Smoking and Anesthesia: Preoperative Abstinence and Perioperative Morbidity


Contents

Cardiovascular System
Respiratory System
  Mucus Secretion and Clearance
  Small Airway Narrowing
Immune System
Hemostasis
Drug Metabolism
Patient Psychology
Postoperative Analgesic Requirements

Smoking continues to be a cause of increased perioperative morbidity.1 Over 1,000 components of cigarette smoke have been identified2 with wide-ranging effects on the cardiovascular, respiratory, and immune systems, hemostasis, drug metabolism, and patient psychology.

Patients who are seen in surgical outpatient clinics before elective surgery should be informed of this increased risk, so that they can stop smoking before admission. However, there are patients who are unwilling or unable to heed this advice. This group of patients poses two broad questions. First, how serious the consequences are of continuing to smoke before operation, and, second, what period of abstinence will provide some degree of amelioration from them.

In an attempt to answer these questions, we review the wide-ranging pathophysiologic and psychologic effects of smoking and critically assess available evidence on the effects of a period of smoking abstinence.

Cardiovascular System

The components of smoke that have major effects on the cardiovascular system are carbon monoxide and nicotine.3 Mainstream cigarette smoke (that drawn through the cigarette into the mouth) contains 1–5% carbon monoxide (CO), which, when diluted in the oropharynx, is inhaled in concentrations of about 400 ppm. The affinity of hemoglobin for CO is more than 200 times greater than for oxygen,4,5 so that the low alveolar concentration of CO found in smokers produces significant carboxyhemoglobin (COHb) levels. The degree of COHb depends on various factors, such as the brand of cigarette smoked, the depth of inhalation, the number of puffs per cigarette, the pattern of smoking during the day, the degree of atmospheric CO pollution, and the level of exercise or pulmonary ventilation while smoking.6 Endogenous production of CO (from hemoglobin metabolism) and average atmospheric CO pollution account for a COHb level of up to 2.5% in nonsmokers, whereas the level of COHb in smokers usually ranges from 3 to 15%. Because of the factors mentioned, different smokers consuming the same number of cigarettes per day may have widely differing COHb levels, for instance, 3–11% for 20 cigarettes/day.7

The two major effects of COHb are a decrease in the amount of Hb available for combination with O2 producing an absolute decrease in oxygen content and a shift of the O2 dissociation curve to the left producing an increased affinity of hemoglobin for oxygen.8 These effects summate to decrease oxygen supply at tissue level. Asmussen and Chiodi9 suggest that the main response to tissue hypoxia induced by COHb, at rest, is tissue oxygen extraction to a lower venous oxygen tension, and this has been confirmed.10 Thus, the myocardium, because of its normally high O2 extraction ratio, is particularly sensitive to even low levels of COHb.
Studies in patients without coronary artery disease undergoing cardiac catheterization show that acute elevations of COHb to 5–10% produce an increased coronary blood flow, a decrease in coronary arteriovenous oxygen difference, and an insignificant decrease in coronary sinus oxygen tension. In patients with coronary artery disease, however, an increase in coronary blood flow is not seen and coronary sinus oxygen tension decreases significantly. In addition, alterations in pyruvate/lactate metabolism may be seen, suggesting the development of areas of myocardial ischemia. The time of onset of exercise-induced angina is reduced by levels of COHb as low as 4.5%. CO may induce alterations in myocardial electrophysiology. This has been observed in monkeys with lowering of the threshold for inducing ventricular fibrillation after myocardial infarction. In humans, 5–9% COHb was associated with the development of abnormal electrocardiograms in seven out of 26 persons, with arrhythmias occurring in two of the seven. Kaul et al. on the other hand, were unable to detect any increased vulnerability of the ventricles to drug-induced arrhythmias, even with 35% COHb.

Carbon monoxide also exerts a negative inotropic effect. This may be due in part to CO binding to cytochrome oxidase (cytochrome a and a3) and myoglobin, interfering with mitochondrial function and muscle contraction. Myoglobin functions as a reservoir of oxygen for muscle metabolism and reacts with CO to form carboxymyoglobin (COMb). Large amounts of extravascular CO are stored in muscle, and measurement of COMb/COHb levels suggest that with a blood COHb level of 10%, 30% of cardiac myoglobin will be saturated. It can be predicted from an analysis of the variables influencing the COMb/COHb ratio that capillary, and therefore tissue, hypoxemia will promote COMb formation. This may be particularly important in local areas of myocardial ischemia, causing further depression of ventricular function.

Although acute exposure of Hb to CO decreases the oxygen content of blood, smokers compensate for the chronic tissue hypoxia by an increase in red blood cell mass and so oxygen content in smokers may well be the same as for nonsmokers. However, this is only achieved at the expense of an increase in blood viscosity with its adverse effect on cardiovascular performance and tissue perfusion. Most of the observed polycythemia seen in smokers is hypoxemia induced, although CO appears to cause a loss of plasma volume. On cessation of smoking, the hematocrit falls, often within a few days.

 Virtually all the inhaled CO is excreted unchanged via the lungs. COHb dissociates according to the equation known as Haldane's first law,

\[
\frac{\text{COHb}}{\text{HbO}_2} = M \cdot \frac{P_{\text{CO}}}{P_{\text{O}_2}}
\]

where M is the affinity constant and P_{\text{CO}} and P_{\text{O}_2} are partial pressures of carbon monoxide and oxygen, respectively. When this equation is applied at the alveolar/pulmonary capillary level, it can be seen that the major factors favoring COHb dissociation and CO elimination are an increase in inspired oxygen partial pressure and an increase in alveolar ventilation, the latter mainly by reducing P_{\text{A CO}}. Impairment of the diffusing capacity of the lung (D_{\text{L CO}}) may be a small but significant factor in some subjects. Lawther and Commins found a half-life of elimination, in nonsmokers with 10% COHb saturation, to be about 4 h. This agrees with other studies where the subjects are at rest while the determinations are made. The effect of increasing alveolar ventilation via exercise on COHb half-life has been calculated and shows a decrease from 4 h while sedentary to about 1 h during strenuous exercise, e.g., football. At low levels of activity—sleeping/studying/typing—the rate of decay increases markedly for small increases in activity. During sleep, the half-life doubles and values of 10–11 h have been reported. Breathing 100% O2 decreases the half-life to 40–80 min and oxygen under hyperbaric conditions reduces it further to about 23 min.

The recommendations for a smoking-free period before anesthesia therefore should be of the order of at least three half-lives. Conroy bases his recommendation of 72 h on one patient in whom it took 19 h for the COHb to fall from 17.9% to 5.0%. This time period seems inappropriately long when compared with a normally reported half-life of approximately 4 h. During the day, a period of 12–18 h (3 COHb half lives) should produce a profound drop in COHb levels. This time period also allows return of the oxygen dissociation curve toward the normal position. A maximum period of 48 h would be expected to be sufficient time for the COHb of all smokers to fall to a nonsmoker's level and produce a rise in oxygen content and availability. This has been demonstrated in pregnant women, in whom smoking abstinence for 48 h produced an 8% increase in available oxygen. The initial mean COHb was 5%. It must be noted, however, that in this study the beneficial change in P_{\text{aO2}} on stopping smoking was thought to be due to pH change rather than to a reduction in COHb levels. The patients most at risk of undergoing anesthesia with significant COHb levels are those who smoke avidly just before retiring to bed, with operations scheduled for the morning, because of the increased half-life associated with sleeping.

Nicotine has profound dose-related effects on the
cardiovascular system and, in blood levels found in smokers (15–50 ng/ml), acts through the sympathoadrenergic system to cause an increase in heart rate, systolic and diastolic blood pressure, and peripheral vasoconstriction.29 Nicotine acts at several sites to produce this pressor response; on the carotid body and aortic chemoreceptors,30 at autonomic ganglia,31 and by causing release of catecholamines from the adrenal medulla and other chromaffin tissue. Increased plasma norepinephrine, epinephrine, growth hormone, and cortisol levels have been demonstrated in smokers.29 In addition, nicotine, as well as CO, alters myocardial electrophysiology,33 lowering the threshold for ventricular fibrillation during an episode of myocardial ischemia in dogs.34 Nicotine is metabolized in the liver, lung, and kidney and both nicotine and the major metabolites, cotinine and nicotine-N-oxide, are rapidly eliminated by the kidney especially in urine of low pH.35 The half-life of nicotine after inhalation or parenteral administration is 30–60 min. Cigarette abstinence is followed by a reduction in pulse rate and blood pressure, peripheral redistribution of body temperature, and decreased catecholamine levels. The pressor response after smoking one cigarette abates after 20–30 min.16,29

Short periods of abstinence are associated with an improvement in cardiovascular fitness when maximal exercise is considered.36 Rode and Shephard37 found that 1 day’s abstinence in six cigarette smokers was associated with a 13–19% decrease in the oxygen cost of breathing after strenuous treadmill exercise. Seppänen38 studied 14 smokers (average 20 cigarettes/day) with mean COHb levels of 9.8% while smoking. Abstinence for at least 12 h (mean COHb 3.1%) improved the physical work capacity at heart rates of 150, 150, and 170 beats/min by 10–20%.

In summary, therefore, the major cardiovascular effects are a pressor response increasing myocardial oxygen consumption due to nicotine and a decrease in oxygen supply due to CO. Short-term abstinence from smoking produces effects in cardiovascular physiology that should be of benefit to patients undergoing anesthesia and operation.

Respiratory System

The respiratory effects of smoking are diverse. An increase in postoperative respiratory morbidity (PORM) was demonstrated by Morton,39 in 1944, who showed that patients who smoked more than 10 cigarettes/day had a sixfold increase in postoperative chest complications. Other studies have confirmed these findings, although the magnitude of increased risk varies according to the definition of PORM. In a prospective study, Wightman,40 in 1968, showed a PORM rate of 14.8% in smokers and 6.3% in nonsmokers following all abdominal operations. PORM criteria were T 99°F, productive cough and physical signs in the chest. Laszlo et al.44 studied a group of patients without chronic bronchitis and showed a PORM rate of 53% for smokers and 22% for nonsmokers after all types of surgery. Postoperative respiratory morbidity was subdivided into three categories on the criteria of 1) altered sputum volume and purulence (bronchitis), 2) chest x-ray changes (collapse), or 3) both (pneumonia). The type of PORM with the most marked difference between smokers and nonsmokers was "bronchitis" and the three patients who developed severe postoperative "bronchitis" after minor surgery were all heavy smokers (>20 cigarettes per day). He found no difference in PORM rate overall between light (<20 cigarettes/day) and heavy smokers. Chalon et al.42 prospectively studied 111 patients classified as light (1–9), moderate (10–19), heavy (20–29), and very heavy (>30 cigarettes/day) smokers. PORM rates were 9%, 23%, 29%, and 43%, respectively, whereas the rate was 7.9% in nonsmokers.

There are three major mechanisms by which smoking may increase PORM—mucus hypersecretion, impairment of tracheobronchial clearance, and small airway narrowing.

Mucus Secretion and Clearance

Mucus clearance is a complex process that requires ciliary activity and mucus with certain rheologic characteristics to provide coupling between ciliary beat and transport.43 Smoking may interfere either with ciliary activity or respiratory tract mucus to impair tracheobronchial clearance. Several components of cigarette smoke are cilistatic when tested against a wide variety of ciliated systems in vitro, including respiratory epithelium. Mendenhall and Shreeve44 demonstrated reduced carmine particle transit time along the mucosa of calf tracheas when exposed to cigarette smoke. Hilding,45 utilizing suspensions of epithelial scrapings with direct observation of ciliary activity, demonstrated tobacco-induced cilistasis. In experiments with opened rat tracheas, absolute cessation or gross slowing of ciliary beating followed insufflation of cigarette smoke over the trachea.46 It is possible to criticize in vitro studies on grounds that the concentration of cigarette smoke may be much higher than that found in usual smoking and the bathing solution for the tracheal preparation may influence ciliary beating. However, in vivo studies with cats using high-speed filming and a "normal" pattern of smoking confirmed Dalhamm’s earlier in vitro work. 47

The coupling between ciliary activity and ciliary transport must be by a substance with well-defined rheologic properties but does not necessarily have to be mucus. Other macromolecular systems with some degree
of cross-linking are transported by in vitro ciliated preparations, e.g., excised frog palate. The property of viscoelasticity is essential, however. Alteration in the viscous or elastic component will alter mucus transport, although it is difficult experimentally to alter only one component. Using reconstituted mucus with varying concentrations of mucus solids, Shih et al. demonstrated that transport was related to concentration of mucus solids, the transport rate decreasing at concentrations greater and less than optimal concentration. Shih concluded that mucus solutions with zero or small elastic modulus were not transported. There was a sharp increase in transport with increase in elastic modulus up to a certain point and then a slow decrease in transport with increasing modulus thereafter. However, even with the highest mucin concentrations tested, absolute clearance rate was such that good clearance still would take place in a normal ciliated system. Using an analytic model, Ross and Corssin observed that moderate changes in mucus viscosity did not affect the transport rate and that, at constant viscosity, increasing elastic modulus led to increasing mucus transport.

Although the mucin composition of smokers and nonsmokers has been shown to be different, studies on the rheologic properties of mucus from asymptomatic smokers, nonsmokers, and following cessation of smoking have, not to our knowledge, been carried out. Thus, it would be difficult to predict what changes in mucociliary clearance might occur with, or giving up, smoking. Measurement of tracheobronchial clearance rates in man have provided conflicting results. Pavia et al. demonstrated no long-term impairment of mucociliary clearance due to cigarette smoking, although a temporary slowing was demonstrated later from the same center. The inhaled particle size was 5 μm. Using 2 μm labeled iron oxide particles, Laurenco showed tracheobronchial clearance was delayed for periods of 1–4 h after inhalation in smokers. Goodman et al. found that tracheal mucus velocity was within normal limits in just under 40% of smokers but markedly depressed in the remainder, notably in patients with clinical simple and obstructive chronic bronchitis but also in young asymptomatic smokers with no spirometric evidence of small airway disease. Nine young ex-smokers, who had given up for at least 7 months before the study, were studied. Five had mucus velocities within the normal range, one was slightly depressed, and three still had markedly depressed mucus velocities. A control group of 10 young nonsmokers all demonstrated normal mucus velocities.

Circumstantial evidence from smoking cessation clinics suggests that the volume of sputum declines over a 6-week period, with perhaps 50% reduction over the first 2 weeks. Some patients report a transient increase in sputum volume in the few days after stopping smoking. This usually is thought to be due to the return of ciliary activity, based on animal studies that demonstrate that smoke-induced ciliostasis may be reversible. Other patients report difficulty with expectoration. Whether this is due to alterations in rheologic characteristics of sputum or lack of respiratory tract irritation from smoke has not been elucidated fully.

In summary, current evidence suggests that chronic cigarette smoking generally leads to a decrease in mucociliary transport. A period of smoking abstinence of several months is associated with return of mucociliary clearance to normal values in a substantial number of smokers, but clearance rates may remain abnormal for many months. The effect of a few days abstinence on mucociliary clearance has not been well documented, although one study found no change in clearance rates after 1 week.

**Small Airway Narrowing**

The third component contributing to PORM is small airway narrowing. A great deal of research has been undertaken in an effort to define the earliest changes in pulmonary pathophysiology due to smoking and whether these are reversible. An early study on 10 young (25–33 yr) smokers before and after three weeks abstinence showed a significant improvement in peak flow (570 ± 58–610 ± 1/min) but no significant change in any other measured parameter. Six subjects were studied after a further 3 weeks of abstinence when there were significant improvements, compared with control values, in inspiratory reserve volume, functional residual capacity, maximal voluntary ventilation, and airway conductance divided by FRC. It is of interest that the initial improvement in peak flow was not sustained—at 6 weeks the value was 575 ± 60 L/min.

Other later studies have looked in greater detail at small airway disease and have shown that smokers with normal gross spirometry—FEV1, VC, etc., still may have significant small airway disease. Tockman et al. compared male, age-matched smokers and nonsmokers. TLC and VC decreased significantly with age, but there was no significant difference between the two groups. There were, however, significant differences in closing capacity and slope of Phase III of the single breath nitrogen test as well as FEV1/VC, RV/TLC, and diffusing capacity for CO(DiCO). Buist et al. studied smokers who attended a smoking cessation clinic after 1, 3, 6, and 12 months. There were no changes in FEV1 or VC throughout the year. The slope of the alveolar plateau phase III had improved at 1 month, and the CV/VC and CV/TLC had improved significantly after 6 months smoking cessation. Using the ratio of dynamic compliance at high respiratory rates to the static compliance as a
measure of small airway disease, 8 out of 12 smokers showed some improvement 2 months after stopping smoking.\textsuperscript{61}

Most studies have looked at improvements that occur after a minimum time of 4–6 weeks after smoking cessation. One study that looked at changes in small airway disease after a short period of abstinence (7 days) found no improvement at all.\textsuperscript{62} The conclusion is that smoking must be stopped for at least 4–6 weeks before any beneficial effect can be detected by respiratory function tests.

Clinical studies on whether short-term abstinence affects PORM rates are scant. Laszlo et al.\textsuperscript{41} includes a group of undefined ex-smokers who appear to have no reduction in PORM rate. Recently, Mitchell et al.\textsuperscript{1} have attempted to define the contribution of various risk factors to overall PORM rate. Following the presence of a postoperative nasogastric tube and upper abdominal surgery (the highest risk factors) was the presence of previous respiratory disease as judged by an observed productive cough, FEV\textsubscript{1} < 60% predicted and a history of purulent sputum on day of the operation. Cigarette smoking, per se, did not contribute further to PORM, suggesting that any contribution of smoking is through mucus hypersecretion and airway narrowing. Of these two, Mitchell et al. concluded that mucus hypersecretion was the more important. The PORM rate for ex-smokers was not determined specifically, but it was found that smokers who had stopped for more than 8 weeks before operation had a lower incidence (25%) of postoperative purulent sputum than those who stopped within 8 weeks of operation (50%).

Other effects of smoking on the respiratory system that must be considered are an increase in nonspecific bronchial reactivity,\textsuperscript{63} an increase in respiratory epithelial permeability,\textsuperscript{64} a paradoxical deterioration in some asthmatics on stopping smoking,\textsuperscript{65} and alterations in pulmonary surfactant.\textsuperscript{66} An increase in nonspecific bronchial reactivity in smokers, well-known to the anesthetist,\textsuperscript{25} can be demonstrated experimentally by a histamine challenge.\textsuperscript{63} The concentration of inhaled histamine required to reduce the specific airway conductance by 35% in smokers is less than 40% of that required in nonsmokers. The time required to decrease this effect of smoking has not been determined.

It has been known for sometime that cigarette smoke may disrupt the epithelial lining of the lung. This first was demonstrated in the guinea pig using horseradish peroxidase as the marker.\textsuperscript{57} Using diethylene triamine penta acetic acid (\textsuperscript{99m}TcDTPA) a similar increase in pulmonary epithelial permeability has been found in asymptomatic smokers.\textsuperscript{64} This is steadily reversible on smoking cessation, although about 7 days is necessary for the airway permeability to return toward normal. It has been suggested that the leaks develop around the corners of the epithelial cells,\textsuperscript{68} probably in the portion of the respiratory bronchioles covered by cuboidal epithelium (where there are the greatest number of corners per unit surface area). The alveoli and more proximal ciliated airways have fewer corners per unit surface area and are probably therefore less important in the genesis of leaky lungs from this effect. The implications of these findings have been discussed with reference to pulmonary inflammation and infection.\textsuperscript{69} The anesthetic implications are unclear.

One curious but noteworthy effect is the finding that cessation of smoking may cause a worsening of existing asthma or the declaration of asthma for the first time.\textsuperscript{68} The etiology of this is uncertain, although an immunologic basis has been suggested.

A low yield of surfactant after endobronchial lavage has been reported in smokers as compared with nonsmokers.\textsuperscript{65} In the subjects who stopped smoking, an increase in the surfactant level was seen after 2 weeks and reached nonsmokers' levels after 1 month's abstinence. Reservations about the interpretation of this study have been expressed.\textsuperscript{70}

**Immune System**

There is evidence from in vitro and animal work that components of cigarette smoke impair the immune response. In humans, increased white blood cell count,\textsuperscript{71} depressed neutrophil chemotaxis,\textsuperscript{72} decreased immunoglobulin levels and low natural killer cell activity,\textsuperscript{73} alterations in immunoregulatory T-cell activity,\textsuperscript{74} and increased IgE levels\textsuperscript{75} have been demonstrated in smokers. One of the components of local pulmonary defense mechanisms, the pulmonary alveolar macrophage (PAM), has been studied extensively. PAM from bronchial lavage are increased in number in smokers, show morphologic abnormalities,\textsuperscript{76} have altered metabolism, including increased secretion of lysozyme\textsuperscript{77} and superoxide anion,\textsuperscript{78} and show decreased adherence to nylon fibers.\textsuperscript{79} Although the relevance of these findings to perioperative morbidity has not been established, it seems likely that smoking will have a detrimental effect on host defense, especially when superimposed on the suppression of the immune response caused by anesthesia and operation.\textsuperscript{80} The time course of a return to normal has not been determined for all individual components, although the decreased adherence of PAM to nylon fibers had resolved after 1 week's abstinence and after 6 weeks' immunoregulatory T-cell activity had returned to normal in heavy smokers. It therefore would seem that a period of smoking cessation for 6 weeks is associated with evidence of a return to normal of some aspects of immune function.
Hemostasis

The incidence of arterial thromboembolic phenomena correlates very strongly with cigarette smoking and certain hematologic factors, e.g., decreased platelet survival time and increased platelet aggregability have been found in smokers. However, it is of interest that platelets taken from nonsmokers may even show decreased aggregability when subjected to cigarette smoke or CO, which raises the probability that cigarette smoke has an indirect action on platelet activity possibly through alterations in free fatty acid profile or through effects on vascular endothelium.

In contradistinction to arterial thrombosis, an increased incidence of venous thrombosis has not been demonstrated in heavy smokers. Two independent studies have demonstrated a lower incidence of deep vein thrombosis (DVT) postmyocardial infarction in smokers than in nonsmokers. Using 125I-fibrinogen, Handley et al. showed an incidence of DVT of 28% in smokers and 51% in nonsmokers up to 10 days after hospital admission. Marks and Emerson showed a more marked difference, 11% for smokers and 62% for nonsmokers. A decreased incidence of postoperative DVT after gynecologic surgery also has been reported. These results are open to several interpretations. It is not possible to differentiate from these studies whether the lower incidence of DVT in smokers is associated with changes in hemostatic/fibrinolytic mechanisms associated with either previous smoking or the sudden cessation of smoking. Sudden cessation of smoking, as in patients hospitalized after acute myocardial infarction, could produce a rebound hypocoagulability state, for example. Deprived smokers may be more restless and and move around more than nonsmokers, although one study found no evidence of this.

The nonsmoking and smoking populations who suffer myocardial infarction may differ in some other hematologic or vascular factor that may have a bearing on the incidence of DVT. Although an increase in fibrinolytic activity has been reported after acute smoking, the general consensus is that chronic smoking is associated with decreased fibrinolytic activity and also inhibition of prostacyclin formation. Although these two factors would suggest an increased incidence of DVT in smokers, available clinical evidence indicates that patients advised to stop smoking some weeks prior to operation may be at an increased risk of developing a DVT compared with those patients who continue to smoke until the time of surgery.

Drug Metabolism

Some components of tobacco smoke cause hepatic enzyme induction. Major evidence that enzyme induc-
not surprising that no clear answer to this problem emerges. What is quite clear is that admission to hospital, with its break to routine life, the emphasis on health and disease, the supportive atmosphere, and the relative difficulty or impossibility of finding a place to have a cigarette, is a prime opportunity for patients to stop smoking. The overall long-term (1 year) success rate in stopping smoking in people who attend smoking cessation clinics is usually disappointingly low (10–25%).87 The success rate of people admitted to hospital for medical reasons may well be higher, especially if advice is given during the admission.88 In view of the undoubted long-term gains from giving up smoking, this opportunity should not be squandered, unless convincing arguments for carrying on smoking preoperatively can be presented.

**Postoperative Analgesic Requirements**

Postoperative analgesic requirement shows a large interpatient variation. Smokers might be expected to show an increased requirement due to enhancement of drug metabolism and anxiety due to deprivation. Acute smoking raises the pain threshold99,100 and this, coupled with the finding that smokers may display a naloxone reversible fall in inspiratory flow rate after smoking,101 raises the possibility that smoking may have an effect on endogenous opioids. There has been, as yet, no confirmatory evidence in terms of raised cerebrospinal fluid (CSF) or plasma opioid levels following smoking—an important step in view of the misgivings about the specificity of naloxone.87

**Summary**

Much less is known of the effects of stopping smoking than of continuing to smoke, and many of the studies on smoking cessation are concerned with long-term effects rather than effects within 48 hr. Studies concerned with this period are required, especially in terms of postoperative respiratory morbidity, before an authoritative assessment can be made of the benefits and risks of stopping smoking in the short period before operation. Present studies are convincing that great benefit will accrue in the cardiovascular system, mainly from carbon monoxide and nicotine elimination, after 12–24 h. A few days may greatly improve ciliary beating and 1–2 weeks provide a significant reduction in sputum volume. However, a minimum period of 4–6 weeks would seem appropriate to greatly influence postoperative respiratory morbidity, although the statement that “one needs 4–6 weeks to influence postoperative respiratory morbidity” must not be misapplied and become “there is no point in giving up smoking unless it is 4–6 weeks prior to operation.” There are no proven disadvantages to the respiratory system from stopping smoking in the short term, and it seems unwise to sacrifice proven advantages for a theoretic consideration that sputum may become “stickier” and more difficult to clear. Less is known with regard to the time course of offset of smoking effects on drug metabolism and the immune system, although 6–8 weeks would be expected to produce some benefit. Positive benefits of continuing to smoke would seem to be a decreased incidence of DVT, although this must be confirmed on postoperative patients, the occasional patient who will start wheezing if he stops smoking and possibly some psychologic effects. We would contend that these effects can be reduced by nonsmoking means—anticoagulants, bronchodilators, and anxiolytics—and do not provide sufficient reason to continue smoking.

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