Pathophysiology of respiratory disease and its significance to anaesthesia

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Abstract

Significant changes occur in the respiratory physiology of healthy patients during anaesthesia. In patients with underlying respiratory pathology, these changes in respiratory physiology may lead to clinical problems during the conduct of anaesthesia and the perioperative period. An understanding of the disease processes that can affect the lungs and pleura allows the anaesthetist to account for the potential complications of these conditions and manage the anaesthetic accordingly.

Keywords ARDS; asthma; COPD; obstructive; pathophysiology; perioperative management; restrictive; risk stratification; trauma

Royal College of Anaesthetists CPD matrix: 1A01, 2A02, 2A03, 2A06

Learning objectives

After reading this article you should be able to:

- perform a thorough preoperative assessment for respiratory pathology and quantify patients' risk of respiratory complications following anaesthesia
- understand the pathophysiological effects of common respiratory diseases
- adapt your anaesthetic practice to allow for these pathophysiological changes

Preoperative assessment

Symptoms, signs, previous respiratory disease and relevant medications should be identified (Table 1). Patients' functional capacity should be assessed by evaluating both their subjective and objective exercise capacity. An estimation of the metabolic equivalents (METs) that a patient can achieve is recommended: 1 MET equates to the resting oxygen consumption of a 70-kg man and is approximately 3.5 ml/kg/minute. The ability to climb one flight of stairs is equivalent to approximately 4 METs; the inability to achieve this marks increased risk of perioperative cardiac complications.[1]

Objective measures of exercise capacity can be used to help stratify risk. These include:

- 6-minute or incremental shuttle walk tests
- walking pulse oximetry
- cardiopulmonary exercise testing.

Patients' risk of pulmonary complications should be estimated based on patient factors and the operative procedure (Box 1).[2] Using these risk stratification tools means that patients can be appropriately managed by consultant surgeons and anaesthetists, and will guide decisions regarding the need for postoperative critical care input.

Pulmonary pathology

Restrictive conditions

Expansion is restricted in these conditions. This can be due to lung parenchymal abnormalities, diseases of the chest wall and pleura, or neuromuscular conditions.

Pulmonary fibrosis: fibrosis may be idiopathic or secondary to other respiratory (e.g. pneumoconiosis) and systemic conditions (e.g. rheumatoid arthritis). This results in inflammation and infiltration of alveolar membranes and bronchiolar walls. Cellular exudate collects in the alveoli and fibroblasts form collagen at the damaged areas. Lung parenchyma architecture is altered leading to the formation of air-filled spaces, a reduced surface area available for gas exchange and reduced distensibility.

Mechanical restriction: chest wall deformities (e.g. kyphoscoliosis) cause abnormal crowding of ribs and compression of the lung and pulmonary vasculature resulting in restricted ventilation. In obesity and pregnancy, as well as a Trendelenburg position during surgery, the weight of abdominal and chest tissue impairs inspiration and reduces diaphragmatic excursion. In cases of intra-abdominal pathology, pain and abdominal wall rigidity can also limit movement of the diaphragm.

Neuromuscular conditions: conditions such as Guillain-Barré, muscular dystrophies and myasthenia gravis (as well as the simple effects of ageing) reduce the function of the muscles of respiration, thereby preventing adequate chest wall movement. This leads to atelectasis, reduced clearance of secretions and an increased incidence of pneumonia. Parkinson's disease can cause reduced upper airway tone which may lead to aspiration pneumonia and respiratory failure.

Consequences of restrictive lung conditions

The following may be seen but will be dependent on the causative condition:

- Increased work of breathing due to reduced lung compliance:
  - Initially the patient can meet an increase in ventilatory demand by increasing the respiratory rate (tidal volume may be relatively fixed).
  - If the condition persists, the patient may tyre and develop respiratory failure.

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Preoperative assessment and investigation of patients with respiratory pathophysiology

Points in history	• Changing or worsening symptoms, e.g. increased cough at night • Symptoms of right or congestive cardiac disease • Smoking history (number of pack years) • Home nebulizers or oxygen therapy • Steroid therapy (frequency of short-course doses or long-term use) • Recent or frequent courses of antibiotics • Previous admissions to hospital and critical care		
Symptoms	Dyspnoea • On exertion/at rest • Orthopnoea • Paroxysmal nocturnal dyspnoea	Cough • Sputum production • Haemoptysis	Chest pain Peripheral oedema Wheeze
Signs	**General signs** Distressed patient, sitting forward Respiratory rate and pattern • Tachypnoea, hypoventilation, stridor, abnormal respiratory pattern Cyanosis, plethoric facies Clubbing, tar-staining to fingers or hair Obesity, pectus excavatum, kyphoscoliosis	**Observations** • Low SpO_2 • Tachycardia • Pyrexia Peripheral oedema Hepatomegaly	**Chest signs** • Dullness or hyperresonance on percussion • Wheeze, crackles • Pleural rub • Absent breath sounds
Investigations	**Blood tests** • Renal function – urea and creatinine (*linked to postoperative pulmonary complications when elevated*) • Albumin (*low level has strong association with postoperative pulmonary complications*) • Full blood count – WCC (*infection*) and Hb (*polycythaemia*, *anaemia*) • Clotting, INR (*may be on warfarin for PE or pulmonary hypertension*) • Arterial blood gases – oxygen and carbon dioxide concentrations, pH (*ABG analysis may differentiate between patients with chronically elevated carbon dioxide levels and patients with acute decompensation*)		**Others** • ECG • Lung function tests • PEFR **Consider:** • CXR • Echocardiography • CPET

ABG, arterial blood gas; CPET, cardiopulmonary exercise testing; CXR, chest X-ray; ECG, electrocardiography; Hb, haemoglobin; PE, pulmonary embolism; PEFR, peak expiratory flow rate; WCC, white cell count.

Table 1

- Reduced total lung capacity (TLC), functional residual capacity (FRC) and residual volume (RV).
- Increased forced expiratory volume in 1 second (FEV_1)/forced vital capacity (FVC) ratio (>80%) (both FEV_1 and FVC are reduced with the FVC reduced to a greater extent).
- Atelectasis.
- Hypoxaemia resulting from V/Q mismatch:
  - Both dead space and shunt are increased.
  - There is disorganized lung architecture affecting both lung parenchyma and blood vessels.
  - Arterial pCO_2 tends to be low or normal due to increased alveolar ventilation.
- Respiratory failure and cor pulmonale.
- Reduced diffusion capacity for carbon monoxide:
  - Manifests as hypoxia at times of increased oxygen demand, such as exercise or during surgery.

Generalized obstructive conditions

Asthma: the symptoms of asthma develop after antigen exposure to the dendritic cells of the airway. This activates T-helper cells, resulting in the release of cytokines and an influx of mast cells, basophils, eosinophils, neutrophils and macrophages. These then release mediators such as histamine, prostaglandins and leukotrienes. The result is airway inflammation, constriction, oedema and increased mucus secretion. This causes intermittent, and usually reversible, air flow obstruction and bronchial hyperreactivity. The chronic inflammation leads to airway remodelling (including epithelial shedding and matrix degradation); the structural change is thought to alter mucociliary clearance and increases the risk of infection.

Chronic obstructive pulmonary disease (COPD): COPD encompasses emphysema and chronic bronchitis. In emphysema, the alveoli and capillary beds are destroyed by loss of cell wall elastin. The gas exchange surface area is reduced and loss of radial traction causes airway narrowing. In bronchitis, there is chronic inflammation and oedema of the lung parenchyma. Goblet cells produce excess mucus which narrows the airways and form plugs. These plugs and desquamated epithelial cells remain in the lungs due to loss of ciliary function providing an ideal medium for bacterial growth.

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