Pathophysiology of respiratory disease and its significance to anaesthesia

Sophie Kimber Craig

Abstract

Significant changes occur in the respiratory physiology of healthy patients during anaesthesia. In patients with underlying respiratory pathology, the changes in respiratory physiology may lead to additional clinical problems during the conduct of anaesthesia and in 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; postoperative pulmonary complications (PPC); 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
- appreciate the impact of anaesthesia on the respiratory system in the context of current available evidence
- 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. Patients' risk of postoperative pulmonary complications (PPC) should be estimated based on patient factors and the operative procedure (Box 1).[1–3] 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.

Effects of anaesthesia on the respiratory system

These effects of general anaesthesia (GA) begin at induction and continue long into the postoperative period:

- Depressed respiratory drive:
  - Ventilatory response to hypoxia and hypercarbia are reduced.
- V/Q mismatch:
  - Reduced muscle tone causes decreased functional residual capacity (FRC).
  - Abnormal regional ventilatory distribution occurs due to positive pressure ventilation during surgery.
  - A fall in cardiac output leads to reduced perfusion.
- Atelectasis:
  - Very common in GA cases when muscle relaxants are used.
  - Compression of lungs and airway closure (when FRC is less than closing volume) contribute.

Risk factors for developing postoperative pulmonary complications (PPC)

- Age — patients from 60 to 69 years old at approximately double the risk of younger counterparts and patients from 70 to 79 years old at three times the risk
- Frailty
- Low preoperative oxygen saturations (<96%) — patients with saturations of 91–95% have twice the risk and <91% a tenfold increase compared with those with saturations >95%
- Recent LRTI
- Respiratory comorbidity
- Anaemia (<100 g dl^{-1})
- Smoking — cessation in advance of surgery (>8 weeks) significantly reduces the risk
- Congestive heart failure
- ASA score ≥2
- Altered GCS — obtunded patients are more likely to suffer postoperative complications
- Nature of surgery — the following are all associated with increased risk of postoperative complications:
  - Prolonged operation (>2 h)
  - Emergency surgery (twofold to sixfold increased risk)
  - Intrathoracic/upper abdominal surgery
  - Open surgery (versus laparoscopic techniques)
  - Repeat surgery
- General anaesthesia — increased PPC compared with regional techniques

Note that spirometry results are not a useful indicator of PPC, nor are ABGs, compared with clinical acumen alone. An abnormal chest X-ray is predictive of PPC (but can also be expected by physical examination findings and is therefore not essential).

Box 1

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The 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 nebulisers 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**	**Cough**	Chest pain
	On exertion/at rest	Sputum production	Peripheral oedema
	Orthopnoea	Haemoptysis	Wheeze
	Paroxysmal nocturnal dyspnoea		
Signs	**General signs**	**Observations**	**Chest signs**
	Distressed patient, sitting forward	Low SpO_2	Dullness or hyperresonance on percussion
	Respiratory rate and pattern	Tachycardia	Wheeze, crackles
	Tachypnoea, hypoventilation, stridor,	Pyrexia	Pleural rub
	abnormal respiratory pattern	Peripheral oedema	Absent breath sounds
	Cyanosis, plethoric facies	Hepatomegaly	
	Clubbing, tar-staining to fingers or hair		
	Obesity, pectus excavatum, kyphoscoliosis		
Investigations	**Blood tests**		**Others**
	Renal function — urea and creatinine *(linked to postoperative pulmonary complications when elevated)*		ECG
			Lung function tests
	Albumin *(low level has strong association with postoperative pulmonary complications)*		PEFR
			Consider:
	Full blood count — WCC *(infection)* and Hb *(polycythaemia, anaemia)*		CXR
	Clotting, INR *(may be on warfarin for PE or pulmonary hypertension)*		Echocardiography
	Arterial blood gases — oxygen & carbon dioxide concentrations, pH *(ABG analysis may differentiate between patients with chronically elevated carbon dioxide levels and patients with acute decompensation)*		CPET

Table 1

  - Worsened by use of very high concentrations of inspired oxygen at induction, which causes rapid gas absorption from under ventilated airways.
  - Continues into postoperative period.
- Sputum retention.
- Reduced respiratory muscle coordination and ventilatory control after surgery:
  - Decreased forced expiratory volume in 1 s (FEV_1) and forced vital capacity (FVC), especially with pain.
  - Can be caused by residual GA drug effects and analgesia.

The effects of anaesthesia are worsened by pre-existing lung diseases, which are outlined in the next section.

Pulmonary pathology

Restrictive conditions

Caused by lung parenchymal abnormalities, diseases of the chest wall and pleura or neuromuscular conditions, expansion is reduced in these conditions.

Pulmonary fibrosis: Fibrosis may be idiopathic or secondary to other respiratory (e.g. pneumoconiosis) and systemic (e.g. rheumatoid arthritis) conditions. 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 tire and develop respiratory failure.
- Reduced total lung capacity (TLC), FRC and residual volume (RV).
- Increased FEV_1/FVC ratio >80% (both FEV_1 and FVC are reduced but the FVC is 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 which 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): The term 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.

Consequences of generalized obstruction: The distinctions between the different causes of generalized airway obstruction are not always clearly defined. The following can be seen:

- Indices of expiratory flow are reduced:
  - This includes the FEV_1, forced expiratory flow ($FEF_{25-75\%}$) and FEV as a percentage of vital capacity FEV_1/FVC % (usually <70%).
  - FVC may also be reduced during an acute attack due to premature airways closure toward the end of expiration.
- Increased airway resistance resulting in increased expiratory time and work of breathing.
- Increased TLC, FRC and RV due to gas trapping behind obstructed airways.
- Reduced lung compliance in emphysema due to loss of elastic tissue and destruction of alveolar walls.
- Arterial hypoxaemia due to ventilation–perfusion inequality:
  - Common in asthmatics (due to reduced alveolar ventilation) and bronchitis (secondary to narrowing and oedema of the airways), resulting in shunting.
  - Emphysema tends to cause an increase in dead space.
  - The degree of hypoxaemia depends predominantly on the amount of blood flow to areas that have low V/Q ratios (i.e. low blood flow to these regions results in minimal shunting and mild hypoxaemia, whereas high blood flow causes severe hypoxaemia).
- Hypoxic pulmonary vasoconstriction (HPV) and collateral ventilation reduce shunt and dead space:
  - HPV is constriction of the pulmonary vasculature (mainly the pre-capillary arterioles), in response to a low oxygen level in the alveoli and is designed to divert blood away from underventilated alveoli in order to reduce shunt.
  - HPV is affected by many physiological factors, including acid-base disturbance, prostacyclins and autonomic nervous system stimulation.
  - HPV is reduced by nitrous oxide and vasodilating drugs, and almost ablated by halogenated anaesthetic agents. Therefore, anaesthesia may worsen hypoxaemia in susceptible patients due to an increase in shunting of blood.
  - Collateral ventilation occurs through holes between alveoli and small airways, allowing ventilation to areas with proximal obstruction of the supplying bronchioles.
- Hypercarbia is usually a late sign:
  - More common in patients with COPD than asthma.
  - Initially compensated for by an increase in minute ventilation but, as disease progresses, there is a reduced sensitivity to raised carbon dioxide levels.
- Hypoxic ventilatory drive to compensate for a chronically elevated CO_2 may occur in COPD.
- Respiratory failure and right ventricular failure.

Localized obstructive conditions

Tracheal obstruction: This is potentially life threatening and stridor is the main symptom. Clinical features depend on the degree (partial or complete) and location of the obstruction (glottic or sub-glottic).

Bronchial obstruction has many causes including foreign bodies and bronchial tumour. The right main bronchus is affected more frequently, due to the less acute angle the right main bronchus makes with the trachea. Complete obstruction can result in absorption atelectasis and collapse distal to the obstruction. Compensatory hyperinflation of adjacent lung may be seen. Perfusion of the unventilated lung is reduced by HPV and the increased PVR (secondary to narrowed extra-alveolar vessels due to the mechanical effect of loss of lung volume). This helps to

reduce the degree of shunt but residual blood flow to the unventilated lung area will still contribute to the hypoxaemia seen. Infection and lung abscess formation are late complications.

Infection and inflammation

The common cold results in an increased risk of airway hyperreactivity and lower respiratory tract infection (LRTI). There are rare case reports of viral myocarditis as a consequence of anaesthesia at the time of LRTI. The following symptoms should lead the clinician to consider postponement of an elective procedure:

- productive cough
- pyrexia
- general malaise
- signs on chest auscultation
- concurrent chest disease
- elevated white cell count.

Pneumonia: Pathogens cause activation of the host's macrophages and inflammatory cascade. Alveolar oedema develops and blood and fibrin enter the alveoli. Epithelial and goblet cells are damaged and deposited in the airway and consolidation occurs resulting in impaired gaseous exchange. There is increased work of breathing, hypoxia and hypercarbia.

Acute respiratory distress syndrome (ARDS): is characterized by the following[4]:

- Reduced PaO_2/FiO_2 ratio with positive end-expiratory pressure (PEEP) $\geq$5 cmH_2O, with severity as follows:
  - mild: 201–300 mmHg
  - moderate: 101–200 mmHg
  - severe: $\leq$100 mmHg.
- Bilateral opacities on chest X-ray or CT not fully explained by effusions, collapse or nodules.
- Onset within 1 week of clinical insult or new/worsening respiratory symptoms.
- Respiratory oedema/failure not fully explained by cardiac failure or fluid overload.

ARDS is associated with pulmonary and non-pulmonary conditions (Table 2), with complex pathophysiology which is divided into an early exudative phase (Figure 1) and a later fibroproliferative phase.[5] The injury produces diffuse alveolar damage that releases pro-inflammatory cytokines followed by infiltration and activation of neutrophils. Neutrophils release mediators, such as oxidants and proteases, which damage the alveolar epithelium and capillary endothelium. This allows proteins to move from the vasculature into the interstitium, followed by fluid and the debris from dying cells. Pulmonary oedema is exacerbated by impaired fluid transport by the epithelium. Alveolar collapse results from loss of surfactant and there is also imbalance of coagulation and fibrinolytic pathways, causing deposition of fibrin in the alveoli. The consequences of ARDS are:

- Hypoxaemia: impaired gas exchange due to shunting and decreased lung compliance.
- Pulmonary hypertension: caused by HPV, airway collapse and hypercarbia.
- Mechanical ventilation is often required due to rapid onset respiratory failure. This may lead to further parenchymal damage due to oxygen toxicity and barotrauma.

Common conditions associated with ARDS[4]

Pulmonary	Non-pulmonary
Pneumonia	Non-pulmonary sepsis
Gastric aspiration	Major trauma
Lung contusion	Pancreatitis
Inhalational injury	Severe burns
Pulmonary vasculitis	Non-cardiogenic shock
Drowning	Drug overdose

Table 2

Trauma

Rib fractures and flail chest: Rib fractures may result in a restrictive-like ventilatory pattern. A flail chest is an area of the thoracic cage where there are fractures in two or more adjacent ribs in at least two places. Paradoxical movement of the chest wall segment is seen and it is often associated with underlying lung contusion.

Lung contusion is 'bruising' of the lung parenchyma. Lung capillary damage leads to parenchymal oedema and blood collecting within the alveolar space. This results in reduced gas exchange and hypoxaemia, increased pulmonary vascular resistance and reduced lung compliance. It also generates an inflammatory response and patients may develop pneumonia or ARDS.

Haemothorax and pneumothorax: Large volumes of gas or blood in the pleural space compress adjacent lung tissue. This results in atelectasis and collapse, increased intrathoracic pressure and loss of negative intra-pleural pressure. Tension pneumothorax can occur if the defect in the lung allows gas into the intra-pleural space but prevents its release. Mediastinal shift with a precipitous fall in cardiac output may result. Significant amounts of blood can be lost into the pleural space (up to 40% of blood volume in each hemithorax).

Insert a chest drain prior to induction of anaesthesia as a pneumothorax may enlarge significantly when using positive pressure ventilation. Assess the patient's haemodynamic status and haemoglobin concentration preoperatively. Cross-matched blood must be available. Be vigilant to the development of a tension pneumothorax or a problem with the chest drain If there are sudden changes in cardiovascular parameters during surgery.

Pulmonary vascular conditions

Pulmonary embolus (PE): This is from deep vein thrombosis, fat, amniotic fluid or injected material that enters the pulmonary circulation. The effect depends on its size and location:

- Large emboli may occlude the proximal pulmonary arteries causing acute haemodynamic compromise as forward-flow from the right heart is prohibited.
- Moderate-sized emboli may cause parenchymal infarction.

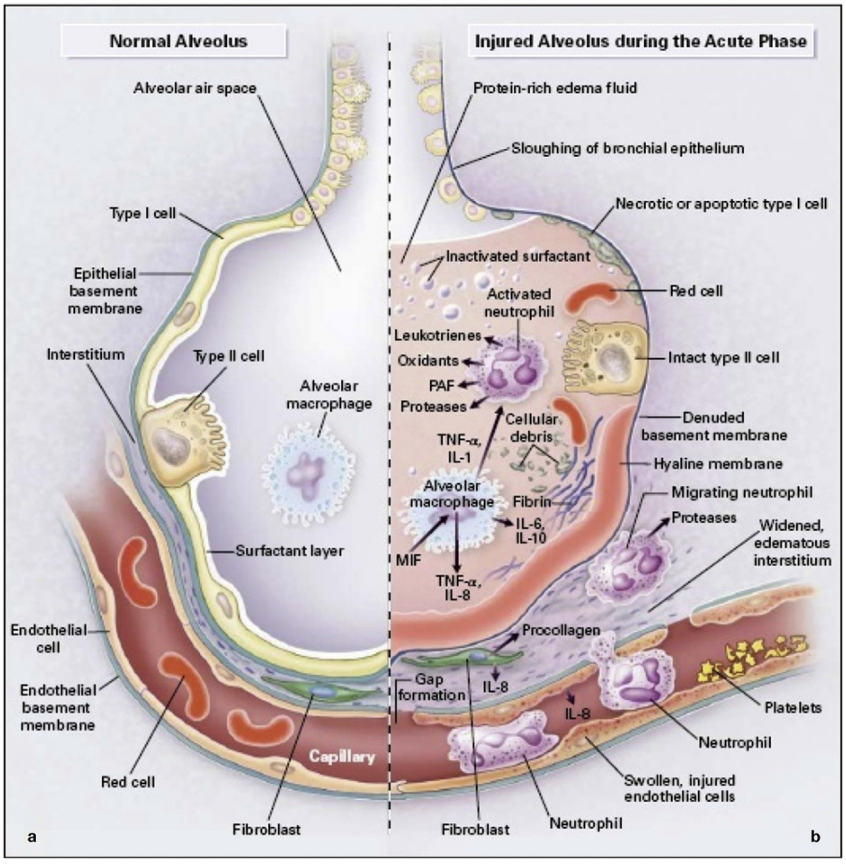

Figure 1 Pathophysiology of acute phase of ARDS (**a**) The normal alveolus. (**b**) The injured alveolus in the acute phase of ARDS. There is sloughing of the bronchial and alveolar epithelial cells with formation of protein-rich hyaline membranes on the denuded basement membrane. Neutrophils adhere to the injured capillary endothelium and marginate through the interstitium into the airspace, which is filled with protein-rich oedema fluid. Alveolar macrophages are activated by macrophage migration inhibitory factor (MIF) and secrete interleukins and tumour necrosis factor-α (TNF-α), which act locally to stimulate chemotaxis and activate neutrophils. IL-1 also stimulates the production of extracellular matrix by fibroblasts. Neutrophils release oxidants, proteases, leukotrienes and other pro-inflammatory molecules (e.g. platelet-activating factor). Anti-inflammatory mediators are also present, including IL-1-receptor antagonist, soluble TNF receptor, autoantibodies against IL-8 and cytokines (e.g. IL-10 and 11). The influx of protein-rich oedema fluid into the alveolus has led to the inactivation of surfactant. (From Ware LB, Matthay M. The acute respiratory distress syndrome. *New Engl J Med* 2000; 342: 1334e49. With kind permission of the Massachusetts Medical Society. Copyright © [2000] Massachusetts Medical Society. All rights reserved.)

- Small emboli may go clinically unrecognized until symptoms of RVF develop secondary to the long-term increased PVR caused by capillary occlusion.

PE under general anaesthesia may be difficult to diagnose. The following may occur:

- sudden fall in end-tidal CO_2 concentration
- tachycardia
- hypotension
- hypoxaemia
- pallor.

Pulmonary hypertension (PH): This may be primary (idiopathic) or secondary. It is defined as a mean pulmonary artery pressure (PAP) >15 mmHg.[6] Primary PH is poorly understood pathologically but increased smooth muscle in the pulmonary arteries is seen. The secondary causes are:

- increased left atrial pressure (e.g. mitral stenosis)
- increased pulmonary blood flow (e.g. ventricular septal defect)
- increased pulmonary vascular resistance due to vasoconstriction, obstruction or obliteration (e.g. PE).

Hypoxaemia may develop on exertion and is present at rest in advanced cases. The condition results in RVF secondary to chronically elevated PAP.

Anaesthetic considerations

Preoperative management

Patients may present for surgery with an acute episode of respiratory disease. Unless surgery is urgent, postponing it until this has fully resolved should be strongly considered. When postponement is not possible, optimize the patient using (where appropriate):

- chest physiotherapy
- oxygen therapy
- antibiotics
- nebulized bronchodilators.

Advise smoking cessation if done weeks in advance of surgery as it can improve respiratory function. Avoid using sedative

drugs as they may lead to deterioration in respiratory function by causing hypoventilation and inhibition of coughing.

Type of anaesthesia and analgesia

Regional anaesthesia (RA), peripheral nerve blockade and local anaesthesia are good alternatives to general anaesthesia; GA is an independent risk factor for PPC compared with RA.[1] However, some regional techniques may be contraindicated in patients with respiratory disease. For example, inter-scalene brachial plexus block may result in respiratory failure due to phrenic nerve blockade.

Intraoperative opioids may cause decreased respiratory effort postoperatively due to central nervous system depression. This will lead to hypercarbia, which can cause further neurological depression resulting in hypoxaemia and respiratory failure particularly in susceptible patients with underlying disease (e.g. severe COPD). Using multi-modal analgesia (e.g. paracetamol and NSAIDs along with local anaesthetic techniques/infiltration) will reduce the dose of opioids required.

Airway management

Keep airway manipulation to a minimum in patients at risk of airway hyper-reactivity. Using laryngeal mask airways (LMAs) rather than endotracheal tubes (ETTs) may reduce the incidence of laryngospasm and bronchospasm. ETTs do offer some advantages though, including the ability to perform tracheobronchial suctioning and more complex ventilation strategies.

Direct laryngoscopy may be difficult in patients with lesions of the oropharynx or larynx. Intubation using a fibreoptic scope may not be appropriate if there is partial obstruction of the airway, as the scope may actually occlude the remaining airway; and a gaseous induction should be considered in these circumstances. Lesions distal to the ETT may cause persistent obstruction following successful endotracheal intubation; involve an ENT specialist in these cases.

Ventilation strategies

Patients with respiratory pathology may require manipulation of their ventilation settings dependent upon the underlying condition:

- Increase FiO_2 to improve hypoxaemia but avoid using FiO_2 of 1.0.
- Use recruitment manoeuvres (plateau pressure of 40 cm H_2O for 7–8s in non-obese patient with normal lungs) to re-inflate collapsed alveoli.
- Use PEEP to maintain open airways.
- Use lung protective ventilation strategies:
  - tidal volumes of 6 ml/kg
  - PEEP $\geq$5 cmH_2O
  - increased respiratory rates
  - permissive hypercapnia.

Recent studies show the benefits of these protective mechanical ventilation settings intraoperatively even in those without a pre-existing lung condition. There is decreased risk of postoperative complications (including pneumonia, pulmonary oedema, respiratory failure and re-intubation) with their use.[7]

Venous thromboembolism (VTE) prophylaxis

All patients should have their VTE risk assessed and appropriate prophylaxis administered (including low molecular weight heparin and compression stockings).

Extubation

Suction bronchial secretions and perform lung recruitment manoeuvres prior to extubation. Always proceed with caution as airway hyper-reactivity increases the risk of bronchospasm and laryngospasm.

Postoperative management

Patients felt to be at increased risk of perioperative respiratory morbidity (see Box 1) should be cared for in an appropriate environment postoperatively (e.g. high dependency or intensive care).

The following strategies can be employed in the perioperative period to help reduce respiratory complications following surgery:

- chest physiotherapy
- nursing in semi-recumbent or upright positions
- incentive spirometry
- early mobilization
- encouragement to cough and deep breathing exercises
- humidified oxygen
- good analgesia. ◆

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FURTHER READING

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West JB. Pulmonary pathophysiology: the essentials. 7th edn. Crawfordsville: Lippincott Williams & Wilkins. A follow-on text from West's Respiratory Physiology that gives comprehensive details of the pathophysiology of respiratory disease, with a particularly useful section on the lung function tessts and what they mean. YouTube has a number of lectures given by West himself, both on physiology and pathophysiology of the respiratory system. They can be found at: https://www.youtube.com/playlist?list=PLE69608EC343F5691 https://www.youtube.com/playlist?list=PL3854F2389B08EF7E respectively.

