Cigarette Smoking

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Risk

- Smoking is the most common cause of preventable death. Half of cigarette smokers die of a smoking-related disease; on average, smokers lose at least 10 y of life expectancy.
- In USA, incidence of smoking is 17.8%: 42.1 million smokers (2013). Consumption in USA peaked in 1965 at 42%. Worldwide consumption still rising, with 5.8 trillion cigarettes smoked per y; fastest consumption growth is in China.
- Native Americans/Alaskan Natives have highest rate of smoking in USA at 26% followed by African Americans at 25.5%.
- Frequency increased with a lower level of educational attainment (24.2% without high school diploma; 41.4% with GED; 5.6% of those with a graduate degree) and poverty; true even in low- to middle-income countries.
- Male:female ratio: 4:3, with young women the fastest-growing group.

Perioperative Risks

- Increased risk of CAD × 2 that of nonsmokers of the same age
- Postop pulm complications up to × 6 that of nonsmokers
- COHb increased (up to 15%)
- Hyperreactive airway
- No increased risk of pulm aspiration
- Reduced risk of postop N/V
- Increased rate of death (odds ratio 1.63) and postop complications in elective surgery and major joint, spine, and neurosurgery

Worry About

- CAD, COPD, PVD, productive cough, and reactive airways
- Increases physiologic age by 8 y (30 packs per y) relative to nonsmokers
- Decreased tolerance to pain, requiring increased doses of analgesics
- Increased rate of postoperative delirium
- Pediatric passive smoking and reactive airways and increased rate of SIDS

Overview

- Addictive habit: Cigarette smoke contains >4000 identifiable constituents, many of which are pharmacologically active, toxic, or have tumorigenic effects. Acute effects relate to CO and nicotine.
- 90% of tobacco smoke is gaseous, consisting of nitrogen, O_2, and carbon monoxide along with gaseous irritants and carbon monoxide. Particulate matter consists of nicotine, tar, and other volatile organics.
- Nicotine stimulates the sympathetic ganglia, causing release of catecholamines from the adrenal medulla and sympathetic nerve endings, increasing BP, HR, and SVR, that persists for 30 min after one cigarette.
- Associated with decreased MAO and increased dopamine levels in the brain.
- Inhaled CO produces up to 5–15% COHb, compared with 0.3–1.6% in nonsmokers. Combined effects of nicotine and COHb put diseased myocardium at risk.
- Irritates the pulm system, increasing mucus production while decreasing ciliary activity and mucus flow, markedly impairing tracheobronchial secretion clearance.
- Chronic use associated with CAD, Htn, COPD, peripheral vascular disease, and numerous cancers.
- Smoking also increases all blood cell lines, platelet reactivity, and fibrinogen.
- Cessation for 3–4 hours results in insignificant hemodynamic side effects from nicotine, and it improves myocardial O_2 supply to demand.
- Cessation of smoking the night before surgery will reduce the COHb and nicotine levels to that of nonsmokers. Cessation 4–6 d will result in a return of ciliary activity.
- Cessation for less than 4 wk has same rate of respiratory and wound healing complications as found in active smokers (OR 1.2); smokers should stop at least 4 wk before surgery.
- Cessation for 2 y reduces risk of MI to that of the nonsmoking population.
- Smoking is the cause of 1 of every 5 deaths in USA and is the leading cause of preventable mortality (480,000 preventable deaths/y).

Etiology

- Habituation and addiction

Usual Treatment

- Nicotine patch and clonidine, varenicline, bupropion, Smokers Anonymous, or self-withdrawal

Treatment

- Cessation for a minimum of 12–24 h decreases COHb and nicotine levels.
- Cessation for ≥4 wk will reduce postop pulm complications.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Oral, pharyngeal, and head and neck cancers		Lesions on exam or intubation	Usually not needed
CV	Increased HR, SVR, and coronary vascular resistance Myocardial ischemia Increased PVR Increased blood viscosity	Exercise tolerance, angina (see Coronary Artery Disease)	Two-flight walk	ECG
RESP	Increased COHb and COPD Decreased FEV_1FVC Increased secretion Decreased clearance Increased airway reactivity	Exercise tolerance, chronic productive cough, character of sputum	Auscultation	CXR if symptomatic Hct, sputum

Key References: Moores LK: Smoking and postoperative pulmonary complications, *Clin Chest Med* 21:139–146, 2000; Barrera R, Shi W, Amar D, et al.: Smoking and timing of cessation: impact on pulmonary complications after thoracotomy, *Chest* 127(6):1977–1983, 2005.

Perioperative Implications

Preoperative Preparation

- Cessation overnight will decrease COHb and nicotine.
- Cessation for 4 wk will decrease postop pulm complications. Cessation for <4 wk does not increase the rate of pulm complications.
- Preop nicotine replacement therapy 4–8 wk before surgery and counseling may reduce postop complication rate and increase the rate of long-term smoking cessation; 60% 3–6 mo abstinent.
- Scheduling of elective surgery should be considered an opportunity to quit smoking.
- If chronic productive cough, consider antibiotic treatment at time of surgical scheduling.

Monitoring

- Routine monitoring.
- SpO_2 monitoring may falsely read higher SpO_2 than actual if COHb is present (SpO_2 = % HbO_2 + % COHb).
- Consider invasive monitoring if symptomatic pulm or cardiac disease.

Airway

- Potential laryngeal hyperreactivity

Premedication/Induction

- Consider deep induction if Hx of reactive airway disease.

Maintenance

- Routine maintenance unless symptomatic cardiac or pulm disease.
- Avoid light depth of anesthesia to reduce potential bronchospasm. Desflurane has a similar rate of bronchospasm as sevoflurane.

Extubation

- Consider deep extubation if severe reactive airway disease but easy to intubate and ventilate, with no aspiration risk.

Adjuvants

- Routine; smoking increases metabolism of theophylline, and it decreases the half-life from 265 to 180 min.

Postoperative Period

- Epidural analgesia may be beneficial in decreasing complications of hypercoagulability, CAD, or COPD.

Anticipated Problems/Concerns

- Long-standing Hx of smoking with symptomatic pulm disease leads to a high risk of developing postop pneumonia due to increased mucus production and decreased ciliary function. Cessation for at least 4 wk is recommended.
- Airway reactivity significantly increased in smokers; abstinence for 24 h does not change this reactivity. Reactivity starts reducing after 24–48 h and reduces to near the level of nonsmokers after 10 d of abstinence.
- Risk of MI decreases after 2 years of cessation; 15 years cessation is required to reduce risk to that of someone who never smokes.