

SMOKING AND ANAESTHESIA ANAESTHESIA TUTORIAL OF THE WEEK 221

2ND MAY 2011

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QUESTIONS

1. Concerning nicotine
 - a) Is an adrenergic agonist.
 - b) It increases the heart rate for 1-2 hours.
 - c) It causes vasoconstriction and increases the blood pressure.
 - d) Levels will decrease after 12-24 hours of abstinence.

2. Carbon monoxide in cigarette smoke
 - a) Binds to haemoglobin and reduces its oxygen carrying capacity.
 - b) Has a positive inotropic effect.
 - c) An increased concentration of carboxyhaemoglobin will cause the pulse oximeter to underestimate the oxygen saturation.
 - d) Shifts the oxygen haemoglobin curve to shift to the right.

3. In chronic smokers
 - a) Pre-oxygenation is not routinely necessary.
 - b) Coughing, breath holding and laryngospasm are common.
 - c) Breathing exercises and physiotherapy are useful along with bronchodilators.
 - d) The threshold for pain is higher than normal and they need less analgesia than non-smokers.

INTRODUCTION

Smoking is a risk factor for intra operative pulmonary complications and a wide range of post-operative pulmonary, cardiovascular and wound related complications (1). It is associated with poorer outcomes in gastrointestinal, orthopaedic, day care, plastic and cardiovascular surgery.

Cigarette smoking causes cough, mucous hypersecretion and airflow obstruction. Passive smokers also have an increased incidence of adverse events. Studies have shown that both active and passive smokers suffered significantly more complications during induction of anaesthesia when compared to non-smokers. Current smoking rates in England are 21% overall. Smoking prevalence is highest in deprived communities.

Schwilk et al (2), compared specific respiratory events such as re-intubation, laryngospasm, bronchospasm, aspiration, hypoventilation and hypoxemia during anaesthesia in smokers and non-smokers. The incidence was found to be 5.5% in smokers and 3.3% in non-smokers. Obese smokers were at the highest risk of respiratory problems during anaesthesia.

Nicotine reaches the brain within seconds after inhalation. Long term tobacco smoking of more than fifty pack years carries a higher risk of post-operative admission to intensive care (3). The number of pack years is calculated by the number of packs smoked per day multiplied by the number of years smoked.

EFFECTS OF NICOTINE AND CARBON MONOXIDE

Cardiovascular system.

Nicotine and carbon monoxide are the main harmful substances in a cigarette smoke. Nicotine stimulates the adrenal medulla to secrete adrenaline. It resets the aortic and carotid body receptors to maintain a higher blood pressure. It also stimulates the sympathetic system which results in an increase in heart rate, blood pressure and peripheral vascular resistance. Myocardial contractility is increased, leading to an increase in oxygen demand and consumption. Nicotine increases intracellular calcium during ischemia. This may exacerbate myocardial cell damage (4).

In smokers, the plasma concentration of nicotine reaches 15-50 ng/ml. The half-life of nicotine is 30-60 minutes (5). Three to four hours of abstinence will improve myocardial oxygen supply : demand ratio. Carbon monoxide binds with cytochrome oxidase and myoglobin and inactivates mitochondrial enzymes in the cardiac muscle (4). There is a decrease in the intracellular oxygen transport and utilisation, and a negative inotropic effect leading to chronic tissue hypoxia. The half-life of carboxy haemoglobin depends chiefly on pulmonary ventilation (6). At rest, the half-life is about 4-6 hours. It has been found that the half-life is longer in males than females.

Respiratory system.

Smoking affects oxygen transport and delivery (7). Irritants in smoke increase mucus secretion. The mucus becomes hyperviscous, with altered elasticity. Cilia become inactive and are destroyed by ciliotoxins. The result is impaired tracheobronchial clearance (8). The integrity of the epithelium is lost because of the irritants in the smoke which result in increased reactivity. Smoking leads to narrowing of small airways, causing an increase in closing volume. There is also an increase in proteolytic and elastolytic enzymes leading to loss of elasticity and emphysema. The risk of lung infection is increased. 25% of smokers suffer from chronic bronchitis (2).

The incidence of chronic obstructive airway disease is higher in smokers. When pulmonary function tests are done, chronic smokers show an obstructive pattern and the passive smokers show evidence of small airways disease as their closing volumes are significantly increased.

Carboxyhaemoglobin levels maybe up to 15% in smokers (9). The amount of carbon monoxide present in the blood of smokers depends on the frequency, method and the type of cigarette smoked.

Carbon monoxide and oxygen both bind to the alpha chain of haemoglobin, but the affinity of carbon monoxide is 250 times greater than oxygen. This results in a reduction in the availability of oxygen binding sites and a reduction in oxygen carrying capacity. Left shift of the oxygen haemoglobin dissociation curve results in reduced oxygen delivery to the tissues.

Administering 100% oxygen will significantly expedite the removal of carbon monoxide. With 100% oxygen, the half-life of carbon monoxide is 40-80 minutes.

Other systems.

Smoking has no effect on the gastric volume or the pH of gastric secretions. Smoking relaxes the gastro-oesophageal sphincter but returns to normal within minutes after stopping. There is no increased risk of acid pulmonary aspiration in smokers.

Nicotine is powerfully addictive.

Impaired humoral activity and cell mediated immunity leads to impaired immune response which results in increased risk of infection and malignancy (8). It also decreases immunoglobulins and leucocyte activity. Smoking also results in increased secretion of anti-diuretic hormone (ADH) leading to dilutional hyponatremia.

Effects of general anaesthesia on smokers

- Central respiratory depression
- Reduced compliance
- Cranial shift of diaphragm
- Atelectasis
- Decreased FRC(Functional residual capacity)
- FRC may exceed closing capacity
- Impaired oxygen exchange
- Increased shunt
- Increased V/Q mismatch
- Increased A –a difference

ANAESTHESIA DRUG REQUIREMENTS IN SMOKERS

There are over 4800 substances isolated from cigarette smoke of which nicotine and carbon monoxide are well known. Others include nitrogen oxides, volatile aldehydes, alkenes and the toxin hydrogen cyanide. In smokers, there are no changes in the action of volatile agents but increased metabolism can lead to higher levels of toxic metabolites (10).

Smoking induces liver microsomal enzymes increasing the metabolism of certain drugs. Chronic smokers require higher doses of analgesics. Smoking significantly influences the requirement for pethidine and morphine. In addition to increased metabolism, there are other mechanisms which are poorly understood. The clinical effects of paracetamol and NSAIDS remains the same as in non-smokers, and there is no evidence of altered pharmacokinetics.

Two studies have shown that the potency of aminosteroid muscle relaxants are decreased. Hepatic and renal failure have been reported following exposure to volatile anaesthetic agents. The amount of fluoride released is related to the duration of exposure, the concentration of the agent and the degree of metabolism. The extent to which the risk becomes significant is difficult to quantify.

Post-operative nausea and vomiting (PONV) in smokers

There is evidence that smokers are less likely to suffer from post-operative nausea and vomiting. Tobacco smoke is also known to induce enzymes like cytochrome P450 and this partially explains the PONV protecting effect of smoking. Chronic exposure to nicotine leads to desensitization of central nicotine receptors which increases the tolerance to emetogenic effects of surgery and anaesthesia.

Pre-operative assessment and smoking cessation

Patients are advised to quit smoking at least four to six weeks prior to surgery. Abstinence for twelve hours is sufficient to get rid of carbon monoxide. Ciliary function improves and nicotine levels return to normal within 12-24 hours. Abstinence for 2 weeks helps return sputum volume to normal levels. Laryngeal and bronchial activity is better in 5-10 days. Improvement in small airway narrowing is seen in 4 weeks but it takes 3 months to see changes in tracheobronchial clearance. Interestingly, stopping smoking in asthmatics may worsen their symptoms and cessation for a brief period increases the risk of laryngospasm and bronchospasm during anaesthesia.

Stopping smoking is also associated with anxiety and withdrawal symptoms.

Smoking poses a significant risk factor for post-operative pulmonary complications. Smokers are more prone to post-operative atelectasis which delays the recovery and predisposes the patients to pneumonia. Also there is an increased incidence of intensive care admissions.

ANAESTHESIA

Airway complications on induction, particularly during facemask ventilation or LMA insertion, are common, and the need for intubation should always be anticipated. Pre-oxygenation should be routine.

Adequate anaesthesia should be administered for intubation to minimise the risk of provoking bronchospasm.

Regional anaesthesia has advantages for patients with long term respiratory complications of smoking. Underlying ischaemic heart disease and hypertension should be identified, and anaesthesia administered to minimise the risk from these factors.

Appropriate analgesia should be prescribed, particularly for abdominal or thoracic surgery where regional techniques such as epidural analgesia may have a role.

Early mobilisation is important to improve lung function and sputum clearance. Often patients manage this themselves to ensure access to a cigarette!

PREVENTION

In the UK, current NHS policy includes efforts to:

- Modernise NHS stop smoking services
- Improve the effectiveness of pharmacotherapy
- Improve the evidence base for smoking cessation work and intelligence on the efficacy of interventions (11).

ANSWERS TO QUESTIONS

1. TFFT
2. TFFF
3. FTTF

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