] Smokers also have lower plasma beta-endorphin levels than non-smokers.[37] In contrast, in subjects without chronic pain, in experimental settings nicotine has antinociceptive effects in response to electrical, cold pressor, thermal, and ischemic pain stimuli.[37]

Effects of smoking on anesthesia

Hans et al.[38] established that the incidence and the severity of coughing during emergence are significantly higher in smokers than in non-smokers. It was observed that 7/7 of patients with a history of smoking whose anesthesia was maintained with sevoflurane exhibited a grade 2 or 3 cough upon emergence, whereas the same cough grade was observed in 6/9 smokers who received propofol. In addition, logistic regression demonstrated that sevoflurane in smokers was associated with a 100% probability of coughing at extubation, whatever its residual concentration, and that at a same residual propofol concentration at extubation, smokers aware at increased risk of coughing compared with non-smokers. These elements further support the assertion that tobacco smoking is associated with an increased risk of respiratory complications compared with non-smoking.[39] Another study comparing airway responses during desflurane vs. sevoflurane administration reported that cigarette smoking, but not the choice of the halogenated anesthetic, placed patients at risk of respiratory complications including coughing.[40] In contrast, in a study investigating cough during emergence from isoflurane anesthesia, smokers were not more likely to cough than non-smokers.[41] In a study by Dennis et al.[42] adverse effects such as coughing, breath holding, and laryngospasm during induction were significantly higher in smokers and passive smokers than in young smokers.

The reports on the effect of smoking on individual neuromuscular blocking drugs vary. Teiria et al.[43] found that 95% of effective dose for vecuronium was higher in smokers. Smokers also needed more frequent doses to maintain neuromuscular block, indicating an increased requirement at the receptor site. The reason is possibly because the smokers stopped smoking about 6 h before surgical operation. Latorre et al.[44] found that, in the case of rocuronium, there was no difference in onset of block or recovery times with the same dose used in smokers and non-smokers. However, Rautoma and Svartling[45] have found that the effective dose of rocuronium was the same as in non-smokers but the maintenance dose was higher, indicating a higher metabolism of the drug in smokers. Puura et al.[46] found even different results with atracurium. Smoking had no effect on neuromuscular blockade. However, in smokers who abstained from smoking for greater than 10 h, recovery was prolonged and the maintenance doses required were smaller.

Wound infection

Previous reports have confirmed that tobacco smoking is associated with surgical-site infections and dehiscence of tissue and wounds.[39] Several pathogenetic mechanisms may be involved. Microvascular disease and severe lung disease due to smoking are known to cause peripheral tissue hypoxia which increases the risk of wound infection and dehiscence.[47] In addition, some studies suggest that smoking and hypoxia reduce collagen synthesis and oxidative, killing mechanisms of neutrophils.[48]

Several reports have found that tobacco smoking impairs wound healing and promotes dehiscence, flap loss, necrosis, skin loss, epidermolysis, and occlusion of autogenous vein bypass grafts, but does not increase morbidity in women after fourth-degree perineal repair.[20,49,50] These facts may imply that smoking increases the risk of surgical-site infection (SSI). Thus, several reports have found that smoking increases the risk of sternal incision infections, fistula in mandibular osteotomy, and wound infections after spine surgery, breast surgery, surgery of fractures of the calcaneus, and lymph node dissection.[51,52] Most of these reports involved studies of clean operations. Other studies of clean operations have failed to identify smoking as a determinant of wound infection in intra-articular fractures of the calcaneus, cardiovascular surgery, breast surgery, spine surgery, and joint replacement.[53,54] Several studies of patients undergoing general surgery procedures and with a high proportion of clean-contaminated or contaminated wounds conclude that tobacco smoking does not increase the risk of SSI.[55]

Length of hospitalization

If smoking increases the risk of lower respiratory tract infection, and past smoking slightly raises the risk of all sites of nosocomial infection, one might expect a longer stay for smokers. Tobacco smokers are hospitalized more frequently than non-smokers[56,57] and several studies have reported that the hospital stay of smokers is longer.[58,59] However, some

studies of different types of patients (urology, coronary artery bypass, and ambulatory surgery) did not support this expectation.[60,61] On the other hand, intensive care unit (ICU) stay is tended to be longer for smokers as found in some reports on medical and cardiovascular surgery patients.[59,62] The role of tobacco smoking in adverse effects during hospitalization is not yet clear.

Mortality

One study revealed that there was no significant difference in the risk of death after orthopaedic surgery for patients who stopped smoking, continued to smoke or were non-smokers at follow-up although the number of events was low.[24] Another study stated that after controlling for confounding variables, patients who stopped smoking for more than six months preoperatively had a significantly reduced mortality risk when compared to patients who continued to smoke before and after surgery.[6] Although it is well established that smoking increases the mortality of patients with oesophagus cancer and those undergoing cardiovascular surgery, there is no report analyzing smoking and mortality in general surgery.[63,64]

Smoking cessation

Hypothetically, cessation less than 3–4 weeks before surgery may benefit postoperative recovery. Preoperative intervention to encourage cessation of smoking even a few days before surgery might benefit many patients for whom the time from diagnosis to operation is short. Interventions for cessation of smoking before surgery, seems to reduce the risk of postoperative complications.[8,65] Abstinence from smoking in relation to surgery might further motivate long-term cessation but also long-term health.

Following smoking cessation, ciliary activity starts to recover within 4–6 days. The sputum volume takes 2–6 weeks to return to normal. There is some improvement in tracheobronchial clearance after 3 months. It takes 5–10 days for laryngeal and bronchial reactivity to settle. There is improvement in small-airway narrowing after four weeks, and marked improvement is seen after six months.

Some studies reveal that the risk or incidence of overall postoperative complications in past smokers is significantly lower than current smokers or that there is no significant difference between past smokers and non-smokers.[7,24,39,66,67] One study revealed that there were significantly higher complications in patients who stopped smoking more than four weeks before breast reconstruction surgery in comparison to non-smokers, although there was no significant difference in postoperative complications between past smokers and current smokers.[68] One study reported that there was no significant difference according to respiratory complications and hospital readmission due to pneumonia between current smokers and patients who had stopped smoking before surgery and non-smokers.[6] One study also revealed that there were no significant differences in the incidence of postoperative complications between current smokers, past smokers, and non-smokers in minor surgery[7] and another study between current and past smokers in hip or knee arthoplasty surgery.[70]

On the other hand, two studies revealed that patients who stopped smoking within two months of surgery had an increased risk of complications when compared with patients who stopped smoking more than two months preoperatively.[69,71] Some studies also stated that current smokers had a significantly higher risk of overall complications than non-smokers.[66,68,70]

Only one study looked at the effect of smoking cessation on sputum levels and respiratory complications in patients undergoing minor surgery.[7] It was stated that past smokers and current smokers had an increased risk of increased sputum than non-smokers, although the risk decreased to the same sputum volume as current smokers, for smokers who stopped smoking more than two weeks before minor surgery. No studies looked at the effect of smoking cessation on sputum levels in major surgical procedures.

However, the minimum duration of abstinence necessary to confer benefit remains unknown. Two clinical studies have reported that cessation of smoking more than three weeks before operation reduced the occurrence of wound healing complications in relation to head and neck and breast reduction surgery.[72,73] Similarly, an experimental trial has reported that four weeks of abstinence from smoking reduced the frequency of wound infections in healthy smokers to the frequency in healthy non-smokers.[74]

Clinically, Warner et al.[75] found a 3-fold decrease in the incidence of pulmonary complications after eight weeks of smoking cessation. Current data also showed an increase in pulmonary complications if patients stop smoking less than eight weeks before surgery. For those who stopped smoking less than eight weeks before surgery, those who stopped closest to surgery were more likely to experience pulmonary complications.[8,76] More recently, Nakagawa et al.[77] showed that smoking abstinence of at least four weeks was necessary to reduce the incidence of pulmonary complications. In this study, the authors separated patients as current smokers, abstinent for 2–4 weeks, and abstinent for over four weeks. In addition, Kotani et al.[78] showed that smokers had a decrease ability to mount effective pulmonary immune defenses that may last for as long as six months after stopping cigarette use.

One study revealed that patients who stopped smoking less than four weeks before orthopaedic surgery had a lower incidence of all pulmonary complications than patients who continued to smoke; although a low number of major pulmonary events occurred.[6] Another study reported that there was no significant difference in the incidence of pulmonary embolism between patients who stopped smoking for less than two months, more than two months before lung cancer surgery, current smokers and non-smokers.[69] A further study found that there was no significant increase in pulmonary complications in patients who stopped smoking four weeks before surgery in comparison to non-smokers.[67] Finally, Myles et al.[39] supported that patients with more severe illness are more likely to reduce their consumption or stop smoking.

Comments

Tobacco smoking is a leading cause of death in Western countries.[79] The evolution of tobacco use within societies can be conceptualized as an epidemic.[80] Its prevalence increases rapidly as manufactured cigarettes are intensively marketed, first among men, then among women. The peak prevalence in most societies exceeds 50% in males. Despite this, a quarter of patients undergoing surgery continue to smoke up to, and after surgery, with advice on smoking cessation varying from surgeon to surgeon.[81–83] However, only 58% of surgeons and 30% of anesthetists routinely advise patients to stop smoking before undergoing a surgical procedure.[83]

Current smoking is less frequent among patients with cancer, chronic obstructive pulmonary disease, and a higher ASA score; this probably means that these patients had been forced to quit smoking more frequently. The presence of cancer, chronic obstructive pulmonary disease, and a higher ASA score can make the preoperative stay and surgical time longer. Therefore, and according to these facts, an increased risk of nosocomial infection in past smokers can be expected.

It is widely documented that stopping smoking before surgery has substantial health benefits in the longer term and should be recommended to every smoker in order for them to gain maximum benefit from their treatment. Patients who continue to smoke before surgery have a higher rate of postoperative complications than non-smokers. However, identification of the optimal period of preoperative smoking cessation on postoperative complications could not be determined. It should also be considered that even a period of temporary short-term abstinence before surgery may also increase patients' confidence in their ability to quit smoking and may lead to a longer term quit attempt in the future. It is also seems to be a benefit from even postoperative smoking cessation on longer-term health outcomes in patients who have undergone a surgical procedure. Furthermore, the duration of preoperative abstinence needed to affect postoperative outcomes is often unclear.

Recent evidence suggests that surgery represents an excellent opportunity to intervene in patients who smoke, with potential benefits to both immediate surgical outcomes and long-term health.[8,75] Due to the fact that many hospitals become smoke-free environments and the availability of effective interventions to help people to stop smoking, the preoperative and postoperative period is an ideal time to help smokers to quit before being admitted to hospital. Helping patients to stop smoking before surgery may also enhance their surgical outcome and thus help them to get the maximum benefit from their healthcare. A reduction in tobacco consumption during hospitalization is also occurred due to surveillance by healthcare personnel and especially in countries where smoking has been prohibited by law for patients, visitors and healthcare personnel.

Furthermore, pharmacotherapy is an important element of strategies to help smokers to stop smoking.[84] The use of these medications approximately doubles the rate of abstinence. Nicotine derived from tobacco use can be replaced using several different delivery systems, including gum, inhalers, nasal spray, patches, and lozenges. All systems are effective in promoting cessation.

Because physicians have an important role in these efforts, it is important to assess their practices and attitudes if effective intervention strategies are to be implemented in surgical settings. Educating surgical subspecialists is only one component of a strategy to provide tobacco interventions to surgical patients. There is strong evidence that provider education, combined with health system changes such as provider reminder systems, are highly effective in increasing the rates of intervention and cessation. Ideally, interventions performed by surgeons and anesthesiologists should be one component of a comprehensive approach that includes collaborations with other healthcare providers. Anesthesiologists should advise smokers to stop smoking during the postoperative ward rounds. This should be done especially in those patients who had smoking-related problems during anesthesia. However, if such health system changes are to be implemented, surgical specialists must understand the relevance to their practice and must be enthusiastic proponents of and participants in the system change.

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