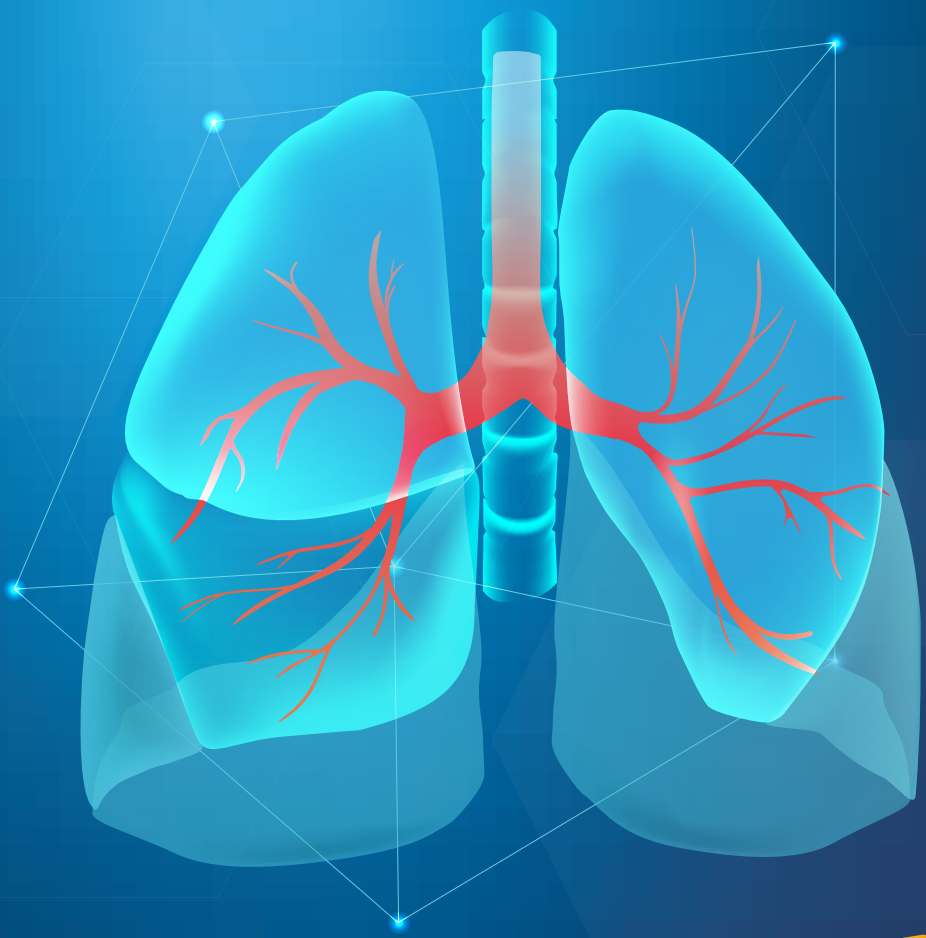
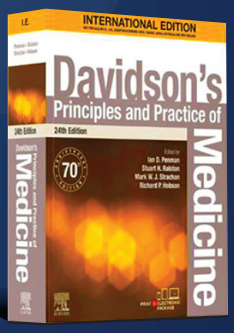
TABLE & CHARTS OF RESPIRATORY SYSTEM

Davidson's Principles of Medicine 24th Edition





SK+F

6 — 9 Thorax
(see opposite)

5 Face, mouth and eyes
Pursed lips
Central cyanosis
Anaemia
Horner's syndrome
(Ch. 25)

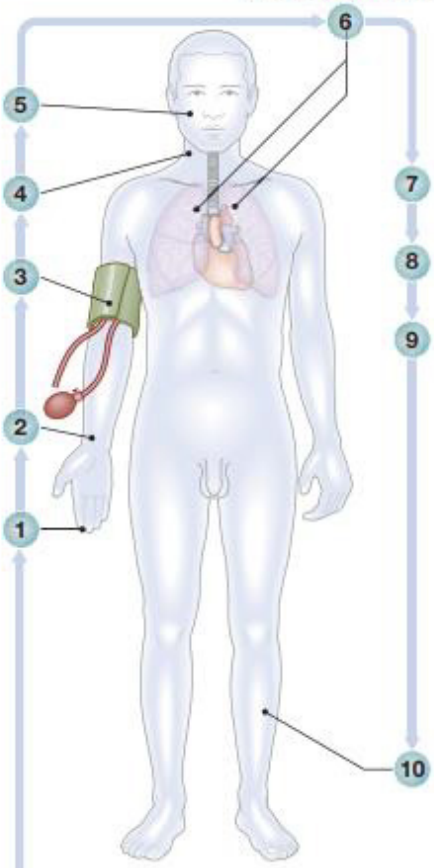
4 Jugular venous pulse
Elevated
Pulsatile

3 Blood pressure
Arterial paradox

2 Radial pulse
Rate
Rhythm

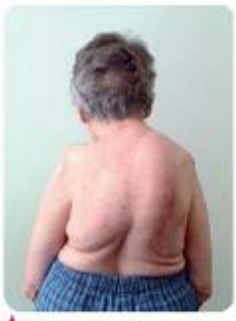
1 Hands
Digital clubbing
Tar staining
Peripheral cyanosis
Signs of occupation
CO_2 retention flap

▲ Finger clubbing



Observation
- Respiratory rate
- Cachexia, fever, rash
- Sputum (see below)
- Fetor
- Locale:
  Oxygen delivery
  (mask, cannulae)
  Nebulisers
  Inhalers

6 Inspection
Deformity
(e.g. pectus excavatum)
Scars
Intercostal indrawing
Symmetry of expansion
Hyperinflation
Paradoxical rib movement
(low flat diaphragm)



▲ Idiopathic kyphoscoliosis

7 Palpation
From the front:
Trachea central
Cricosternal distance
Cardiac apex displaced
Expansion
From behind:
Cervical lymphadenopathy
Expansion

8 Percussion
Resonant or dull
'Stony dull' (effusion)

9 Auscultation
Breath sounds:
normal, bronchial, louder or softer
Added sounds:
wheezes, crackles, rubs
Spoken voice (vocal resonance):
absent (effusion), increased (consolidation)
Whispered voice:
whispering pectoriloquy

10 Leg oedema
Salt and water retention
Cor pulmonale
Venous thrombosis

Sputum

▲ Serous/frothy/pink
Pulmonary oedema

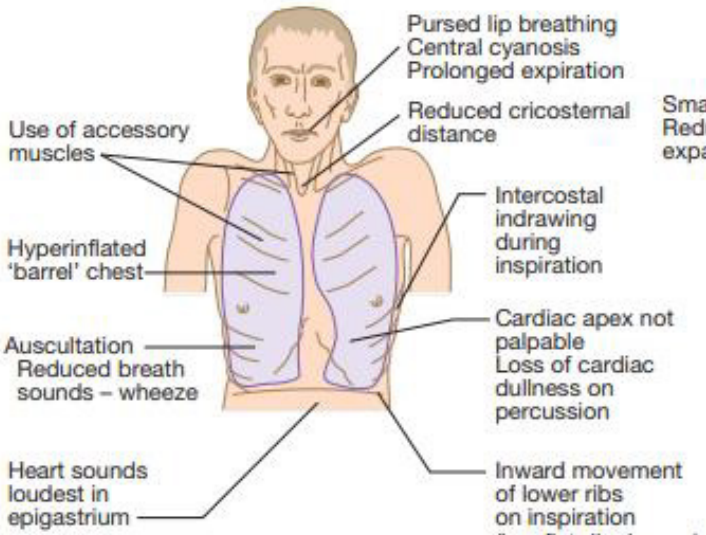
▲ Mucopurulent
Bronchial or pneumonic infection

▲ Purulent
Bronchial or pneumonic infection

▲ Blood-stained
Cancer, tuberculosis, bronchiectasis, pulmonary embolism

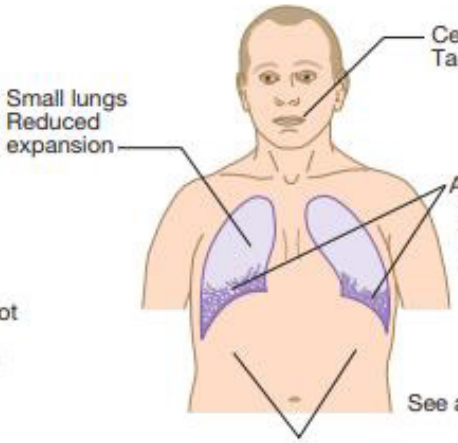
Insets (idiopathic kyphoscoliosis) Courtesy of Dr I. Smith, Papworth Hospital, Cambridge; (serous, mucopurulent and purulent sputum) Courtesy of Dr J. Fowerаker, Papworth Hospital, Cambridge.

Chronic obstructive pulmonary disease



Also: raised jugular venous pressure (JVP), peripheral oedema from salt and water retention and/or cor pulmonale

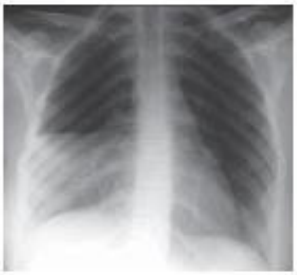
Pulmonary fibrosis



See also Fig. 17.56

Also: finger clubbing common in idiopathic pulmonary fibrosis; raised JVP and peripheral oedema if cor pulmonale

Right middle lobe pneumonia



Obscures R heart border on X-ray

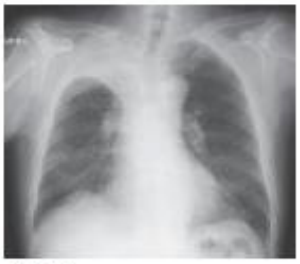
Inspection
- Tachypnoea
- Central cyanosis (if severe)

Palpation
- ↓Expansion on R

Percussion
- Dull R mid-zone and axilla

Auscultation
- Bronchial breath sounds and ↑vocal resonance over consolidation and whispering pectoriloquy
- Pleural rub if pleurisy

Right upper lobe collapse



X-ray
- Deviated trachea (to R)
- Elevated horizontal fissure
- ↓Volume R hemithorax
- Central (hilar) mass may be seen

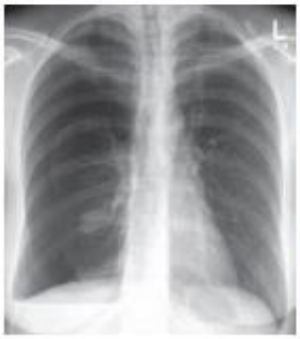
Inspection
- ↓Volume R upper zone

Palpation
- Trachea deviated to R
- ↓Expansion R upper zone

Percussion
- Dull R upper zone

Auscultation
- ↓Breath sounds with central obstruction

Right pneumothorax



Inspection
- Tachypnoea (pain, deflation reflex)

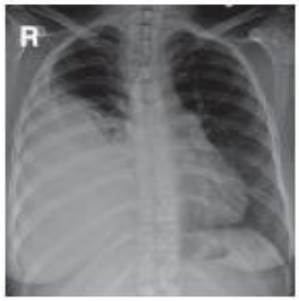
Palpation
- ↓Expansion R side

Percussion
- Resonant or hyper-resonant on R

Auscultation
- Absent breath sounds on R

Tension pneumothorax also causes
- Deviation of trachea to opposite side
- Tachycardia and hypotension

Large right pleural effusion



Inspection
- Tachypnoea

Palpation
- ↓Expansion on R
- Trachea and apex may be moved to L

Percussion
- Stony dull
- R mid- and lower zones

Auscultation
- Absent breath sounds and vocal resonance R base
- Bronchial breathing or crackles above effusion

Insets (upper lobe collapse) From http://3.bp.blogspot.com; (pneumothorax) http://chestatlas.com; (pleural effusion) www.ispub.com.

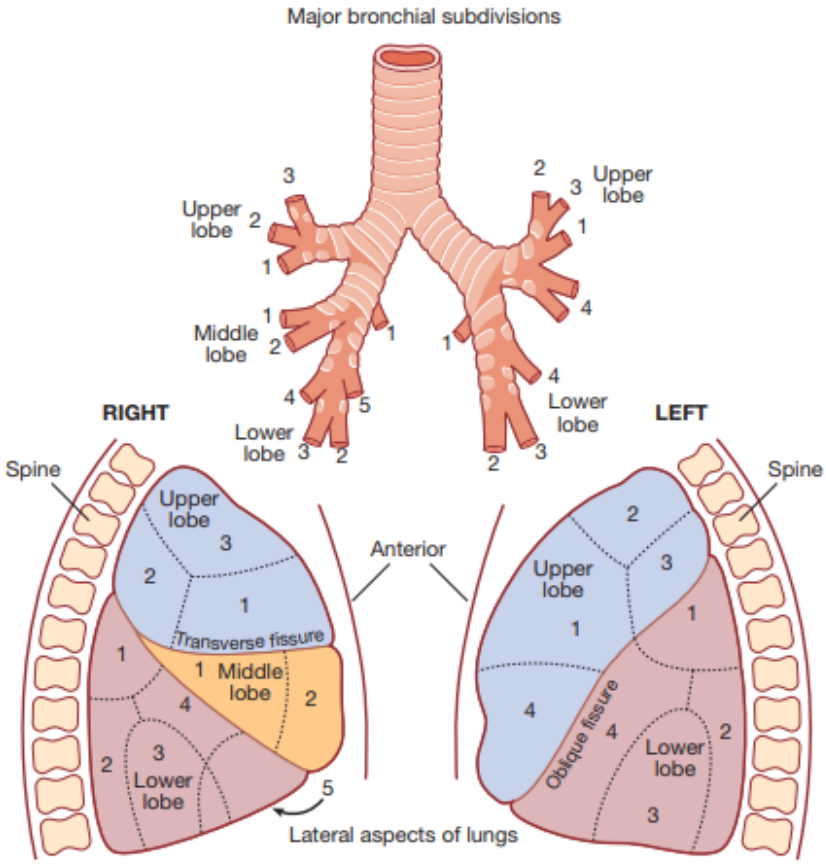


Fig. 17.1 The major bronchial divisions and the fissures, lobes and segments of the lungs. The angle of the oblique fissure means that the left upper lobe is largely anterior to the lower lobe. On the right, the transverse fissure separates the upper from the anteriorly placed middle lobe, which is matched by the lingular segment on the left side. The site of a lobe determines whether physical signs are mainly anterior or posterior. Each lobe is composed of two or more bronchopulmonary segments that are supplied by the main branches of each lobar bronchus. *Bronchopulmonary segments*: **Right** *Upper lobe*: (1) Anterior, (2) Posterior, (3) Apical. *Middle lobe*: (1) Lateral, (2) Medial. *Lower lobe*: (1) Apical, (2) Posterior basal, (3) Lateral basal, (4) Anterior basal, (5) Medial basal. **Left** *Upper lobe*: (1) Anterior, (2) Apical, (3) Posterior, (4) Lingular. *Lower lobe*: (1) Apical, (2) Posterior basal, (3) Lateral basal, (4) Anterior basal.

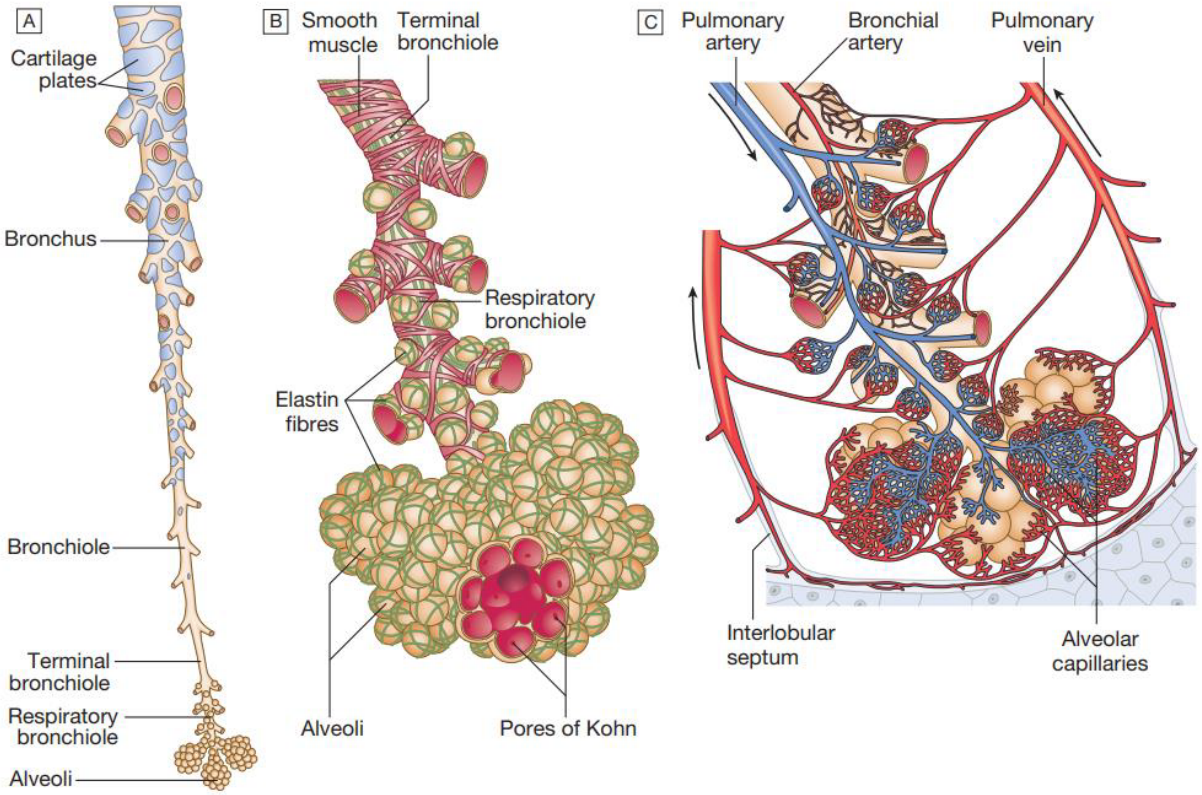


Fig. 17.2 Functional anatomy of the lung. A The tapering, branching bronchus is armoured against compression by plates of cartilage. The more distal bronchioles are collapsible, but held patent by surrounding elastic tissue. B The unit of lung supplied by a terminal bronchiole is called an acinus. The bronchiolar wall contains smooth muscle and elastin fibres. The latter also run through the alveolar walls. Gas exchange occurs in the alveoli, which are connected to each other by the pores of Kohn. C Vascular anatomy of an acinus. Both the pulmonary artery (carrying desaturated blood) and the bronchial artery (systemic supply to airway tissue) run along the bronchus. The venous drainage to the left atrium follows the interlobular septa. *From www.Netter.com: Illustrations 155 (bronchus, acinus) and 191 (circulation), Elsevier.*

17.1 Respiratory function in old age

- **Reserve capacity**: a significant reduction in function can occur with ageing with only minimal effect on normal breathing, but the ability to combat acute disease is reduced.
- **Decline in FEV_1**: the FEV_1/FVC (forced expiratory volume/forced vital capacity, p. 55) ratio falls by around 0.2% per year from 70% at the age of 40–45 years, due to a decline in elastic recoil in the small airways with age. Smoking accelerates this decline threefold on average. Symptoms usually occur only when FEV_1 drops below 50% of predicted.
- **Increasing ventilation–perfusion mismatch**: the reduction in elastic recoil causes a tendency for the small airways to collapse during expiration, particularly in dependent areas of the lungs, thus reducing ventilation.
- **Reduced ventilatory responses to hypoxia and hypercapnia**: older people may be less tachypnoeic for any given fall in PaO_2 or rise in $PaCO_2$.
- **Impaired defences against infection**: due to reduced numbers of glandular epithelial cells, which lead to a reduction in protective mucus.
- **Decline in maximum oxygen uptake**: due to a combination of impairments in muscle, and the respiratory and cardiovascular systems. This leads to a reduction in cardiorespiratory reserve and exercise capacity.
- **Loss of chest wall compliance**: due to reduced intervertebral disc spaces and ossification of the costal cartilages; respiratory muscle strength and endurance also decline. These changes become important only in the presence of other respiratory disease.

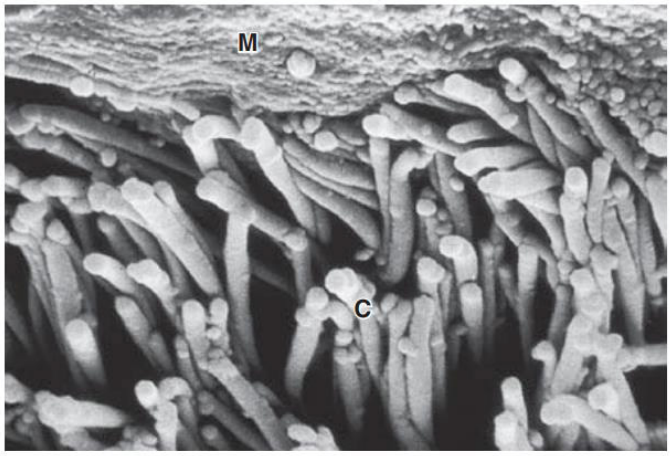


Fig. 17.3 The mucociliary escalator. Scanning electron micrograph of the respiratory epithelium showing large numbers of cilia (C) overlaid by the mucus 'raft' (M).

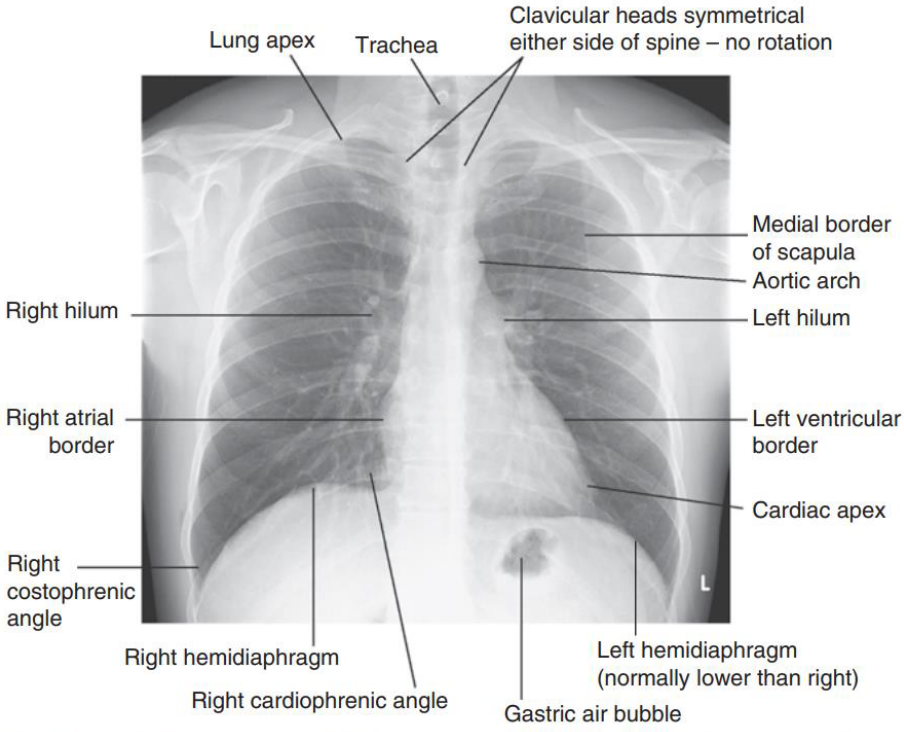


Fig. 17.4 The normal chest X-ray. The lung markings consist of branching and tapering lines radiating out from the hila. Where airways and vessels turn towards the film, they can appear as open or filled circles (see upper pole of right hilum). The scapulae may overlie the lung fields; trace the edge of bony structures to avoid mistaking them for pleural or pulmonary shadows. To check for hyperinflation, count the ribs; if more than 10 are visible posteriorly above the diaphragm, the lungs are hyperinflated. *From Innes JA. Davidson's Essentials of medicine. Edinburgh: Churchill Livingstone, Elsevier Ltd; 2009.*

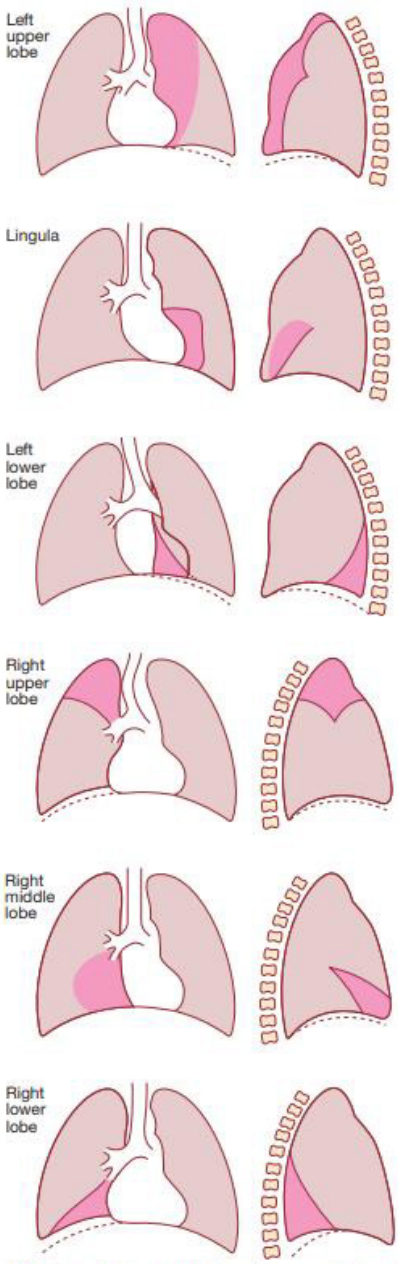


Fig. 17.5 Radiological features of lobar collapse caused by bronchial obstruction. The dotted line in the drawings represents the normal position of the diaphragm. The dark pink area represents the extent of shadowing seen on the X-ray.

17.3 Common chest X-ray abnormalities

Pulmonary and pleural shadowing

- Consolidation: infection, infarction, inflammation and, rarely, bronchoalveolar cell carcinoma
- Lobar collapse: mucus plugging, tumour, compression by lymph nodes
- Solitary nodule: see page 560
- Multiple nodules: miliary tuberculosis (TB), dust inhalation, metastatic malignancy, healed varicella pneumonia, rheumatoid disease
- Ring shadows, tramlines and tubular shadows: bronchiectasis
- Cavitating lesions: tumour, abscess, infarct, pneumonia (*Staphylococcus/Klebsiella*), granulomatosis with polyangiitis (formerly known as Wegener's granulomatosis)
- Reticular, nodular and reticulonodular shadows: diffuse parenchymal lung disease, infection
- Pleural abnormalities: fluid, plaques, tumour

Increased translucency

- Bullae
- Pneumothorax
- Oligaemia

Hilar abnormalities

- Unilateral hilar enlargement: TB, lung cancer, lymphoma
- Bilateral hilar enlargement: sarcoid, lymphoma, TB, silicosis

Other abnormalities

- Hiatus hernia
- Surgical emphysema

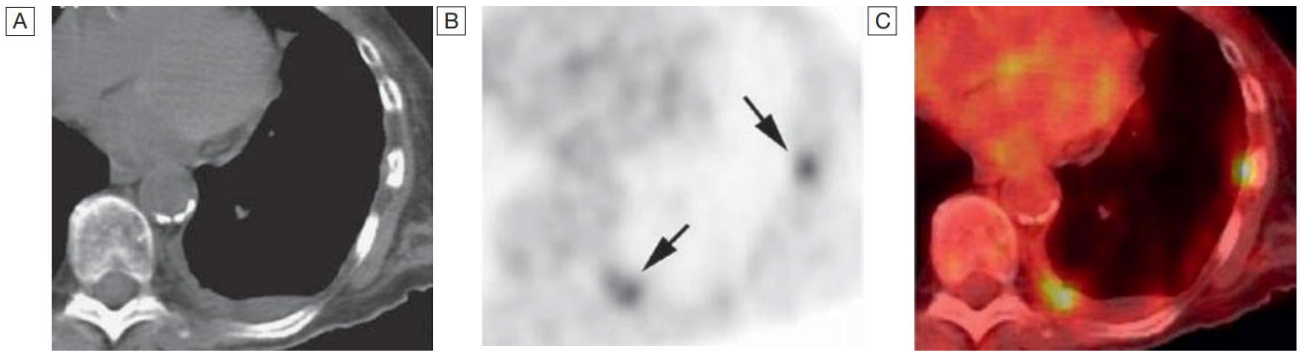


Fig. 17.6 Computed tomography and positron emission tomography combined to reveal intrathoracic metastases. A In a patient with lung cancer, CT shows some posterior pleural thickening. B PET scanning reveals FDG uptake in two pleural lesions (arrows). C The lesions are highlighted in yellow in the combined PET/CT image. *A–C, From http://radiology.rsnajnls.org.*

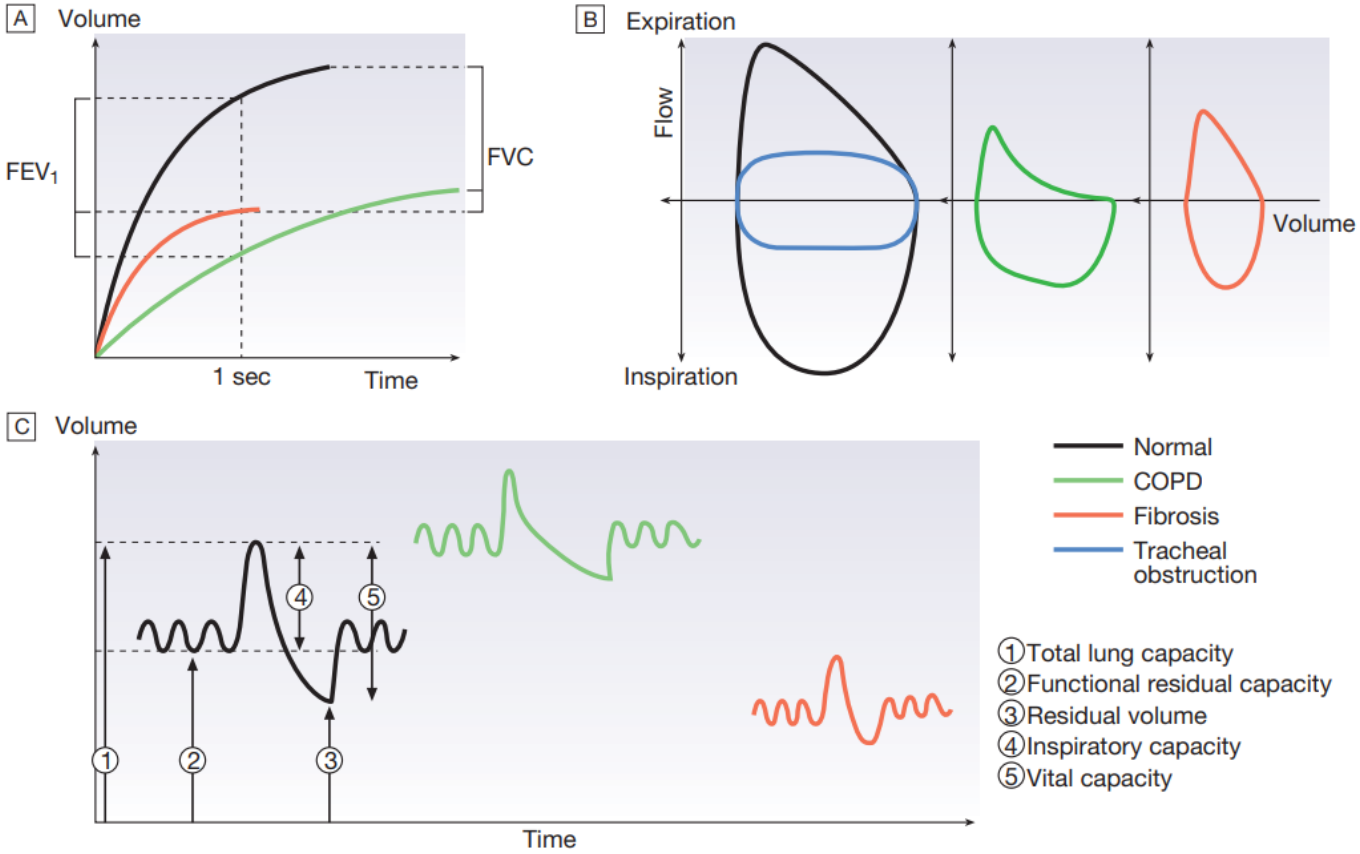


Fig. 17.7 Respiratory function tests in health and disease. A Volume/time traces from forced expiration in health, chronic obstructive pulmonary disease (COPD) and fibrosis. COPD causes slow, prolonged and limited exhalation. In fibrosis, forced expiration results in rapid expulsion of a reduced forced vital capacity (FVC). Forced expiratory volume (FEV_1) is reduced in both diseases but disproportionately so, compared to FVC, in COPD. B The same data plotted as flow/volume loops. In COPD, collapse of intrathoracic airways limits flow, particularly during mid- and late expiration. The blue trace illustrates large airway obstruction, which particularly limits peak flow rates. C Lung volume measurement. Volume/time graphs during quiet breathing with a single maximal breath in and out. COPD causes hyperinflation with increased residual volume. Fibrosis causes a proportional reduction in all lung volumes.

17.4 How to interpret respiratory function abnormalities

	Asthma	Chronic bronchitis	Emphysema	Pulmonary fibrosis
FEV_1	↓↓	↓↓	↓↓	↓
FVC	↓	↓	↓	↓↓
FEV_1/FVC	↓	↓	↓	→/↑
TL_{CO}	→	→	↓↓	↓↓
K_{CO}	→/↑	→	↓	→/↓
TLC	→/↑	↑	↑↑	↓
RV	→/↑	↑	↑↑	↓

(RV = residual volume; TLC = total lung capacity; see text for other abbreviations)

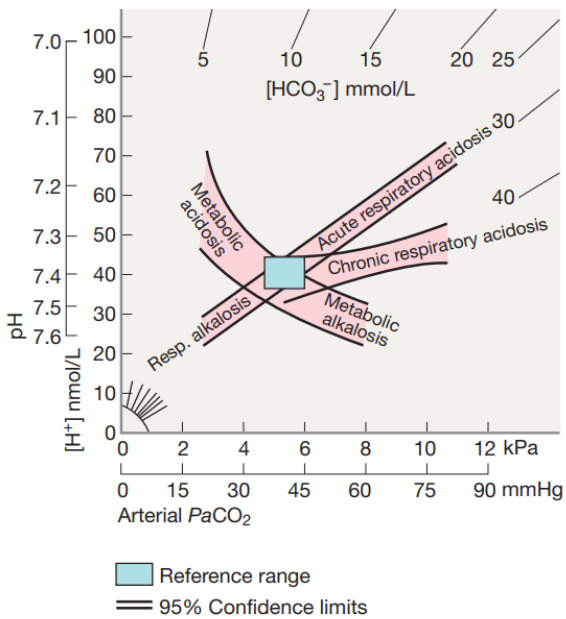


Fig. 17.8 Changes in blood [H^+], $PaCO_2$ and plasma [HCO_3^-] in acid–base disorders. The rectangle indicates normal limits for [H^+] and $PaCO_2$. The bands represent 95% confidence limits of single disturbances in human blood. To determine the likely cause of an acid–base disorder, plot the values of [H^+] and $PaCO_2$ from an arterial blood gas measurement. The diagram indicates whether any acidosis or alkalosis results primarily from a respiratory disorder of $PaCO_2$ or from a metabolic derangement. *Reprinted with permission from Elsevier (Flenley D. Lancet 1971; 1:1921).*

i 17.5 Cough

Origin	Common causes	Clinical features
Pharynx	Post-nasal drip	History of chronic rhinitis
Larynx	Laryngitis, tumour, whooping cough, croup	Voice or swallowing altered, harsh or painful cough Paroxysms of cough, often associated with stridor
Trachea	Tracheitis	Raw retrosternal pain with cough
Bronchi	Bronchitis (acute) and chronic obstructive pulmonary disease (COPD) Asthma Eosinophilic bronchitis Lung cancer	Dry or productive, worse in mornings Usually dry, worse at night Features similar to asthma but airway hyper-reactivity absent Persistent (often with haemoptysis)
Lung parenchyma	Tuberculosis Pneumonia Bronchiectasis Pulmonary oedema Interstitial fibrosis	Productive (often with haemoptysis) Dry initially, productive later Productive, changes in posture induce sputum production Often at night (may be productive of pink, frothy sputum) Dry and distressing
Drug side-effect	Angiotensin-converting enzyme (ACE) inhibitors	Dry cough
Aspiration	Gastro-oesophageal reflux disease (GORD)	History of acid reflux, heartburn, hiatus hernia Obesity

Adapted from Munro JF, Campbell IW. Macleod's Clinical examination, 10th edn. Edinburgh: Churchill Livingstone, Elsevier Ltd; 2000.

i 17.6 Causes of breathlessness

System	Acute dyspnoea	Chronic exertional dyspnoea
Cardiovascular	*Acute pulmonary oedema (p. 463)	Chronic heart failure (p. 463) Myocardial ischaemia (angina equivalent) (p. 180)
Respiratory	*Acute severe asthma *Acute exacerbation of COPD *Pneumothorax *Pneumonia *Pulmonary embolus ARDS Inhaled foreign body (especially in children) Lobar collapse Laryngeal oedema (e.g. anaphylaxis)	*COPD *Chronic asthma Lung cancer Interstitial lung disease (sarcoidosis, fibrosing alveolitis, extrinsic allergic alveolitis, pneumoconiosis) Chronic pulmonary thromboembolism Lymphangitis carcinomatosis (may cause intolerable breathlessness) Large pleural effusion(s)
Others	Metabolic acidosis (e.g. diabetic ketoacidosis, lactic acidosis, uraemia, overdose of salicylates, ethylene glycol poisoning) Psychogenic hyperventilation (anxiety- or panic-related)	Severe anaemia Obesity Deconditioning

*Denotes a common cause.
(ARDS = acute respiratory distress syndrome; COPD = chronic obstructive pulmonary disease)

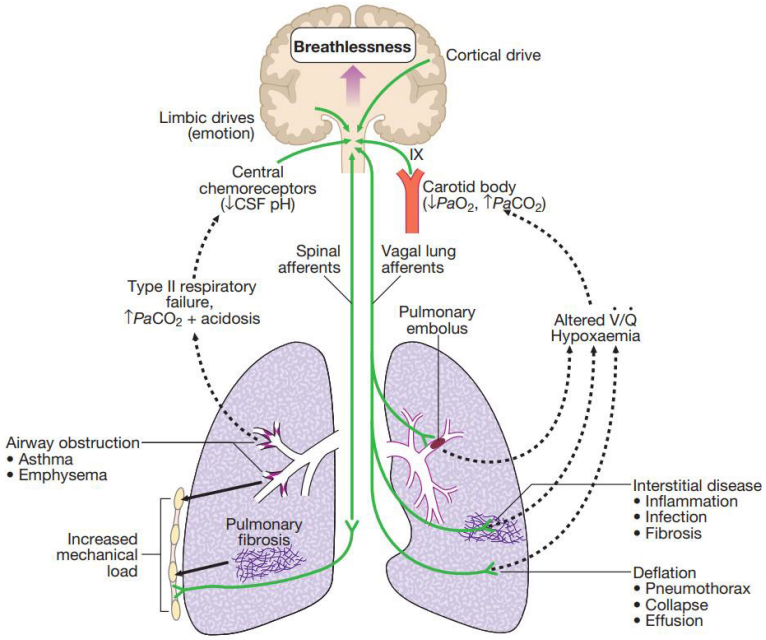


Fig. 17.9 Respiratory stimuli contributing to breathlessness. Mechanisms by which disease can stimulate the respiratory motor neurons in the medulla. Breathlessness is usually felt in proportion to the sum of these stimuli. Further explanation is given on page 179. (CSF = cerebrospinal fluid; V̇/Q̇ = ventilation/perfusion match)

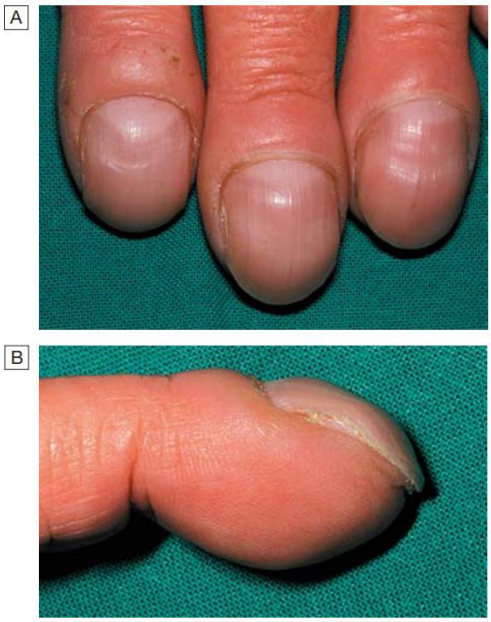


Fig. 17.10 Finger clubbing. A Anterior view. B Lateral view. *From Douglas G, Nicol F, Robertson C. Macleod's Clinical examination, 13th edn. Edinburgh: Churchill Livingstone, Elsevier Ltd; 2013.*

i 17.7 Factors suggesting psychogenic hyperventilation

- 'Inability to take a deep breath'
- Frequent sighing/erratic ventilation at rest
- Short breath-holding time in the absence of severe respiratory disease
- Difficulty in performing and/or inconsistent spirometry measures
- High score (over 26) on Nijmegen questionnaire
- Induction of symptoms during submaximal hyperventilation
- Resting end-tidal $CO_2 < 4.5\%$
- Associated digital and/or perioral paraesthesiae

i 17.8 Differential diagnosis of finger clubbing

Congenital or familial (5–10%)

Acquired

Thoracic (~80%)
- Chronic suppurative conditions:
  - Pulmonary tuberculosis
  - Bronchiectasis
  - Lung abscess
  - Empyema
  - Cystic fibrosis
- Tumours:
  - Lung cancer
  - Mesothelioma
  - Fibroma
- Pulmonary fibrosis

Cardiovascular
- Cyanotic congenital heart disease
- Infective endocarditis
- Arteriovenous shunts and aneurysms

Gastrointestinal
- Cirrhosis
- Inflammatory bowel disease
- Coeliac disease

Others
- Thyrotoxicosis (thyroid acropachy)
- Primary hypertrophic osteoarthropathy

17.9 Causes of haemoptysis

Bronchial disease

- Cancer*
- Bronchiectasis*
- Acute bronchitis*
- Bronchial adenoma
- Foreign body

Parenchymal disease

- Tuberculosis*
- Suppurative pneumonia
- Parasites (e.g. hydatid disease, flukes)
- Lung abscess
- Trauma
- Actinomycosis
- Mycetoma

Lung vascular disease

- Pulmonary infarction*
- Goodpasture's syndrome (p. 612)
- Polyarteritis nodosa
- Idiopathic pulmonary haemosiderosis

Cardiovascular disease

- Acute left ventricular failure*
- Mitral stenosis
- Aortic aneurysm

Blood disorders

- Leukaemia
- Haemophilia
- Anticoagulants

*More common causes.

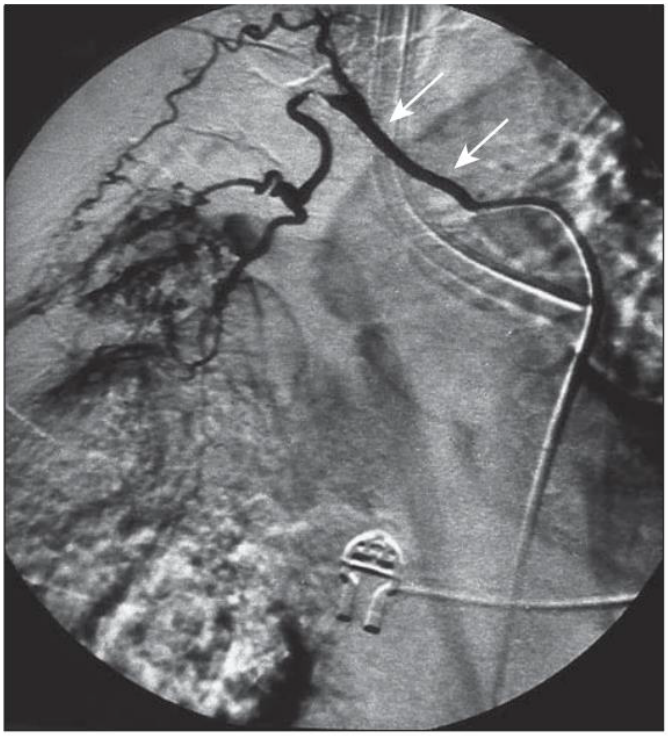


Fig. 17.11 Bronchial artery angiography. An angiography catheter has been passed via the femoral artery and aorta into an abnormally dilated right bronchial artery (arrows). Contrast is seen flowing into the lung. This patient had post-tuberculous bronchiectasis and presented with massive haemoptysis. Bronchial artery embolisation was successfully performed.

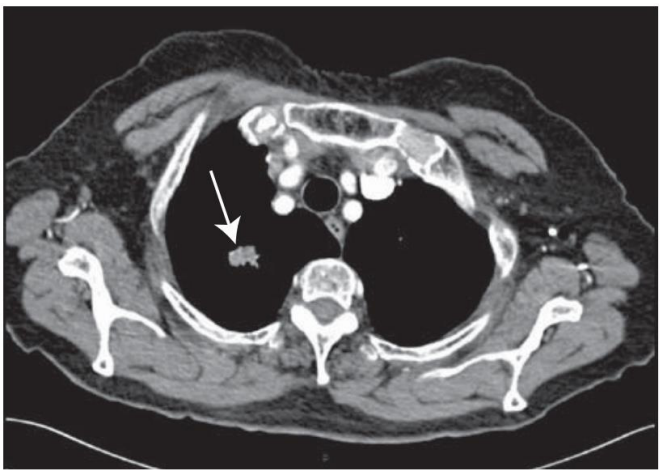


Fig. 17.12 Thoracic CT scan showing a solitary pulmonary nodule identified in the right upper lobe (arrow).

17.10 Causes of pulmonary nodules

Common causes

- Lung cancer
- Single metastasis
- Localised pneumonia
- Lung abscess
- Tuberculoma
- Pulmonary infarct

Uncommon causes

- Benign tumour
- Lymphoma
- Arteriovenous malformation
- Hydatid cyst (p. 298)
- Bronchogenic cyst
- Rheumatoid nodule
- Granulomatosis with polyangiitis (Wegener's granulomatosis)
- Pulmonary sequestration
- Pulmonary haematoma
- 'Pseudotumour' – fluid collection in a fissure
- Aspergilloma (usually surrounded by air crescent)
- *Cryptococcus*
- *Aspergillus* nodule

17.11 Clinical and radiographic features distinguishing benign from malignant nodules

Feature	Risk of malignancy	Feature	Risk of malignancy
Characteristics of nodule		**Characteristics of patient**	
Size	Nearly all >3 cm but fewer than 1% <4 mm are malignant	Age	Increases with age and is uncommon below age of 40
Margin	Usually smooth in benign lesions Spiculated suggests malignancy	Smoking history	Increases in proportion to duration and amount smoked
Calcification or fat	Laminated or central deposition of calcification suggests granuloma 'Popcorn' pattern suggests hamartoma Fat may suggest hamartoma or lipoid granuloma	Other	Increased by history of lung cancer in first-degree relative and by exposure to asbestos, silica, uranium and radon
Location	70% of lung cancers occur in upper lobes Benign lesions are equally distributed throughout upper and lower lobes		

*Linear or sheet-like lung opacities are unlikely to represent neoplasms and do not require follow-up. Some nodular opacities may be sufficiently typical of scarring for follow-up not to be warranted.

Adapted from MacMahon H, Austin JH, Gamsu G, et al. Guidelines for management of small pulmonary nodules detected on CT scans: a statement from the Fleischner Society. Radiology 2005; 237:395–400.

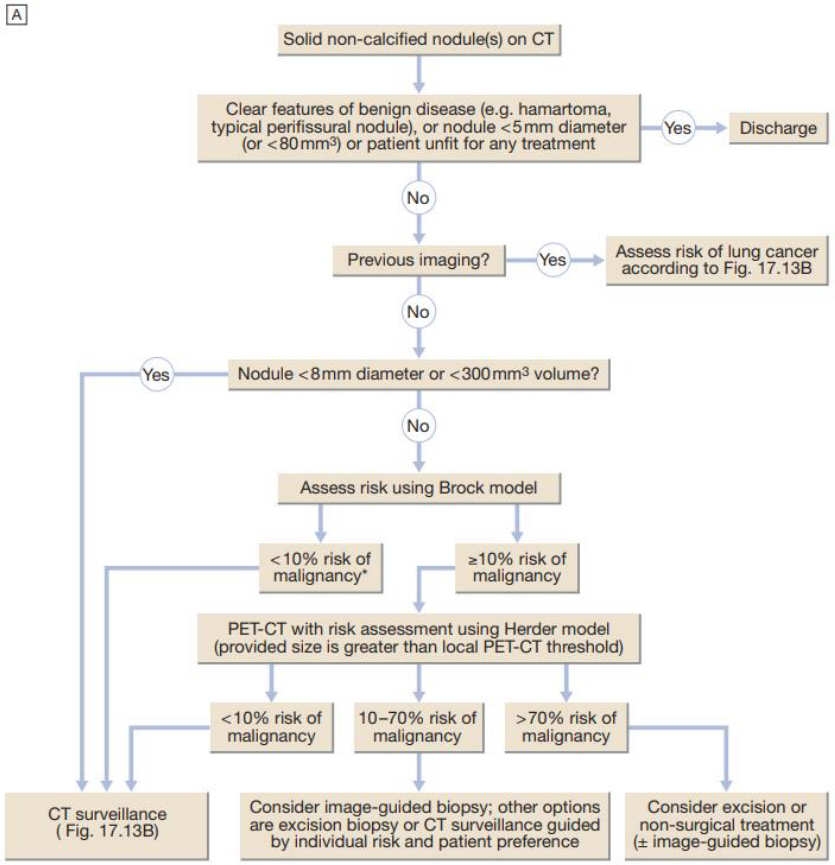


Fig. 17.13 Recommendations on the assessment of a solid pulmonary nodule. A Initial approach to solid pulmonary nodules. The Brock model is an online calculator that can also be downloaded as an app (https://brocku.ca/lung-cancer-risk-calculator). The model integrates data on age, sex, family history of cancer, the presence of emphysema, nodule size, nodule type, nodule position, nodule count and speculation, and calculates the probability that a nodule will become malignant within a 2- to 4-year follow-up period. Herder is a similar model. *Consider positron emission tomography–computed tomography (PET-CT) for larger nodules in young patients with low risk by Brock score, as this score was developed in a screening cohort (50–75 years) and so performance in younger patients is unproven. *Continues overleaf.*

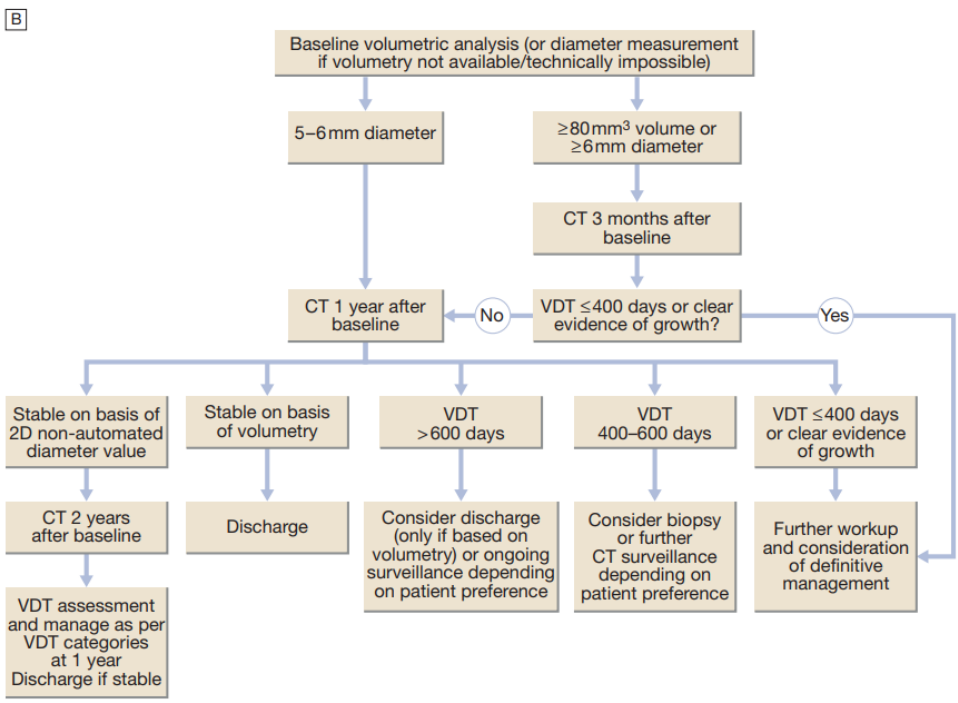


Fig. 17.13, cont'd B Solid pulmonary nodule surveillance algorithm. (VDT = volume doubling time) *From Callister ME, Baldwin DR, Akram AR, et al. British Thoracic Society Guidelines on the investigation and management of pulmonary nodules. Thorax 2015; Suppl. 2:ii1–ii54.*

17.12 Causes of pleural effusion

Common causes

- Pneumonia ('parapneumonic effusion')
- Tuberculosis
- Pulmonary infarction*
- Malignant disease
- Cardiac failure*
- Subdiaphragmatic disorders (subphrenic abscess, pancreatitis etc.)

Uncommon causes

- Hypoproteinaemia* (nephrotic syndrome, liver failure, malnutrition)
- Connective tissue diseases* (particularly systemic lupus erythematosus and rheumatoid arthritis)
- Post-myocardial infarction syndrome
- Acute rheumatic fever
- Meigs' syndrome (ovarian tumour plus pleural effusion)
- Myxoedema*
- Uraemia*
- Asbestos-related benign pleural effusion

*May cause bilateral effusions.

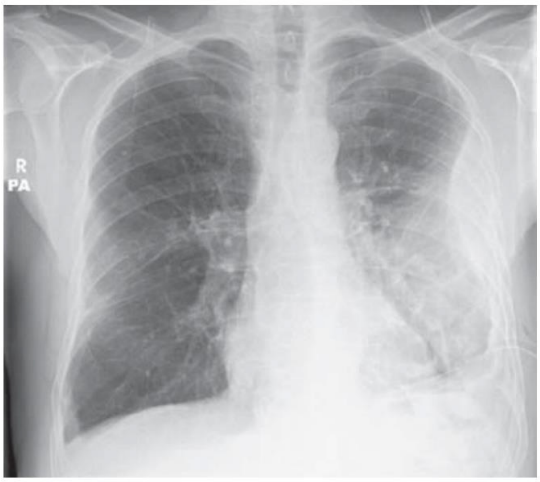


Fig. 17.14 Chest X-ray showing a 'D'-shaped shadow in the left mid-zone, consistent with an empyema. In this case, an intercostal chest drain has been inserted but the loculated collection of pus remains.

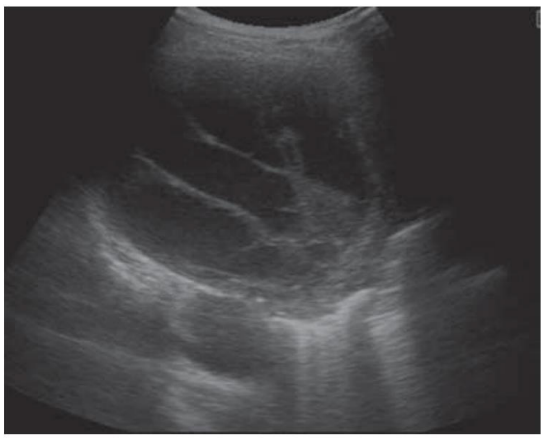


Fig. 17.15 Pleural ultrasound showing septation. *Courtesy of Dr P. Sivasothy, Department of Respiratory Medicine, Addenbrooke's Hospital, Cambridge.*

17.13 Pleural effusion: main causes and features

Cause	Appearance of fluid	Type of fluid	Predominant cells in fluid	Other diagnostic features
Tuberculosis	Serous, usually amber-coloured	Exudate	Lymphocytes (occasionally polymorphs)	Positive tuberculin test Isolation of *Mycobacterium tuberculosis* from pleural fluid (20%) Positive pleural biopsy (80%) Raised adenosine deaminase
Malignant disease	Serous, often blood-stained	Exudate	Serosal cells and lymphocytes Often clumps of malignant cells	Positive pleural biopsy (40%) Evidence of malignancy elsewhere
Cardiac failure	Serous, straw-coloured	Transudate	Few serosal cells	Other signs of cardiac failure Response to diuretics
Pulmonary infarction	Serous or blood-stained	Exudate (rarely transudate)	Red blood cells Eosinophils	Evidence of pulmonary infarction Obvious source of embolism Factors predisposing to venous thrombosis
Rheumatoid disease	Serous Turbid if chronic	Exudate	Lymphocytes (occasionally polymorphs)	Rheumatoid arthritis: rheumatoid factor and anti-cyclic citrullinated peptide (anti-CCP) antibodies Cholesterol in chronic effusion; very low glucose in pleural fluid
Systemic lupus erythematosus (SLE)	Serous	Exudate	Lymphocytes and serosal cells	Other signs of SLE Antinuclear factor or anti-DNA positive
Acute pancreatitis	Serous or blood-stained	Exudate	No cells predominate	Higher amylase in pleural fluid than in serum
Obstruction of thoracic duct	Milky	Chyle	None	Chylomicrons

17.14 Light's criteria for distinguishing pleural transudate from exudate

Exudate is likely if one or more of the following criteria are met:

- Pleural fluid protein:serum protein ratio >0.5
- Pleural fluid LDH:serum LDH ratio >0.6
- Pleural fluid LDH > two-thirds of the upper limit of normal serum LDH

(LDH = lactate dehydrogenase)

17.15 Clinical features of empyema

Systemic features

- Pyrexia, usually high and remittent
- Rigors, sweating, malaise and weight loss
- Polymorphonuclear leucocytosis, high C-reactive protein

Local features

- Pleural pain; breathlessness; cough and sputum, usually because of underlying lung disease; copious purulent sputum if empyema ruptures into a bronchus (bronchopleural fistula)
- Clinical signs of pleural effusion

17.16 How to interpret blood gas abnormalities in respiratory failure

	Type I Hypoxia (PaO_2 <8.0 kPa (60 mmHg)) Normal or low $PaCO_2$ (≤6 kPa (45 mmHg))		Type II Hypoxia (PaO_2 <8.0 kPa (60 mmHg)) Raised $PaCO_2$ (>6 kPa (45 mmHg))	
	Acute	Chronic	Acute	Chronic
H^+	→	→	↑	→ or ↑
Bicarbonate	→	→	→	↑
Causes	Acute asthma Pulmonary oedema Pneumonia Lobar collapse Pneumothorax Pulmonary embolus ARDS	COPD Lung fibrosis Lymphangitic carcinomatosis Right-to-left shunts	Acute severe asthma Acute exacerbation of COPD Upper airway obstruction Acute neuropathies/paralysis Narcotic drugs Primary alveolar hypoventilation Flail chest injury	COPD Sleep apnoea Kyphoscoliosis Myopathies/muscular dystrophy Ankylosing spondylitis

(ARDS = acute respiratory distress syndrome; COPD = chronic obstructive pulmonary disease)

17.17 Assessment and management of 'acute on chronic' type II respiratory failure

Initial assessment

Patient may not appear distressed, despite being critically ill

- Conscious level (response to commands, ability to cough)
- CO_2 retention (warm periphery, bounding pulses, flapping tremor)
- Airways obstruction (wheeze, prolonged expiration, hyperinflation, intercostal indrawing, pursed lips)
- Cor pulmonale (peripheral oedema, raised jugular venous pressure, hepatomegaly, ascites)
- Background functional status and quality of life
- Signs of precipitating cause (see Box 17.15)

Investigations

- Arterial blood gases (severity of hypoxaemia, hypercapnia, acidaemia, bicarbonate)
- Chest X-ray

Management

- Maintenance of airway
- Treatment of specific precipitating cause
- Frequent physiotherapy ± pharyngeal suction
- Nebulised bronchodilators
- Controlled oxygen therapy:
  - Start with 24% Venturi mask
  - Aim for a PaO_2 > 7 kPa (52 mmHg) (a PaO_2 < 5 (37 mmHg) is dangerous)
- Antibiotics if evidence of infection
- Diuretics if evidence of fluid overload

Progress

- If $PaCO_2$ continues to rise or a safe PaO_2 cannot be achieved without severe hypercapnia and acidaemia, mechanical ventilatory support may be required

17.18 Indications for lung transplantation

Parenchymal lung disease

- Cystic fibrosis
- Emphysema
- Pulmonary fibrosis
- Obliterative bronchiolitis
- Langerhans cell histiocytosis (p. 613)
- Lymphangioleiomyomatosis (p. 613)

Pulmonary vascular disease

- Primary pulmonary hypertension
- Thromboembolic pulmonary hypertension
- Veno-occlusive disease
- Eisenmenger's syndrome (p. 532)

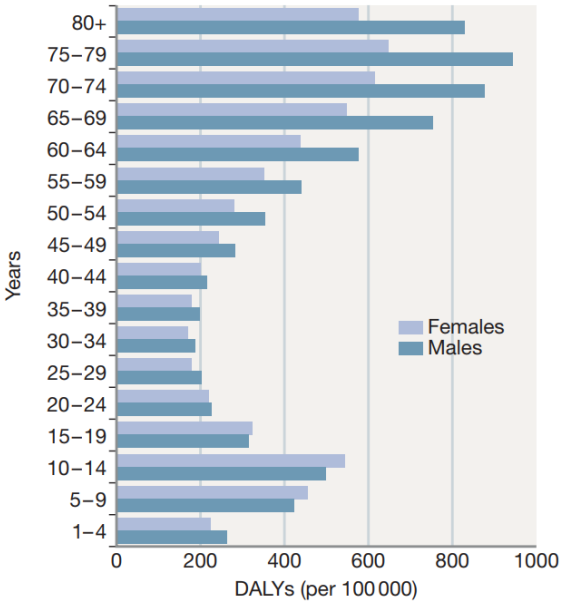


Fig. 17.16 The burden of asthma, measured by disability life years (DALYs) per 100 000 population. The burden of asthma is greatest in children approaching adolescence and the elderly. The burden is similar in males and females at ages below 30–34 but at older ages the burden is higher in males. *From The Global Asthma Report 2014. Copyright 2014 The Global Asthma Network.*

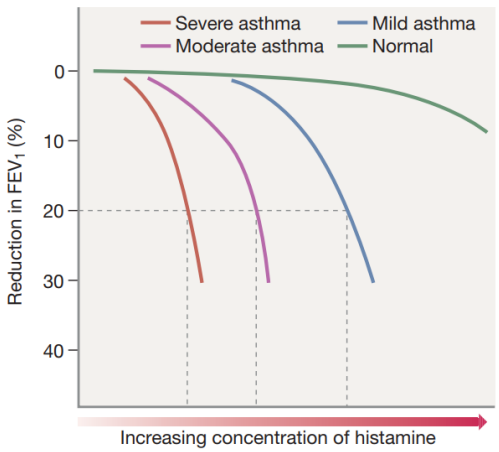


Fig. 17.17 Airway hyper-reactivity in asthma. This is demonstrated by bronchial challenge tests with sequentially increasing concentrations of either histamine, or methacholine or mannitol. The reactivity of the airways is expressed as the concentration or dose of either chemical required to produce a specific decrease (usually 20%) in the forced expired volume in 1 second (FEV_1) (PC_{20} or PD_{20}, respectively).

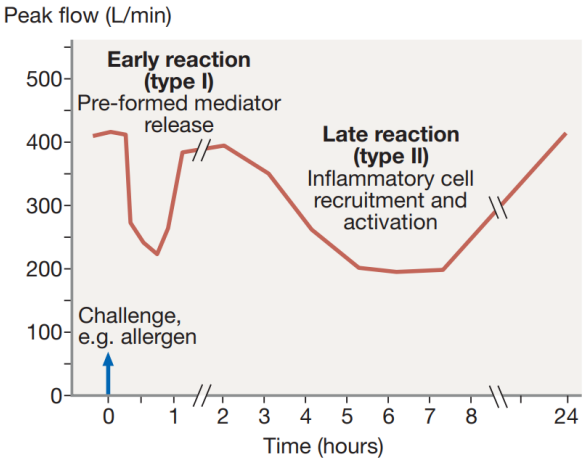


Fig. 17.18 Changes in peak flow following allergen challenge. A similar biphasic response is observed following a variety of different challenges. Occasionally, an isolated late response is seen with no early reaction.

17.19 How to make a diagnosis of asthma

Compatible clinical history *plus either/or*:

- FEV_1 ≥12% (and 200 mL) increase following administration of a bronchodilator/trial of glucocorticoids. Greater confidence is gained if the increase is >15% and >400 mL
- >20% diurnal variation on ≥ 3 days in a week for 2 weeks on PEF diary
- FEV_1 ≥15% decrease after 6 mins of exercise

(FEV_1 = forced expiratory volume in 1 sec; PEF = peak expiratory flow)

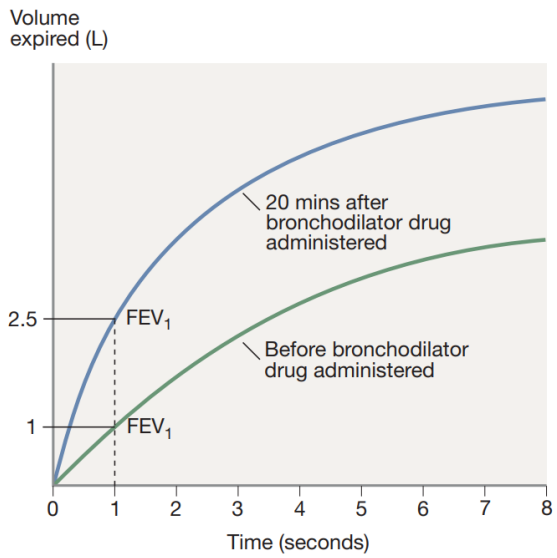


Fig. 17.19 Reversibility test. Forced expiratory manœuvres before and 20 minutes after inhalation of a β_2-adrenoceptor agonist. Note the increase in forced expiratory volume in 1 second (FEV_1) from 1.0 to 2.5 L.

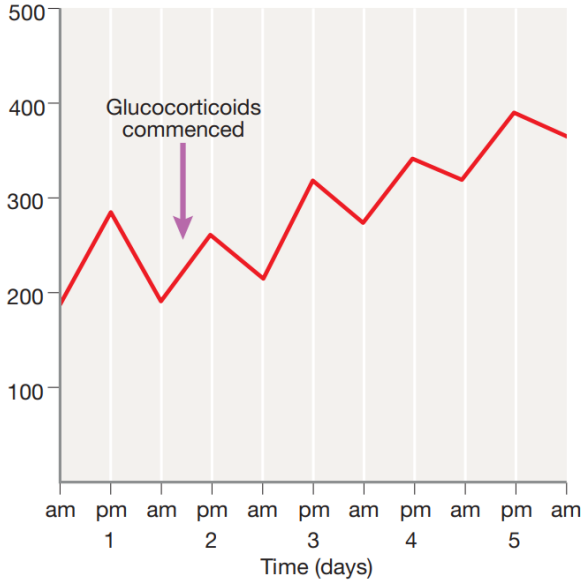


Fig. 17.20 Serial recordings of peak expiratory flow (PEF) in a patient with asthma. Note the sharp overnight fall (morning dip) and subsequent rise during the day. Following the introduction of glucocorticoids, there is an improvement in PEF rate and reduction of morning dipping.

i 17.20 Levels of asthma control

Characteristic	Controlled	Partly controlled (any present in any week)	Uncontrolled
Daytime symptoms	None (≤ twice/week)	> twice/week	≥ 3 features of partly controlled asthma present in any week
Limitations of activities	None	Any	
Nocturnal symptoms/awakening	None	Any	
Need for rescue/'reliever' treatment	None (≤ twice/week)	> twice/week	
Lung function (PEF or FEV_1)	Normal	< 80% predicted or personal best (if known) on any day	
Exacerbation	None	≥ 1/year	1 in any week

(FEV1 = forced expiratory volume in 1 sec; PEF = peak expiratory flow)

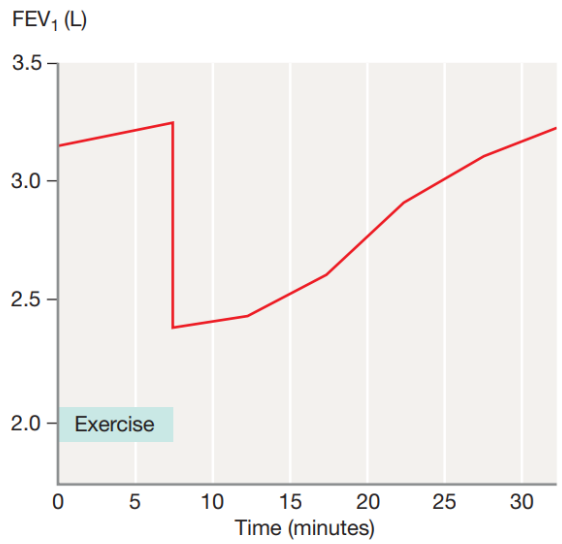


Fig. 17.21 Exercise-induced asthma. Serial recordings of forced expiratory volume in 1 second (FEV_1) in a patient with bronchial asthma before and after 6 minutes of strenuous exercise. Note initial rise on completion of exercise, followed by sudden fall and gradual recovery. Adequate warm-up exercise or pre-treatment with a β_2-adrenoceptor agonist, nedocromil sodium or a leukotriene antagonist can protect against exercise-induced symptoms.

17.21 Asthma in pregnancy

- **Clinical course**: women with well-controlled asthma usually have good pregnancy outcomes. Pregnancy in women with more severe asthma can precipitate worsening control and lead to increased maternal and neonatal morbidity.
- **Labour and delivery**: 90% have no symptoms.
- **Safety data**: good for β_2-agonists, inhaled glucocorticoids, theophyllines, oral prednisolone, and chromones.
- **Oral leukotriene receptor antagonists**: no evidence that these harm the fetus and they should not be stopped in women who have previously demonstrated significant improvement in asthma control prior to pregnancy.
- **Glucocorticoids**: women on maintenance prednisolone >7.5 mg/day should receive hydrocortisone 100 mg 3–4 times daily during labour.
- **Prostaglandin $F_{2\alpha}$**: may induce bronchospasm and should be used with extreme caution.
- **Breastfeeding**: use medications as normal.
- **Uncontrolled asthma**: associated with maternal (hyperemesis, hypertension, pre-eclampsia, vaginal haemorrhage, complicated labour) and fetal (intrauterine growth restriction and low birth weight, preterm birth, increased perinatal mortality, neonatal hypoxia) complications.

- Remove the cap and shake the inhaler
- Breathe out gently and place the mouthpiece into the mouth
- Incline the head backwards to minimise oropharyngeal deposition
- Simultaneously, begin a slow deep inspiration, depress the canister and continue to inhale
- Hold the breath for 10 seconds

Fig. 17.23 How to use a metered-dose inhaler.

17.22 Immediate assessment of acute severe asthma

Acute severe asthma

- PEF 33–50% predicted (<200 L/min)
- Heart rate ≥110 beats/min
- Respiratory rate ≥25 breaths/min
- Inability to complete sentences in 1 breath

Life-threatening features

- PEF <33% predicted (<100 L/min)
- SpO_2 <92% or PaO_2 <8 kPa (60 mmHg) (especially if being treated with oxygen)
- Normal or raised $PaCO_2$
- Silent chest
- Cyanosis
- Feeble respiratory effort
- Bradycardia or arrhythmias
- Hypotension
- Exhaustion
- Delirium
- Coma

Near-fatal asthma

- Raised $PaCO_2$ and/or requiring mechanical ventilation with raised inflation pressures

(PEF = peak expiratory flow)

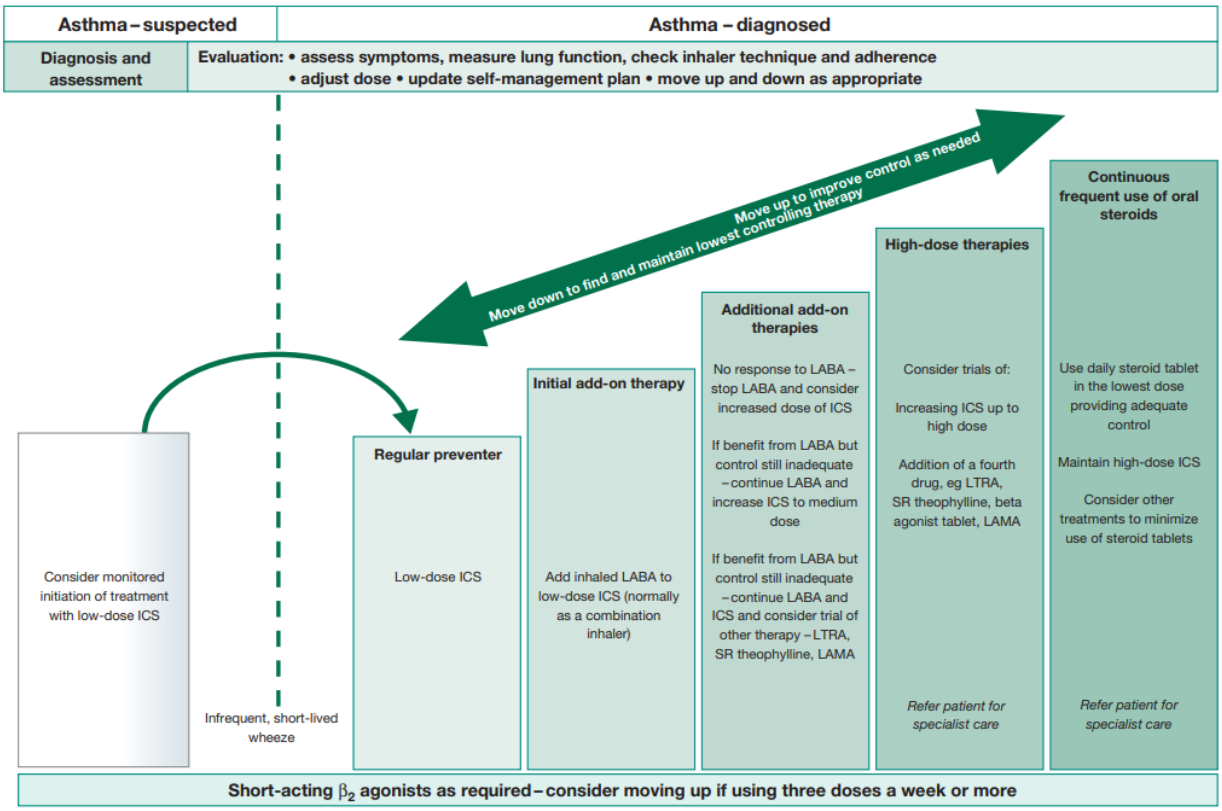


Fig. 17.22 Management approach in adults based on asthma control. (ICS = inhaled corticosteroids (glucocorticoids); LABA = long-acting β_2-agonist; LAMA = long-acting muscarinic antagonist; LTRA = leukotriene receptor antagonist; SR = sustained-release) *From British Thoracic Society and SIGN guideline 153: British guideline on the management of asthma (2016).*

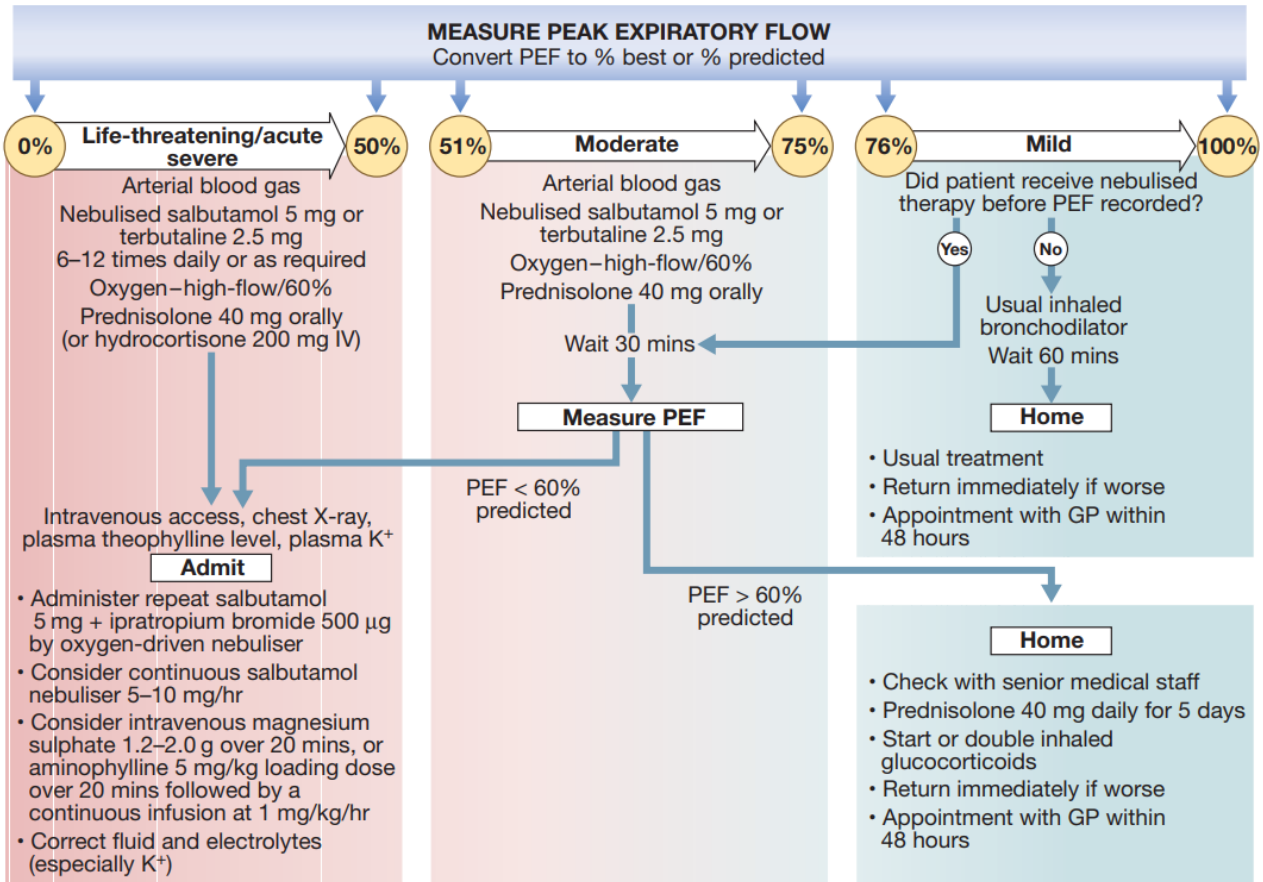


Fig. 17.24 Immediate treatment of patients with acute severe asthma.

17.23 Indications for assisted ventilation in acute severe asthma

- Coma
- Respiratory arrest
- Deterioration of arterial blood gas tensions despite optimal therapy:
  - PaO_2 <8 kPa (60 mmHg) and falling
  - $PaCO_2$ >6 kPa (45 mmHg) and rising
  - pH low and falling (H^+ high and rising)
- Exhaustion, delirium, drowsiness

17.24 Risk factors for development of chronic obstructive pulmonary disease

Environmental factors

- Tobacco smoke: accounts for 95% of cases in the UK
- Indoor air pollution: cooking with biomass fuels in confined areas in developing countries
- Occupational exposures, such as coal dust, silica and cadmium
- Low birth weight: may reduce maximally attained lung function in young adult life
- Lung growth: childhood infections or maternal smoking may affect growth of lung during childhood, resulting in a lower maximally attained lung function in adult life
- Infections: recurrent infection may accelerate decline in FEV_1; persistence of adenovirus in lung tissue may alter local inflammatory response, predisposing to lung damage; HIV infection is associated with emphysema
- Low socioeconomic status
- Cannabis smoking

Host factors

- Genetic factors: α_1-antitrypsin deficiency; other COPD susceptibility genes are likely to be identified
- Airway hyper-reactivity

(FEV_1 = forced expiratory volume in 1 sec)

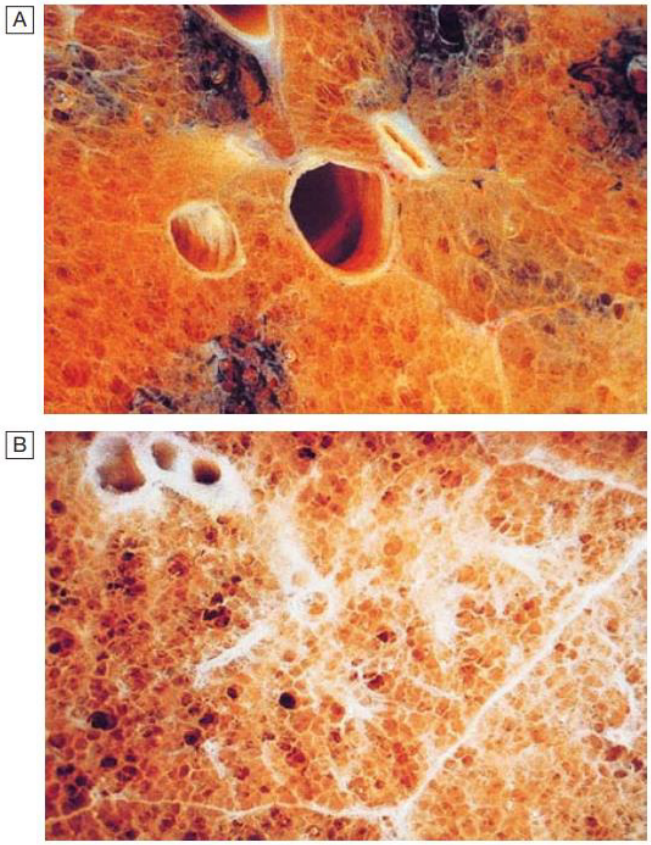


Fig. 17.26 The pathology of emphysema. A Normal lung. B Emphysematous lung, showing gross loss of the normal surface area available for gas exchange. *B, Courtesy of the British Lung Foundation.*

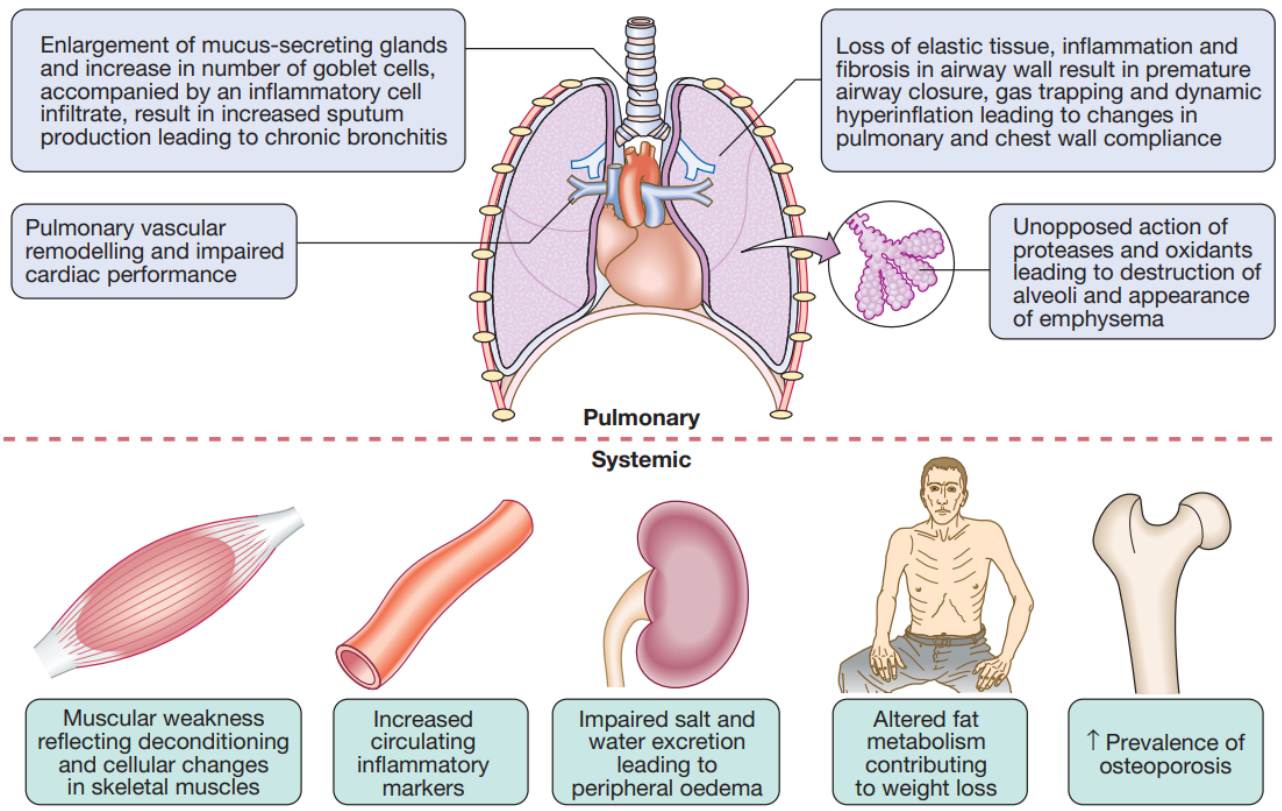


Fig. 17.25 The pulmonary and systemic features of chronic obstructive pulmonary disease.

17.25 Modified Medical Research Council (MRC) dyspnoea scale

Grade	Degree of breathlessness related to activities
0	No breathlessness, except with strenuous exercise
1	Breathlessness when hurrying on the level or walking up a slight hill
2	Walks slower than contemporaries on level ground because of breathlessness or has to stop for breath when walking at own pace
3	Stops for breath after walking about 100 m or after a few minutes on level ground
4	Too breathless to leave the house, or breathless when dressing or undressing

17.26 Spirometric classification of COPD severity based on post-bronchodilator FEV_1

		Severity of airflow obstruction post-bronchodilator		
PD FEV_1/FVC	FEV_1% predicted	ATS/ERS (2004)	GOLD (2008)	NICE Clinical Guideline 101 (2010)
<0.7	≥80%	Mild	Stage I – mild	Stage I – mild[1]
<0.7	50–79%	Moderate	Stage II – moderate	Stage II – moderate
<0.7	30–49%	Severe	Stage III – severe	Stage III – severe
<0.7	<30%	Very severe	Stage IV – very severe[2]	Stage IV – very severe[2]

[1]Mild COPD should not be diagnosed on lung function alone if the patient is asymptomatic. [2]Or FEV_1 <50% with respiratory failure.
(ATS/ERS = American Thoracic Society/European Respiratory Journal; FEV_1 = forced expiratory volume in 1 sec; GOLD = Global Initiative for Chronic Obstructive Lung Disease; PD = post-bronchodilator)
Adapted from National Institute for Health and Care Excellence (NICE) CG101 – Chronic obstructive pulmonary disease in over 16s: diagnosis and management; 2010.

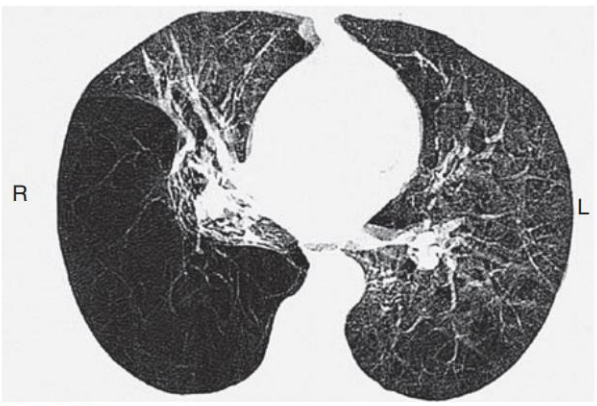


Fig. 17.27 Gross emphysema. High-resolution computed tomogram showing emphysema, most evident in the right lower lobe.

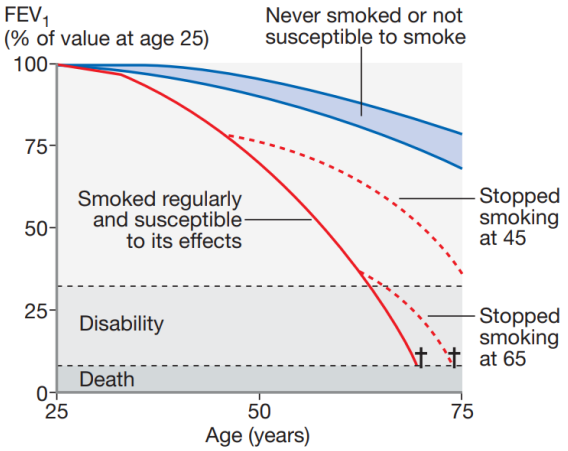


Fig. 17.28 Model of annual decline in FEV_1 with accelerated decline in susceptible smokers. When smoking is stopped, subsequent loss is similar to that in healthy non-smokers. (FEV_1 = forced expiratory volume in 1 second)

17.27 Prescription of long-term oxygen therapy in COPD

Arterial blood gases are measured in clinically stable patients on optimal medical therapy on at least two occasions 3 weeks apart:

- PaO_2 <7.3 kPa (55 mmHg) irrespective of $PaCO_2$ and FEV_1 <1.5 L
- PaO_2 7.3–8 kPa (55–60 mmHg) plus pulmonary hypertension, peripheral oedema or nocturnal hypoxaemia
- the patient has stopped smoking

Use at least 15 hrs/day at 2–4 L/min to achieve a PaO_2 >8 kPa (60 mmHg) without unacceptable rise in $PaCO_2$

17.28 Calculation of the BODE index

	Points on BODE index			
Variable	0	1	2	3
FEV_1	≥65	50–64	36–49	≤35
Distance walked in 6 mins (m)	≥350	250–349	150–249	≤149
MRC dyspnoea scale*	0–1	2	3	4
Body mass index	>21	≤21		

A patient with a BODE score of 0–2 has a mortality rate of around 10% at 52 months, whereas a patient with a BODE score of 7–10 has a mortality rate of around 80% at 52 months.

*See Box 17.25.
(BODE – see text; FEV_1 = forced expiratory volume in 1 sec)

17.29 Obstructive pulmonary disease in old age

- **Asthma**: may appear de novo in old age, so airflow obstruction should not always be assumed to be due to COPD.
- **Peak expiratory flow recordings**: older people with poor vision have difficulty reading PEF meters.
- **Perception of bronchoconstriction**: impaired by age, so an older patient's description of symptoms may not be a reliable indicator of severity.
- **Stopping smoking**: the benefits on the rate of loss of lung function decline with age but remain valuable up to the age of 80.
- **Metered-dose inhalers**: many older people cannot use these because of difficulty coordinating and triggering the device. Even mild cognitive impairment virtually precludes their use. Frequent demonstration and re-instruction in the use of all devices are required.
- **Mortality rates for acute asthma**: higher in old age, partly because patients under-estimate the severity of bronchoconstriction and also develop a lower degree of tachycardia and pulsus paradoxus for the same degree of bronchoconstriction.
- **Treatment decisions**: advanced age in itself is not a barrier to intensive care or mechanical ventilation in an acute episode of asthma or COPD, but this decision may be difficult and should be shared with the patient (if possible), the relatives and the GP.

17.30 Causes of bronchiectasis

Congenital

- Cystic fibrosis
- Ciliary dysfunction syndromes:
  - Primary ciliary dyskinesia (immotile cilia syndrome)
  - Kartagener's syndrome (sinusitis and transposition of the viscera)
- Primary hypogammaglobulinaemia (p. 79)

Acquired: children

- Severe infections in infancy (especially whooping cough, measles)
- Primary tuberculosis
- Inhaled foreign body

Acquired: adults

- Suppurative pneumonia
- Pulmonary tuberculosis
- Allergic bronchopulmonary aspergillosis complicating asthma (p. 596)
- Bronchial tumours

17.31 Symptoms of bronchiectasis

- **Cough**: chronic, daily, persistent
- **Sputum**: copious, continuously purulent
- **Pleuritic pain**: when infection spreads to involve pleura, or with segmental collapse due to retained secretions
- **Haemoptysis**:
  - Streaks of blood common, larger volumes with exacerbations of infection
  - Massive haemoptysis requiring bronchial artery embolisation sometimes occurs
- **Infective exacerbation**: increased sputum volume with fever, malaise, anorexia
- **Halitosis**: frequently accompanies purulent sputum
- **General debility**: difficulty maintaining weight, anorexia, exertional breathlessness

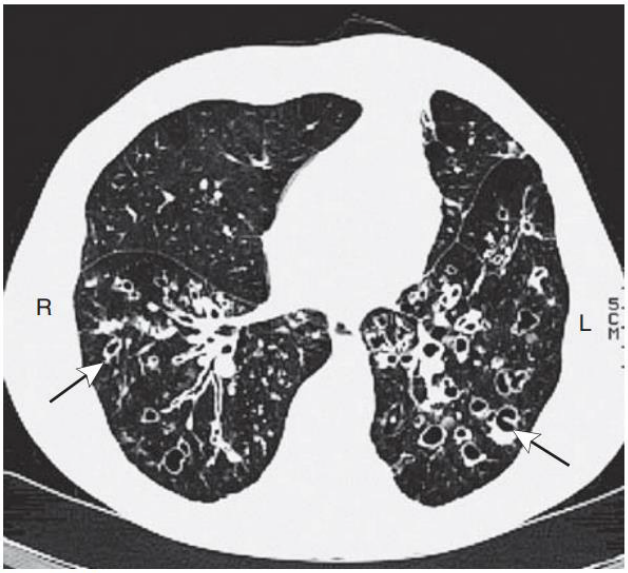


Fig. 17.29 Computed tomogram of bronchiectasis. Extensive dilatation of the bronchi, with thickened walls (arrows) in both lower lobes.

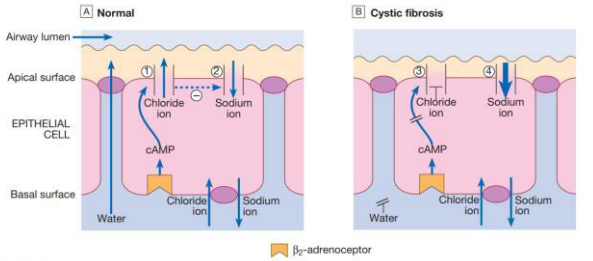


Fig. 17.30 Cystic fibrosis: basic defect in the pulmonary epithelium. A The cystic fibrosis gene (*CFTR*) codes for a chloride channel (1) in the apical (luminal) membrane of epithelial cells in the conducting airways. This is normally controlled by cyclic adenosine monophosphate (cAMP) and indirectly by β-adrenoceptor stimulation, and is one of several apical ion channels that control the quantity and solute content of airway-lining fluid. Normal channels appear to inhibit the adjacent epithelial sodium channels (2). B In cystic fibrosis, one of many cystic fibrosis gene defects causes absence or defective function of this chloride channel (3). This leads to reduced chloride secretion and loss of inhibition of sodium channels, with excessive sodium resorption (4) and dehydration of the airway lining. The resulting abnormal airway-lining fluid predisposes to infection by mechanisms still to be fully explained.

17.32 Complications of cystic fibrosis

Respiratory

- Progressive airway obstruction
- Infective exacerbations of bronchiectasis
- Respiratory failure
- Spontaneous pneumothorax
- Haemoptysis
- Lobar collapse due to secretions
- Pulmonary hypertension
- Nasal polyps

Gastrointestinal and hepatic

- Malabsorption and steatorrhoea
- Distal intestinal obstruction syndrome
- Biliary cirrhosis
- Portal hypertension, varices and splenomegaly
- Gallstones

Others

- Diabetes (25% of adults)
- Delayed puberty
- Male infertility
- Stress incontinence due to repeated forced cough
- Psychosocial problems
- Osteoporosis
- Arthropathy
- Cutaneous vasculitis

17.33 Treatments that reduce chest exacerbations and/or improve lung function in cystic fibrosis

Therapy	Patients treated
Nebulised recombinant human DNase 2.5 mg daily	Age ≥5, FVC >40% predicted
Nebulised tobramycin 300 mg twice daily, given in alternate months	Patients colonised with *Pseudomonas aeruginosa*
Regular oral azithromycin 500 mg 3 times a week	Patients colonised with *P. aeruginosa*
Nebulised hypertonic saline 4 mL 7%, twice daily	Age ≥6, FEV_1 > 40% predicted

(FEV_1 = forced expiratory volume in 1 sec; FVC = forced vital capacity)

17.34 Cystic fibrosis in adolescence

Issues for the patient

- Move to adult CF centre – loss of trusted paediatric team
- Feelings of being different from peers due to chronic illness
- Demanding treatments that conflict with social and school life
- Pressure to take responsibility for self-care
- Relationship/fertility concerns

Issues for the patient's parents

- Loss of control over patient's treatment – feeling excluded
- Loss of trusted paediatric team
- Need to develop trust in adult team
- Feelings of helplessness when adolescent rebels or will not take treatment

Issues for the CF team

- Reluctance or refusal by patient to engage with transition
- Management of deterioration due to non-adherence
- Motivation of adolescents to self-care
- Provision of adolescent-friendly health-care environment

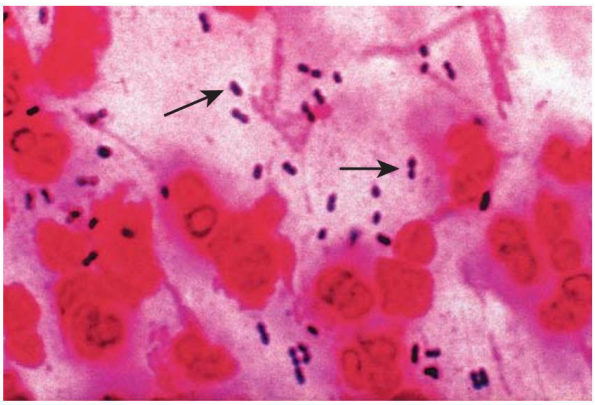


Fig. 17.31 Gram stain of sputum showing Gram-positive diplococci characteristic of *Streptococcus pneumoniae* (arrows).

17.35 Factors that predispose to pneumonia

- Cigarette smoking
- Upper respiratory tract infections
- Alcohol
- Glucocorticoid therapy
- Old age
- Recent influenza infection
- Pre-existing lung disease
- HIV
- Indoor air pollution

17.36 Organisms causing community-acquired pneumonia

Bacteria

- *Streptococcus pneumoniae*
- *Mycoplasma pneumoniae*
- *Legionella pneumophila*
- *Chlamydia pneumoniae*
- *Haemophilus influenzae*
- *Staphylococcus aureus*
- *Chlamydia psittaci*
- *Coxiella burnetii* (Q fever)
- *Klebsiella pneumoniae* (Freidländer's bacillus)

Viruses

- Influenza, parainfluenza
- Measles
- Herpes simplex
- Varicella
- Adenovirus
- Cytomegalovirus
- Coronaviruses (SARS-CoV and MERS-CoV)

(MERS = Middle East respiratory syndrome; SARS = severe acute respiratory syndrome)

17.37 Differential diagnosis of pneumonia

- Pulmonary infarction
- Pulmonary/pleural tuberculosis
- Pulmonary oedema (can be unilateral)
- Pulmonary eosinophilia (p. 611)
- Malignancy: bronchoalveolar cell carcinoma
- Cryptogenic organising pneumonia/bronchiolitis obliterans organising pneumonia (COP/BOOP) (p. 606)

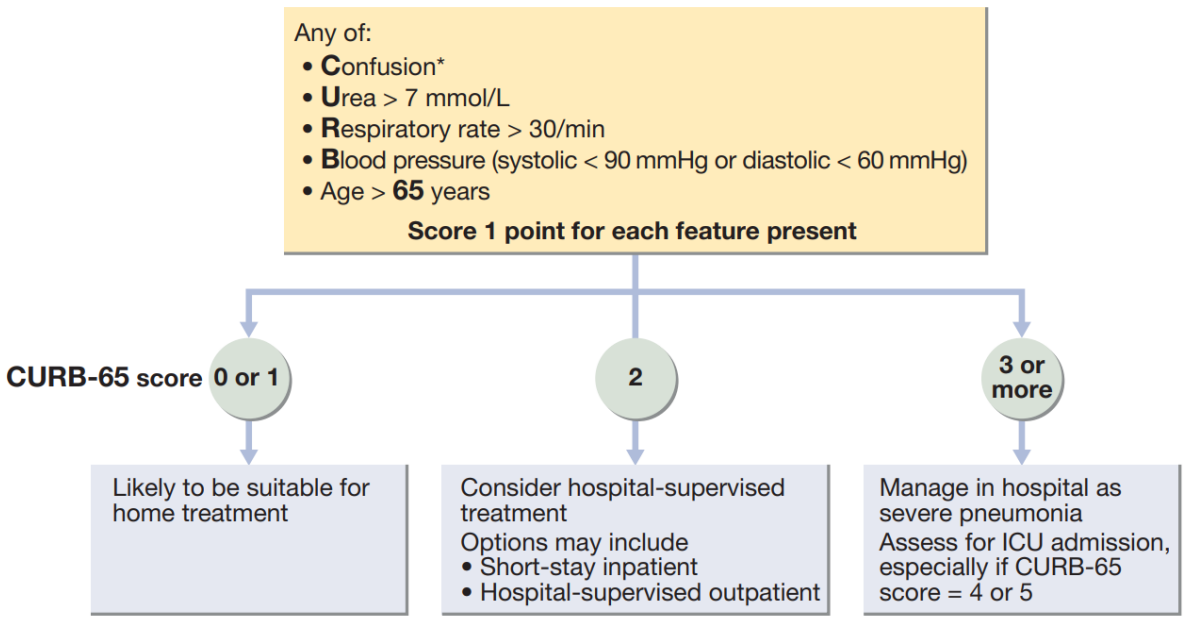


Fig. 17.32 **Hospital CURB-65.** *Defined as a mental test score of 8 or less, or new disorientation in person, place or time. (ICU = intensive care unit; urea of 7 mmol/L ≅ 20 mg/dL)

17.38 Investigations in community-acquired pneumonia

Blood

Full blood count
- Very high (>20×10⁹/L) or low (<4×10⁹/L) white cell count: marker of severity
- Neutrophil leucocytosis >15×10⁹/L: suggests bacterial aetiology
- Haemolytic anaemia: occasional complication of *Mycoplasma*

Urea and electrolytes
- Urea >7 mmol/L (~20 mg/dL): marker of severity
- Hyponatraemia: marker of severity

Liver function tests
- Abnormal if basal pneumonia inflames liver
- Hypoalbuminaemia: marker of severity

Erythrocyte sedimentation rate/C-reactive protein
- Non-specifically elevated

Blood culture
- Bacteraemia: marker of severity

Cold agglutinins
- Positive in 50% of patients with *Mycoplasma*

Arterial blood gases
- Measure when SaO_2 <93% or when clinical features are severe, to assess ventilatory failure or acidosis

Sputum

Sputum samples
- Gram stain (see Fig. 17.31), culture and antimicrobial sensitivity testing

Oropharynx swab
- Polymerase chain reaction for *Mycoplasma pneumoniae* and other atypical pathogens

Urine
- Pneumococcal and/or *Legionella* antigen

Chest X-ray

Lobar pneumonia
- Patchy opacification evolves into homogeneous consolidation of affected lobe
- Air bronchogram (air-filled bronchi appear lucent against consolidated lung tissue) may be present (Fig. 17.33)

Bronchopneumonia
- Typically patchy and segmental shadowing

Complications
- Para-pneumonic effusion, intrapulmonary abscess or empyema

Staphylococcus aureus
- Suggested by multilobar shadowing, cavitation, pneumatoceles and abscesses

Pleural fluid
- Always aspirate and culture when present in more than trivial amounts, preferably with ultrasound guidance

17.39 Indications for referral to ITU

- CURB score of 4–5 (see Fig. 17.32), failing to respond rapidly to initial management
- Persisting hypoxia (PaO_2 <8 kPa (60 mmHg)), despite high concentrations of oxygen
- Progressive hypercapnia
- Severe acidosis
- Circulatory shock
- Reduced conscious level

17.41 Complications of pneumonia

- Para-pneumonic effusion – common
- Empyema (p. 564)
- Retention of sputum causing lobar collapse
- Deep vein thrombosis and pulmonary embolism
- Pneumothorax, particularly with *Staphylococcus aureus*
- Suppurative pneumonia/lung abscess
- ARDS, renal failure, multi-organ failure (p. 198)
- Ectopic abscess formation (*Staph. aureus*)
- Hepatitis, pericarditis, myocarditis, meningoencephalitis
- Arrhythmias (e.g. atrial fibrillation)
- Pyrexia due to drug hypersensitivity

(ARDS = acute respiratory distress syndrome)

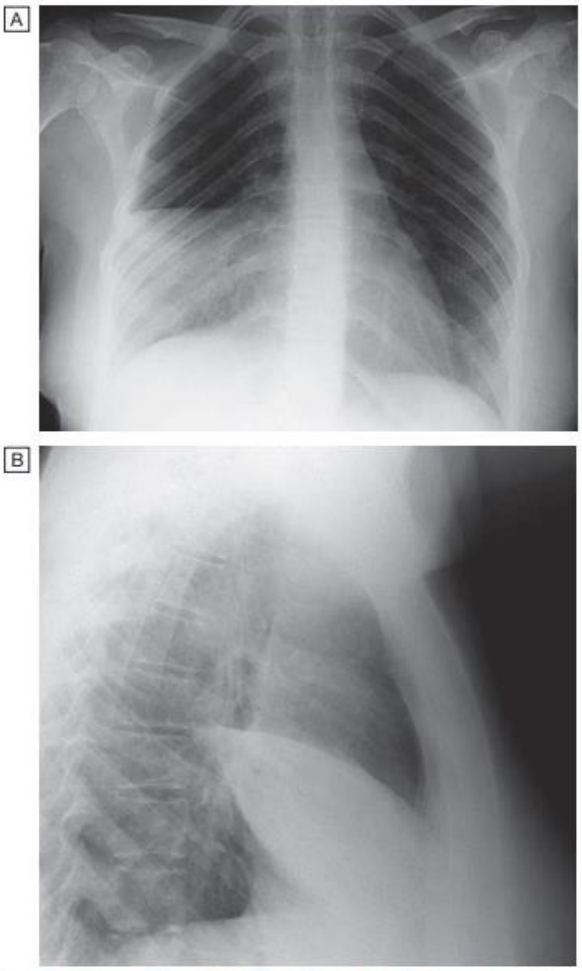


Fig. 17.33 Pneumonia of the right middle lobe. A Posteroanterior view: consolidation in the right middle lobe with characteristic opacification beneath the horizontal fissure and loss of normal contrast between the right heart border and lung. B Lateral view: consolidation confined to the anteriorly situated middle lobe.

17.40 Antibiotic treatment for community-acquired pneumonia*

Uncomplicated CAP
- Amoxicillin 500 mg 3 times daily orally

If patient is allergic to penicillin
- Clarithromycin 500 mg twice daily orally ***or*** Erythromycin 500 mg 4 times daily orally

If *Staphylococcus* is cultured or suspected
- Flucloxacillin 1–2 g 4 times daily IV ***plus***
- Clarithromycin 500 mg twice daily IV

If *Mycoplasma* or *Legionella* is suspected
- Clarithromycin 500 mg twice daily orally or IV ***or*** Erythromycin 500 mg 4 times daily orally IV ***plus***
- Rifampicin 600 mg twice daily IV in severe cases

Severe CAP
- Clarithromycin 500 mg twice daily IV ***or*** Erythromycin 500 mg 4 times daily IV ***plus***
- Co-amoxiclav 1.2 g 3 times daily IV ***or*** Ceftriaxone 1–2 g daily IV ***or*** Cefuroxime 1.5 g 3 times daily IV ***or***
- Amoxicillin 1 g 4 times daily IV ***plus*** flucloxacillin 2 g 4 times daily IV

*Antibiotic use in individual patients should take into account local guidance and antibiotic sensitivity patterns.
Adapted from British Thoracic Society Guidelines.

17.42 Factors predisposing to hospital-acquired pneumonia

Reduced host defences against bacteria

- Reduced immune defences (e.g. glucocorticoid treatment, diabetes, malignancy)
- Reduced cough reflex (e.g. post-operative)
- Disordered mucociliary clearance (e.g. anaesthetic agents)
- Bulbar or vocal cord palsy

Aspiration of nasopharyngeal or gastric secretions

- Immobility or reduced conscious level
- Vomiting, dysphagia (N.B. stroke disease), achalasia or severe reflux
- Nasogastric intubation

Bacteria introduced into lower respiratory tract

- Endotracheal intubation/tracheostomy
- Infected ventilators/nebulisers/bronchoscopes
- Dental or sinus infection

Bacteraemia

- Abdominal sepsis
- Intravenous cannula infection
- Infected emboli

17.43 Respiratory infection in old age

- **Increased risk of and from respiratory infection**: because of reduced immune responses, increased closing volumes, reduced respiratory muscle strength and endurance, altered mucus layer, poor nutritional status and the increased prevalence of chronic lung disease.
- **Predisposing factors**: other medical conditions may predispose to infection, e.g. swallowing difficulties due to stroke increase the risk of aspiration pneumonia.
- **Atypical presentation**: older patients often present with delirium, rather than breathlessness or cough.
- **Mortality**: the vast majority of deaths from pneumonia in developed countries occur in older people.
- **Influenza**: has a much higher complication rate, and morbidity and mortality. Vaccination significantly reduces morbidity and mortality in old age but uptake is poor.
- **Tuberculosis**: most cases in old age represent reactivation of previous, often unrecognised, disease and may be precipitated by glucocorticoid therapy, diabetes mellitus and the factors above. Cryptic miliary tuberculosis is an occasional alternative presentation. Older people more commonly suffer adverse effects from antituberculous chemotherapy and require close monitoring.

17.44 Clinical features of suppurative pneumonia

Symptoms

- Cough with large amounts of sputum, sometimes fetid and blood-stained
- Pleural pain common
- Sudden expectoration of copious amounts of foul sputum if abscess ruptures into a bronchus

Clinical signs

- High remittent pyrexia
- Profound systemic upset
- Digital clubbing may develop quickly (10–14 days)
- Consolidation on chest examination; signs of cavitation rarely found
- Pleural rub common
- Rapid deterioration in general health, with marked weight loss if not adequately treated

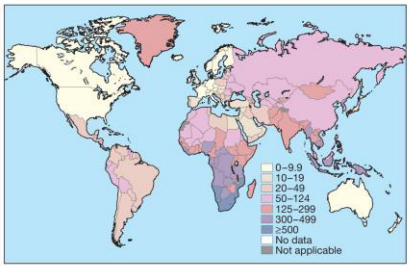


Fig. 17.34 Worldwide incidence of tuberculosis (2014). Estimated new cases (all forms) per 100 000 population. *From World Health Organisation. Global tuberculosis report, 20th edn. Geneva: WHO; 2015.*

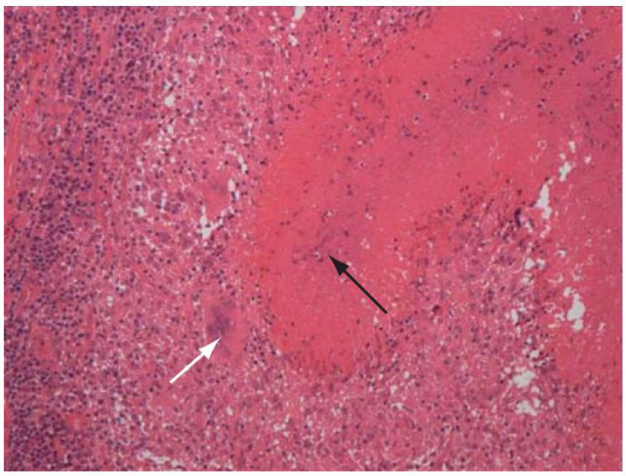


Fig. 17.35 Tuberculous granuloma. Normal lung tissue is lost and replaced by a mass of fibrous tissue with granulomatous inflammation characterised by large numbers of macrophages and multinucleate giant cells (white arrow). The central area of this focus shows caseous degeneration (black arrow). *Courtesy of Dr William Wallace, Department of Pathology, Royal Infirmary of Edinburgh.*

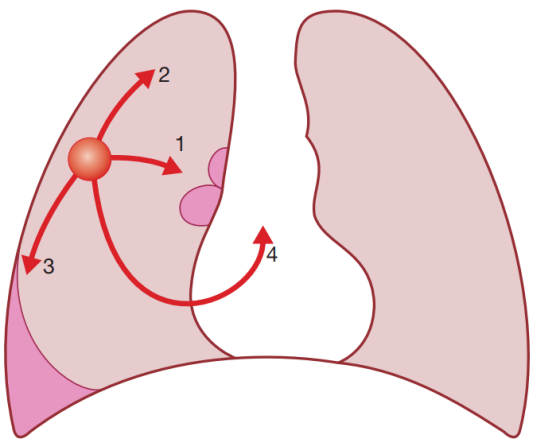


Fig. 17.36 Primary pulmonary tuberculosis. (1) Spread from the primary focus to hilar and mediastinal lymph glands to form the 'primary complex', which heals spontaneously in most cases. (2) Direct extension of the primary focus – progressive pulmonary tuberculosis. (3) Spread to the pleura – tuberculous pleurisy and pleural effusion. (4) Blood-borne spread: *few bacilli* – pulmonary, skeletal, renal, genitourinary infection, often months or years later; *massive spread* – miliary pulmonary tuberculosis and meningitis.

17.45 Factors increasing the risk of tuberculosis

Patient-related

- Age (children > young adults < elderly)
- First-generation immigrants from high-prevalence countries
- Close contacts of patients with smear-positive pulmonary TB
- Overcrowding (prisons, collective dormitories); homelessness (doss houses and hostels)
- Chest X-ray evidence of self-healed TB
- Primary infection <1 year previously
- Smoking: cigarettes, bidis (Indian cigarettes made of tobacco wrapped in temburini leaves) and cannabis

Associated diseases

- Immunosuppression: HIV, anti-tumour necrosis factor (TNF) and other biologic therapies, high-dose glucocorticoids, cytotoxic agents
- Malignancy (especially lymphoma and leukaemia)
- Diabetes mellitus
- Chronic kidney disease
- Silicosis
- Gastrointestinal disease associated with malnutrition (gastrectomy, jejuno-ileal bypass, cancer of the pancreas, malabsorption)
- Deficiency of vitamin D or A
- Recent measles in children

17.46 Natural history of untreated primary tuberculosis

Time from infection	Manifestations
3–8 weeks	Primary complex, positive tuberculin skin test
3–6 months	Meningeal, miliary and pleural disease
Up to 3 years	Gastrointestinal, bone and joint, and lymph node disease
Around 8 years	Renal tract disease
From 3 years onwards	Post-primary disease due to reactivation or re-infection

Adapted from Davies PDO, ed. Clinical tuberculosis. London: Hodder Arnold; 1998.

17.47 Features of primary tuberculosis

Infection (4–8 weeks)

- Influenza-like illness
- Skin test conversion
- Primary complex

Disease

- Lymphadenopathy: hilar (often unilateral), paratracheal or mediastinal
- Collapse (especially right middle lobe)
- Consolidation (especially right middle lobe)
- Obstructive emphysema
- Cavitation (rare)
- Pleural effusion
- Miliary
- Meningitis
- Pericarditis

Hypersensitivity

- Erythema nodosum
- Phlyctenular conjunctivitis
- Dactylitis

17.48 Cryptic tuberculosis

- Age over 60 years
- Intermittent low-grade pyrexia of unknown origin
- Unexplained weight loss, general debility (hepatosplenomegaly in 25–50%)
- Normal chest X-ray
- Blood dyscrasias; leukaemoid reaction, pancytopenia
- Negative tuberculin skin test
- Confirmation by biopsy with granulomas and/or acid-fast bacilli in liver or bone marrow

17.49 Clinical presentations of pulmonary tuberculosis

- Chronic cough, often with haemoptysis
- Pyrexia of unknown origin
- Unresolved pneumonia
- Exudative pleural effusion
- Asymptomatic (diagnosis on chest X-ray)
- Weight loss, general debility
- Spontaneous pneumothorax

17.50 Complications of chronic pulmonary tuberculosis

Pulmonary

- Massive haemoptysis
- Cor pulmonale
- Fibrosis/emphysema
- Atypical mycobacterial infection
- Lung/pleural calcification
- Aspergilloma/chronic aspergillosis
- Obstructive airways disease
- Bronchiectasis
- Bronchopleural fistula

Non-pulmonary

- Empyema necessitans
- Laryngitis
- Enteritis*
- Anorectal disease*
- Amyloidosis
- Poncet's polyarthritis

*From swallowed sputum.

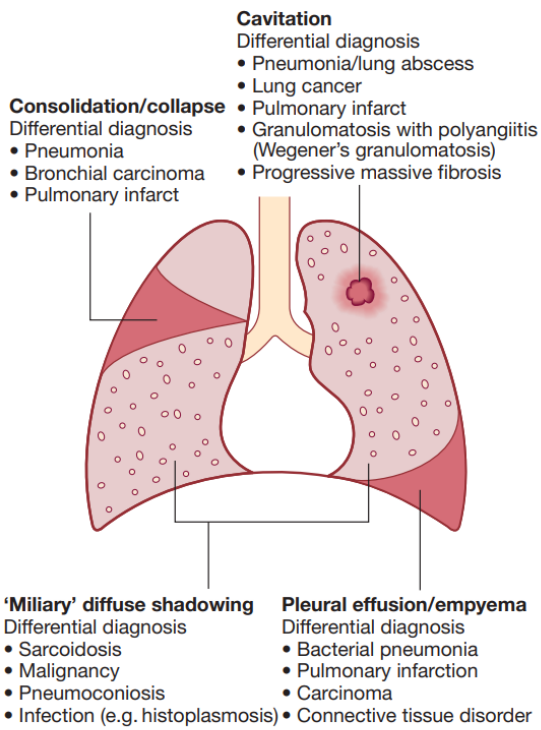


Fig. 17.37 Chest X-ray: major manifestations and differential diagnosis of pulmonary tuberculosis. Less common manifestations include pneumothorax, acute respiratory distress syndrome (ARDS; p. 198), cor pulmonale and localised emphysema.

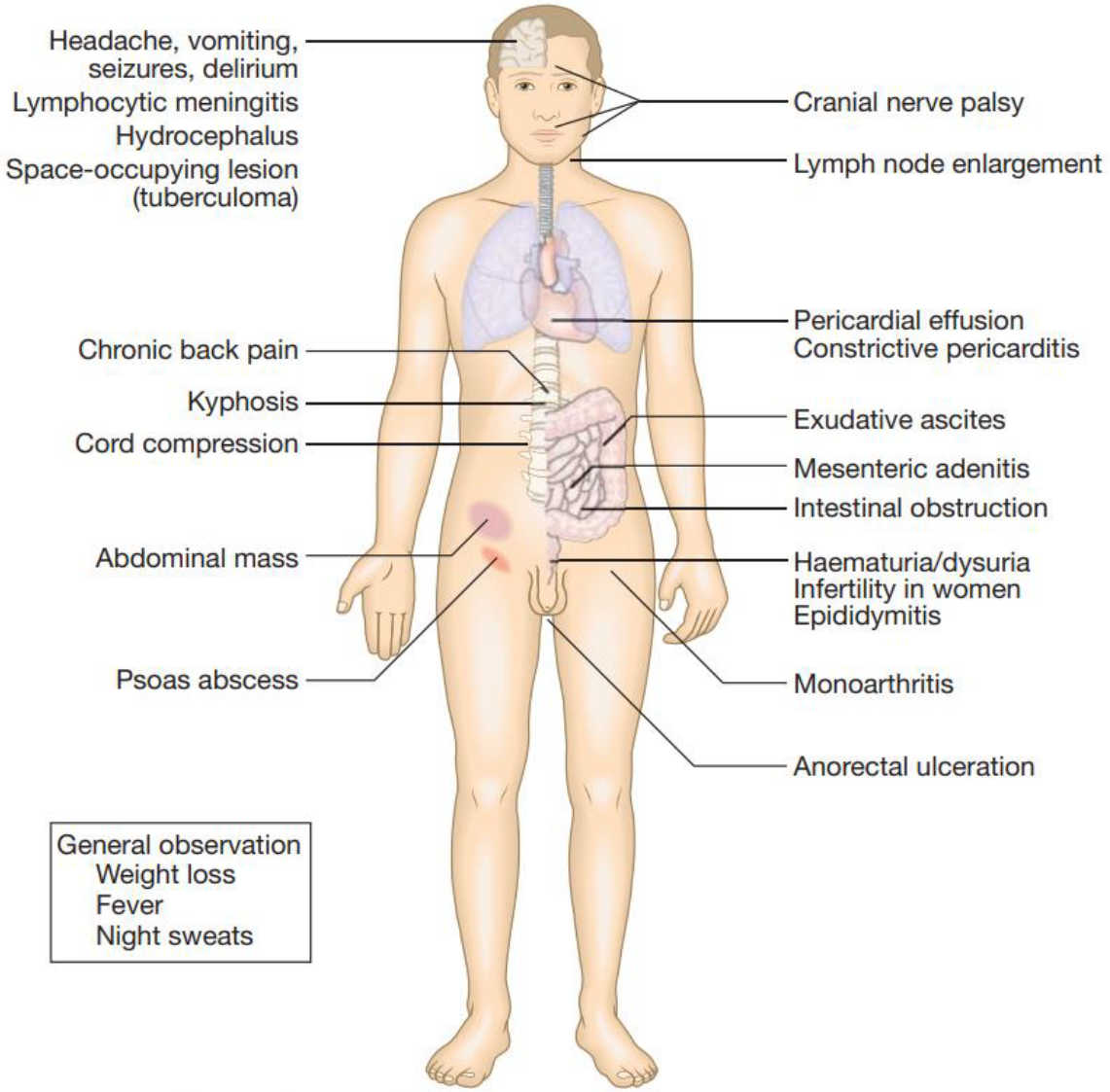


Fig. 17.38 **Systemic presentations of extrapulmonary tuberculosis.**

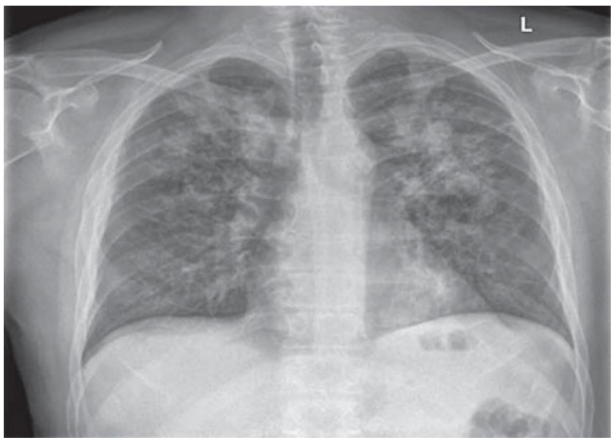


Fig. 17.39 **Typical changes of tuberculosis.** The chest X-ray shows bilateral upper lobe airspace shadowing with cavitation.

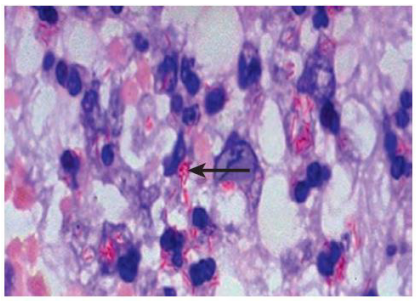


Fig. 17.40 **Positive Ziehl–Neelsen stain.** Mycobacteria (arrow) retain the red carbol fuchsin stain, despite washing with acid and alcohol. *Courtesy of Adam Hill.*

17.51 Diagnosis of tuberculosis

Specimens required

Pulmonary

- Sputum* (induced with nebulised hypertonic saline if patient not expectorating)
- Bronchoscopy with washings or BAL
- Gastric washing* (mainly used for children)

Extrapulmonary

- Fluid examination (cerebrospinal, ascitic, pleural, pericardial, joint): yield classically very low
- Tissue biopsy (from affected site): bone marrow/liver may be diagnostic in disseminated disease

Diagnostic tests

- Tuberculin skin test: low sensitivity/specificity; useful only in primary or deep-seated infection
- Stain
  - Ziehl–Neelsen
  - Auramine fluorescence
- Nucleic acid amplification
- Culture
  - Solid media (Löwenstein–Jensen, Middlebrook)
  - Liquid media (e.g. MGIT)
- Pleural fluid: adenosine deaminase
- Response to empirical antituberculous drugs (usually seen after 5–10 days)

Baseline blood tests

- Full blood count, C-reactive protein, erythrocyte sedimentation rate, urea and electrolytes, liver function tests

*At least two but preferably three, including an early morning sample.
(BAL = bronchoalveolar lavage; MGIT = mycobacteria growth indicator tube)

i 17.52 Treatment of new tuberculosis patients (World Health Organisation recommendations)

Intensive phase	Continuation phase	Comments
Standard regimen		
2 months of HRZE	4 months of HR	
2 months of HRZE	4 months of HRE	Applies only in countries with high levels of isoniazid resistance in new TB patients, and where isoniazid drug susceptibility testing in new patients is not done (or results are unavailable) before the continuation phase begins
Dosing frequency		
Daily*	Daily	Optimal
Daily*	3 times/week	Acceptable alternative for any new patient receiving directly observed therapy
3 times/week	3 times/week	Acceptable alternative, provided that the patient is receiving directly observed therapy and is NOT living with HIV or living in an HIV-prevalent setting

*Daily (rather than 3 times weekly) intensive-phase dosing may help to prevent acquired drug resistance in TB patients starting treatment with isoniazid resistance.
(H = isoniazid; R = rifampicin; Z = pyrazinamide; E = ethambutol)
Adapted from World Health Organisation. Treatment of tuberculosis guidelines, 4th edn; 2010.

i 17.53 Main adverse reactions of first-line antituberculous drugs

	Isoniazid	Rifampicin	Pyrazinamide	Streptomycin	Ethambutol
Mode of action	Cell wall synthesis	DNA transcription	Unknown	Protein synthesis	Cell wall synthesis
Major adverse reactions	Peripheral neuropathy[1] Hepatitis[2] Rash	Febrile reactions Hepatitis Rash Gastrointestinal disturbance	Hepatitis Gastrointestinal disturbance Hyperuricaemia	8th nerve damage Rash	Retrobulbar neuritis[3] Arthralgia
Less common adverse reactions	Lupoid reactions Seizures Psychoses	Interstitial nephritis Thrombocytopenia Haemolytic anaemia	Rash Photosensitisation Gout	Nephrotoxicity Agranulocytosis	Peripheral neuropathy Rash

[1]The risk may be reduced by prescribing pyridoxine. [2]More common in patients with a slow acetylator status and in alcoholics. [3]Reduced visual acuity and colour vision may be reported with higher doses and are usually reversible.

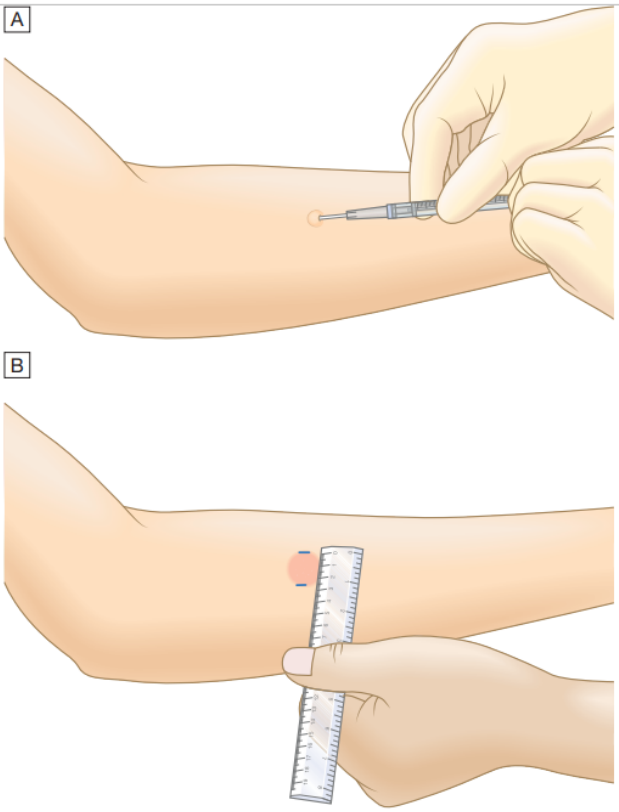


Fig. 17.41 The tuberculin skin test. A The reaction to the intradermal injection of tuberculin purified protein derivative (PPD) on the inner surface of the forearm is read between 48 and 72 hours. B The diameter of the indurated area should be measured across the forearm and is positive when ≥5 mm.

i 17.56 Factors predisposing to pulmonary fungal disease

Systemic factors

- Haematological malignancy
- HIV
- Diabetes mellitus
- Chronic alcoholism
- Radiotherapy
- Glucocorticoids, cytotoxic chemotherapy, biologic therapies and other immunosuppressant medication

Local factors

- Tissue damage by suppuration or necrosis
- Alteration of normal bacterial flora by antibiotic therapy

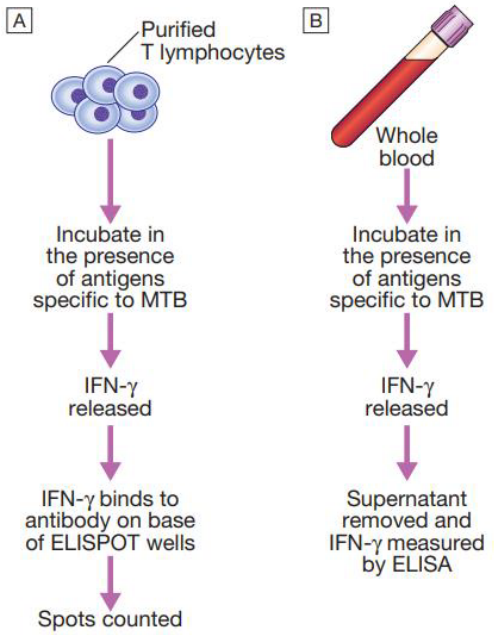


Fig. 17.42 The principles of interferon-gamma release assays (IGRAs). A sample of either (A) purified T cells (T-SPOT.TB test) or (B) whole blood (QuantiFERON–TB Gold test) is incubated in the presence of antigens specific to *Mycobacterium tuberculosis* (MTB). The release of interferon-gamma (IFN-γ) by the cells is measured by enzyme-linked immunosorbent assay (ELISA). (ELISPOT = enzyme-linked immunosorbent spot assay)

i 17.54 Factors contributing to the emergence of drug-resistant tuberculosis

- Drug shortages
- Poor-quality drugs
- Lack of appropriate supervision
- Transmission of drug-resistant strains
- Prior antituberculosis treatment
- Treatment failure (smear-positive at 5 months)

i 17.55 Site-specific opportunistic mycobacterial disease

Pulmonary

- *M. xenopi*
- *M. kansasii*
- *M. malmoense*
- MAC
- *M. abscessus* (in cystic fibrosis)

Lymph node

- MAC
- *M. malmoense*
- *M. fortuitum*
- *M. chelonei*

Soft tissue/skin

- *M. leprae*
- *M. ulcerans* (prevalent in Africa, northern Australia and South-east Asia)
- *M. marinum*
- *M. fortuitum*
- *M. chelonae*

Disseminated

- MAC (HIV-associated)
- *M. haemophilum*
- *M. genavense*
- *M. fortuitum*
- *M. chelonae*
- BCG

(BCG = bacille Calmette–Guérin; MAC = *Mycobacterium avium* complex – *M. scrofulaceum*, *M. intracellulare* and *M. avium*)

17.57 Classification of bronchopulmonary aspergillosis

- Allergic bronchopulmonary aspergillosis (asthmatic pulmonary eosinophilia)
- Extrinsic allergic alveolitis (*Aspergillus clavatus*)
- Intracavitary aspergilloma
- Invasive pulmonary aspergillosis
- Chronic and subacute pulmonary aspergillosis

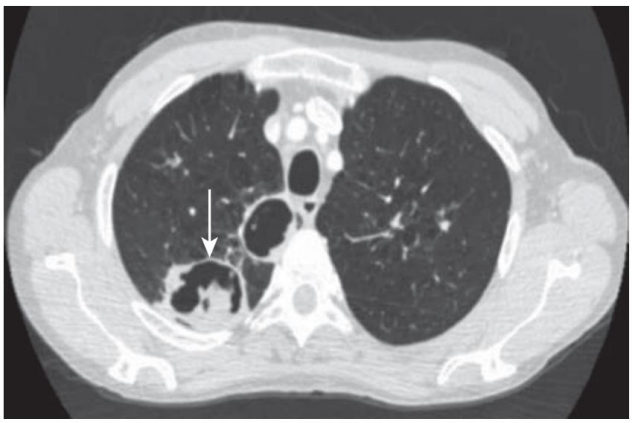


Fig. 17.44 Computed tomogram of aspergilloma in the left upper lobe. The rounded fungal ball is separated from the wall of the cavity by an 'air crescent' (arrow).

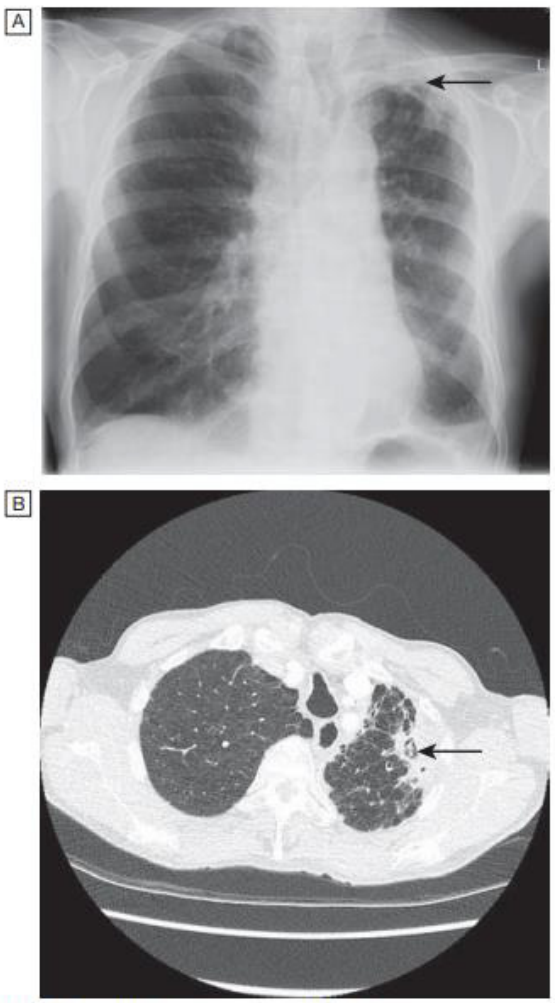


Fig. 17.45 Chronic pulmonary aspergillosis. A The chest X-ray shows pleural thickening with loss of lung volume at the left apex (arrow). B High-resolution computed tomography reveals multiple small cavities and pleural thickening with an aspergilloma and surrounding air crescent (arrow) in one of the cavities. *Courtesy of Professor David Denning, National Aspergillosis Centre, Manchester, UK.*

17.59 Risk factors for invasive aspergillosis

- Neutropenia: risk related to duration and degree
- Solid organ or allogeneic stem cell transplantation
- Prolonged high-dose glucocorticoid therapy
- Leukaemia and other haematological malignancies
- Cytotoxic chemotherapy
- Advanced HIV disease
- Severe chronic obstructive pulmonary disease
- Critically ill patients on intensive care units
- Chronic granulomatous disease

17.60 Criteria for the diagnosis of probable invasive pulmonary aspergillosis

Host factors

- Recent history of neutropenia ($<0.5\times10^9$/L for ≥10 days) temporally related to the onset of fungal disease
- Recipient of allogeneic stem cell transplant
- Prolonged use of glucocorticoids (average minimum 0.3 mg/kg/day prednisolone or equivalent) for >3 weeks (excludes allergic bronchopulmonary aspergillosis)
- Treatment with other recognised T-cell immune suppressants, such as ciclosporin, tumour necrosis factor, alpha-blockers, specific monoclonal antibodies (e.g. alemtuzumab) or nucleoside analogues during the last 90 days
- Inherited severe immune deficiency, e.g. chronic granulomatous disease or severe combined immune deficiency (p. 79)

Clinical criteria[1]

- The presence of one of the following on CT:
  - Dense, well-circumscribed lesion(s) with or without a halo sign
  - Air crescent sign
  - Cavity

Tracheobronchitis

- Tracheobronchial ulceration, nodule, pseudomembrane, plaque or eschar seen on bronchoscopy

Mycological criteria

- Mould in sputum, BAL fluid or bronchial brush, indicated by one of the following:
  - Recovery of fungal elements indicating a mould of *Aspergillus*
  - Recovery by culture of a mould of *Aspergillus*
- Indirect tests (detection of antigen or cell wall constituents)
  - Galactomannan antigen in plasma, serum or BAL fluid
  - β-1,3-glucan detected in serum (detects other species of fungi, as well as *Aspergillus*)[2]

[1]Must be consistent with the mycological findings and temporally related to current episode. [2]May be useful as a preliminary screening tool for invasive aspergillosis.

(BAL = bronchoalveolar lavage)

Adapted from De Pauw B, Walsh TJ, Donnelly JP, et al. Revised definitions of invasive fungal disease from the European Organisation for Research and Treatment of Cancer/Mycoses Study Group. Clin Infect Dis 2008; 46:1813–1821.

17.61 The burden of lung cancer

- 1.8 million new cases worldwide each year
- Most common cancer in men
- Rates rising in women:
  - Female lung cancer deaths outnumber male in some Nordic countries
  - Has overtaken breast cancer in several countries
- More than a threefold increase in deaths since 1950
- More than 50% of cases have metastatic disease at diagnosis

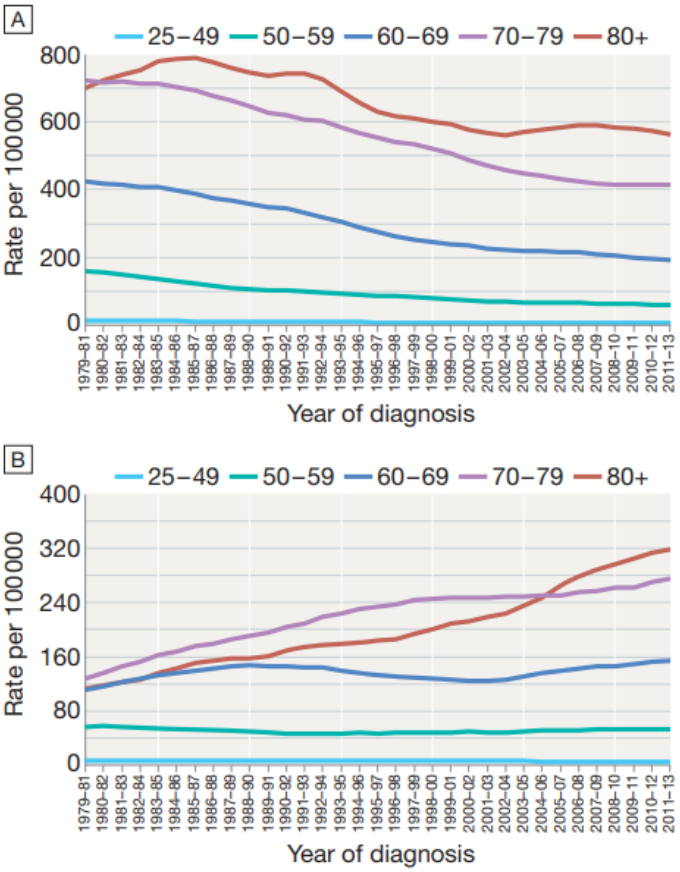


Fig. 17.46 Mortality trends from lung cancer in UK, 1979–2013, by age and year of death. A Males. B Females. Note the decline in mortality from lung cancer in men and increase in mortality in older women towards the end of this period, reflecting changes in smoking habits. *From Cancer Research UK: http://www.cancerresearchuk.org/health-professional/cancer-statistics/statistics-by-cancer-type/lung-cancer/mortality. Accessed January 2017.*

i 17.62 Common cell types in lung cancer

Cell type	%
Adenocarcinoma	35–40
Squamous	25–30
Small-cell	15
Large-cell	10–15

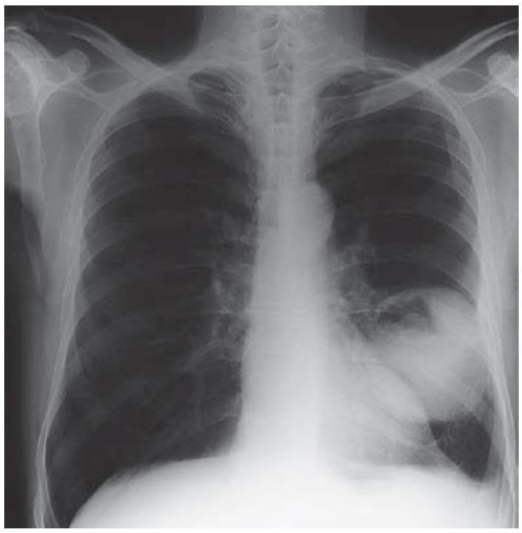


Fig. 17.47 Large cavitated lung cancer in left lower lobe.

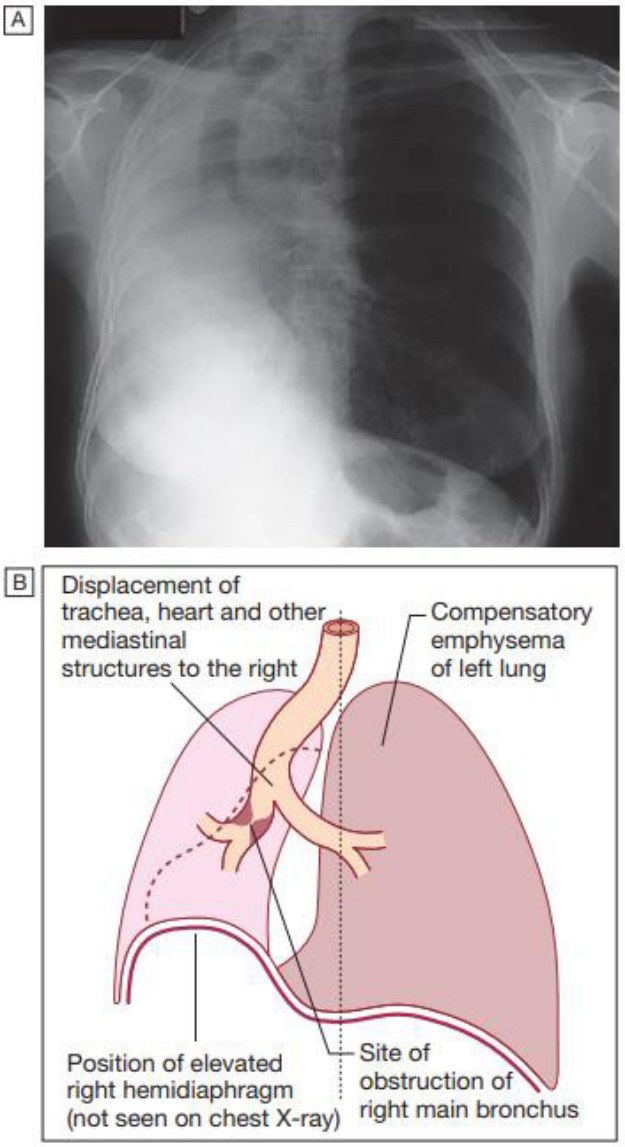


Fig. 17.48 Collapse of the right lung: effects on neighbouring structures. A Chest X-ray. B The typical abnormalities are highlighted.

i 17.63 Causes of large bronchus obstruction

Common

- Lung cancer or adenoma
- Enlarged tracheobronchial lymph nodes (malignant or tuberculous)
- Inhaled foreign bodies (especially right lung)
- Bronchial casts or plugs consisting of inspissated mucus or blood clot (especially asthma, cystic fibrosis, haemoptysis, debility)
- Collections of mucus or mucopus retained in the bronchi as a result of ineffective expectoration (especially postoperative following abdominal surgery)

Rare

- Aortic aneurysm
- Giant left atrium
- Pericardial effusion
- Congenital bronchial atresia
- Fibrous bronchial stricture (e.g. following tuberculosis or bronchial surgery/lung transplant)

17.64 Non-metastatic extrapulmonary manifestations of lung cancer

Endocrine (Ch. 18)

- Inappropriate antidiuretic hormone (ADH, vasopressin) secretion, causing hyponatraemia
- Ectopic adrenocorticotrophic hormone secretion
- Hypercalcaemia due to secretion of parathyroid hormone-related peptides
- Carcinoid syndrome (p. 678)
- Gynaecomastia

Neurological (Ch. 25)

- Polyneuropathy
- Myelopathy
- Cerebellar degeneration
- Myasthenia (Lambert–Eaton syndrome, p. 1142)

Other

- Digital clubbing
- Hypertrophic pulmonary osteoarthropathy
- Nephrotic syndrome
- Polymyositis and dermatomyositis
- Eosinophilia

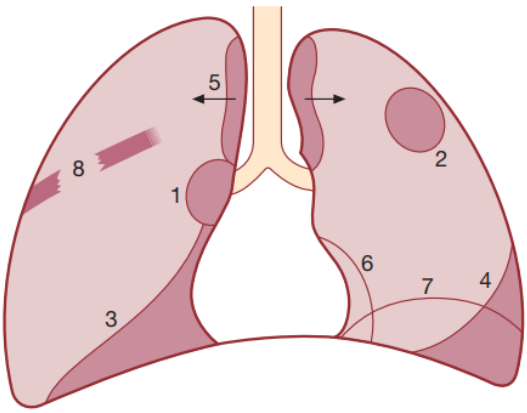


Fig. 17.49 Common radiological presentations of lung cancer. (1) Unilateral hilar enlargement suggests a central tumour or hilar glandular involvement. However, a peripheral tumour in the apex of a lower lobe can look like an enlarged hilar shadow on the posteroanterior X-ray. (2) Peripheral pulmonary opacity (p. 560) is usually irregular but well circumscribed, and may contain irregular cavitation. It can be very large. (3) Lung, lobe or segmental collapse is usually caused by tumour occluding a proximal bronchus. Collapse may also be due to compression of a bronchus by enlarged lymph glands. (4) Pleural effusion usually indicates tumour invasion of the pleural space or, very rarely, infection in collapsed lung tissue distal to a lung cancer. (5) Paratracheal lymphadenopathy may cause widening of the upper mediastinum. (6) A malignant pericardial effusion may cause enlargement of the cardiac shadow. (7) A raised hemidiaphragm may be caused by phrenic nerve palsy. Screening will show paradoxical upward movement when the patient sniffs. (8) Osteolytic rib destruction indicates direct invasion of the chest wall or metastatic spread.

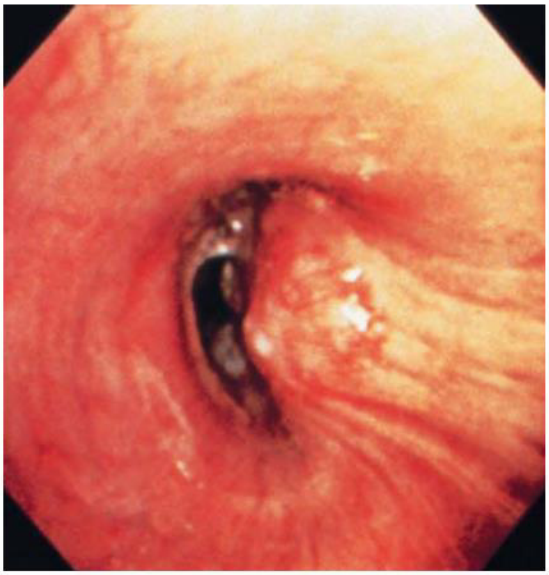


Fig. 17.50 Bronchoscopic view of a lung cancer. There is distortion of mucosal folds, partial occlusion of the airway lumen and abnormal tumour tissue.

17.66 Causes of a mediastinal mass

Superior mediastinum

- Retrosternal goitre
- Persistent left superior vena cava
- Prominent left subclavian artery
- Thymic tumour
- Dermoid cyst
- Lymphoma
- Aortic aneurysm

Anterior mediastinum

- Retrosternal goitre
- Dermoid cyst
- Thymic tumour
- Lymphoma
- Aortic aneurysm
- Germ cell tumour
- Pericardial cyst
- Hiatus hernia through the diaphragmatic foramen of Morgagni

Posterior mediastinum

- Neurogenic tumour
- Paravertebral abscess
- Oesophageal lesion
- Aortic aneurysm
- Foregut duplication

Middle mediastinum

- Lung cancer
- Lymphoma
- Sarcoidosis
- Bronchogenic cyst
- Hiatus hernia

17.65 Rare types of lung tumour

Tumour	Status	Histology	Typical presentation	Prognosis
Adenosquamous carcinoma	Malignant	Tumours with areas of unequivocal squamous and adeno-differentiation	Peripheral or central lung mass	Stage-dependent
Neuro-endocrine (carcinoid) tumour (p. 678)	Low-grade malignant	Neuro-endocrine differentiation	Bronchial obstruction, cough	95% 5-year survival with resection
Bronchial gland adenoma	Benign	Salivary gland differentiation	Tracheobronchial irritation/obstruction	Local resection curative
Bronchial gland carcinoma	Low-grade malignant	Salivary gland differentiation	Tracheobronchial irritation/obstruction	Local recurrence
Hamartoma	Benign	Mesenchymal cells, cartilage	Peripheral lung nodule	Local resection curative
Bronchoalveolar carcinoma	Malignant	Tumour cells line alveolar spaces	Alveolar shadowing, productive cough	Variable, worse if multifocal

<table>
<tr><th rowspan="2">Tumour stage</th><th colspan="4">Lymph node spread</th></tr>
<tr><th>N0
(None)</th><th>N1
(Ipsilateral hilar)</th><th>N2
(Ipsilateral mediastinal or subcarinal)</th><th>N3
(Contralateral or supraclavicular)</th></tr>
<tr><td>T1a (≤1 cm)</td><td>IA1 (92%)</td><td rowspan="5">IIB (53%)</td><td rowspan="5">IIIA (36%)</td><td rowspan="5">IIIB (26%)</td></tr>
<tr><td>T1b (>1 to ≤2 cm)</td><td>IA2 (83%)</td></tr>
<tr><td>T1c (>2 to ≤3 cm)</td><td>IA3 (77%)</td></tr>
<tr><td>T2a (>3 to ≤4 cm)</td><td>IB (68%)</td></tr>
<tr><td>T2b (>4 cm to ≤5 cm)</td><td>IIA (60%)</td></tr>
<tr><td>T3 (>5 cm)</td><td>IIB (53%)</td><td rowspan="2">IIIA (36%)</td><td rowspan="2">IIIB (26%)</td><td rowspan="2">IIIC (13%)</td></tr>
<tr><td>T4 (>7 cm or invading heart, vessels, oesophagus, carina etc.)</td></tr>
<tr><td>M1a Lung metastasis/effusion</td><td colspan="4" rowspan="2">IVA (10%)</td></tr>
<tr><td>M1b Single extrathoracic metastasis</td></tr>
<tr><td>M1c Multiple extrathoracic metastases</td><td colspan="4">IVB (0%)</td></tr>
</table>

Fig. 17.51 Tumour stage and 5-year survival in non-small-cell lung cancer. The figure shows the relationship between tumour extent (size, lymph node status and metastases) and prognosis (% survival at 5 years for each clinical stage). *Based on data from Detterbeck FC, Boffa DJ, Kim AW, Tanoue T. The eighth edition lung cancer stage classification. Chest 2017; 151:193–203.*

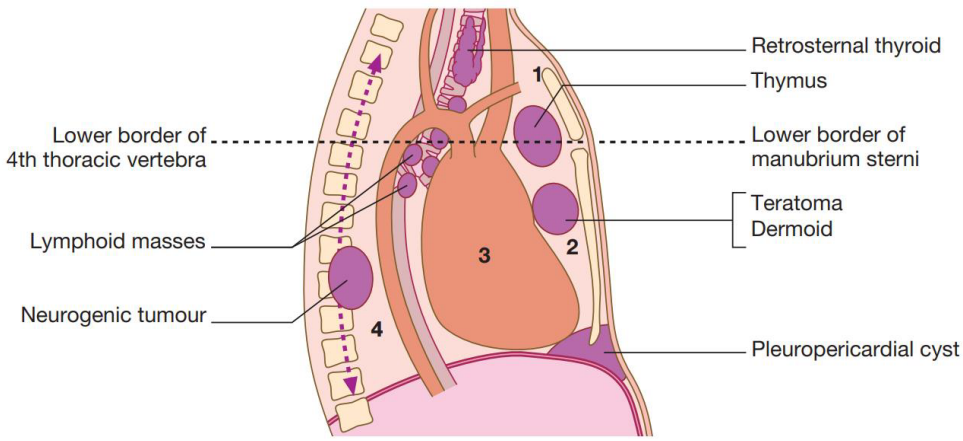


Fig. 17.52 The divisions of the mediastinum. (1) Superior mediastinum. (2) Anterior mediastinum. (3) Middle mediastinum. (4) Posterior mediastinum. Sites of the more common mediastinal tumours are also illustrated. *From Johnson N McL. Respiratory medicine. Oxford: Blackwell Science; 1986.*

i 17.67 Clinical features of malignant mediastinal invasion

Trachea and main bronchi
- Stridor, breathlessness, cough, pulmonary collapse

Oesophagus
- Dysphagia, oesophageal displacement or obstruction on barium swallow examination

Phrenic nerve
- Diaphragmatic paralysis

Left recurrent laryngeal nerve
- Paralysis of left vocal cord with hoarseness and 'bovine' cough

Sympathetic trunk
- Horner's syndrome

Superior vena cava
- SVC obstruction: non-pulsatile distension of neck veins, subconjunctival oedema, and oedema and cyanosis of head, neck, hands and arms; dilated anastomotic veins on chest wall

Pericardium
- Pericarditis and/or pericardial effusion

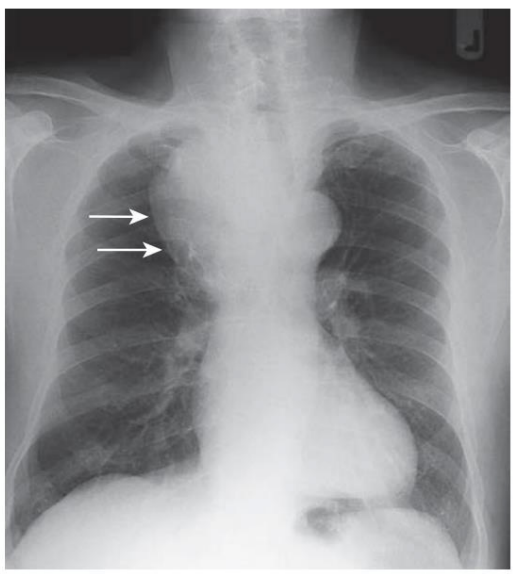


Fig. 17.53 Intrathoracic goitre (arrows) extending from right upper mediastinum.

17.68 Features common to the diffuse parenchymal lung diseases

Clinical presentation

- Cough: usually dry, persistent and distressing
- Breathlessness: usually slowly progressive; insidious onset; acute in some cases

Examination findings

- Crackles: typically bilateral and basal
- Clubbing: common in idiopathic pulmonary fibrosis but also seen in other types, e.g. asbestosis
- Central cyanosis and signs of right heart failure in advanced disease

Radiology

- Chest X-ray: typically small lung volumes with reticulonodular shadowing but may be normal in early or limited disease
- High-resolution computed tomography: combinations of ground glass changes, reticulonodular shadowing, honeycomb cysts and traction bronchiectasis, depending on stage of disease

Pulmonary function

- Typically restrictive ventilatory defect with reduced lung volumes and impaired gas transfer; exercise tests assess exercise tolerance and exercise-related fall in SaO_2

17.69 Conditions that mimic diffuse parenchymal lung disease

Infection

- Viral pneumonia
- *Pneumocystis jirovecii*
- *Mycoplasma pneumoniae*
- Tuberculosis
- Parasite, e.g. filariasis
- Fungal infection

Malignancy

- Leukaemia and lymphoma
- Lymphangitic carcinomatosis
- Multiple metastases
- Bronchoalveolar carcinoma

Pulmonary oedema

Aspiration pneumonitis

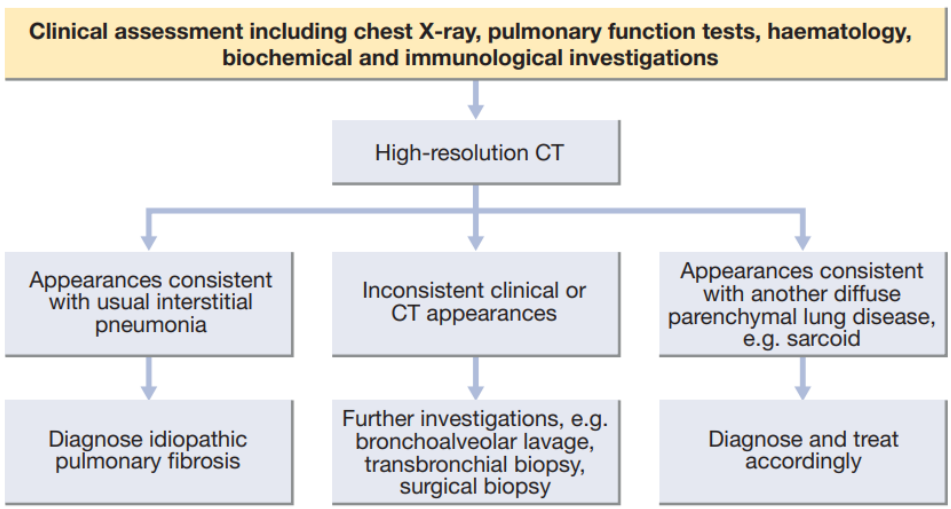


Fig. 17.54 Algorithm for the investigation of patients with interstitial lung disease following initial clinical and chest X-ray examination.

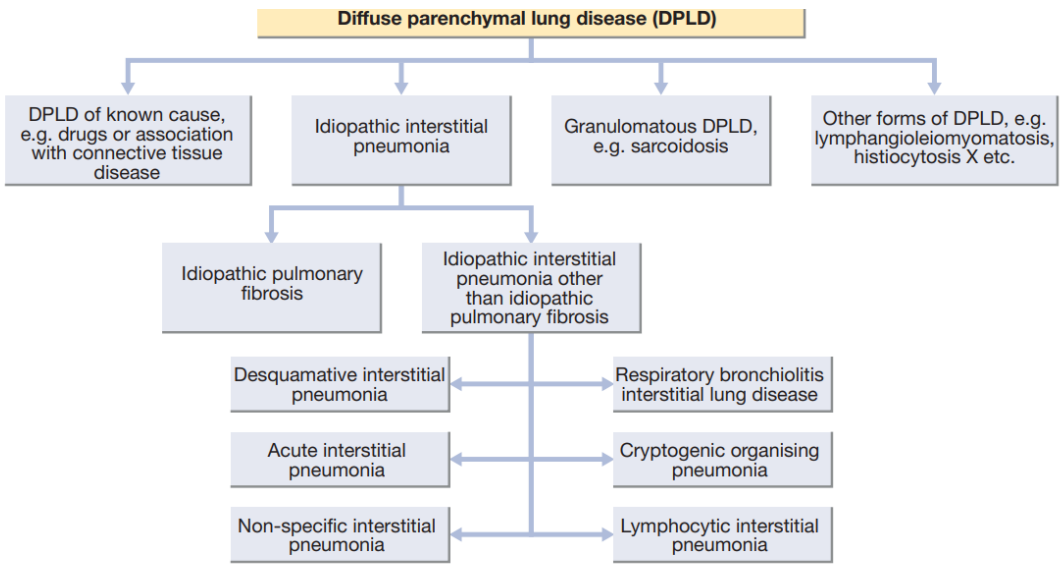


Fig. 17.55 Classification of diffuse parenchymal lung disease.

i 17.70 Idiopathic interstitial pneumonias

Clinical diagnosis	Notes
Usual interstitial pneumonia (UIP)	Idiopathic pulmonary fibrosis – see text
Non-specific interstitial pneumonia (NSIP)	See page 608
Respiratory bronchiolitis–interstitial lung disease	More common in men and smokers. Usually presents at age 40–60 years. Smoking cessation may lead to improvement. Natural history unclear
Acute interstitial pneumonia	Often preceded by viral upper respiratory tract infection. Severe exertional dyspnoea, widespread pneumonic consolidation and diffuse alveolar damage on biopsy. Prognosis often poor
Desquamative interstitial pneumonia (DIP)	More common in men and smokers. Presents at age 40–60 years. Insidious onset of dyspnoea. Clubbing in 50%. Biopsy shows increased macrophages in alveolar space, septal thickening and type II pneumocyte hyperplasia. Prognosis generally good
Cryptogenic organising pneumonia ('bronchiolitis obliterans organising pneumonia' – BOOP)	Presents as clinical and radiological pneumonia. Systemic features and markedly raised erythrocyte sedimentation rate common. Finger clubbing absent. Biopsy shows florid proliferation of immature collagen (Masson bodies) and fibrous tissue. Response to glucocorticoids classically excellent
Lymphocytic interstitial pneumonia (LIP)	More common in women, slow onset over years. Investigate for associations with connective tissue disease or HIV. Unclear whether glucocorticoids are helpful

i 17.71 Investigations in diffuse parenchymal lung disease

Laboratory investigations

- Full blood count: lymphopenia in sarcoid; eosinophilia in pulmonary eosinophilias and drug reactions; neutrophilia in hypersensitivity pneumonitis
- Ca^{2+}: may be elevated in sarcoid
- Lactate dehydrogenase: may be elevated in active alveolitis
- Serum angiotensin-converting enzyme: non-specific indicator of disease activity in sarcoid
- Erythrocyte sedimentation rate and C-reactive protein: non-specifically raised
- Autoimmune screen: anti-cyclic citrullinated peptide (anti-CCP) and other autoantibodies may suggest connective tissue disease

Radiology

- See Box 17.68

Pulmonary function

- See Box 17.68

Bronchoscopy

- Bronchoalveolar lavage: differential cell counts may point to sarcoid and drug-induced pneumonitis, pulmonary eosinophilias, hypersensitivity pneumonitis or cryptogenic organising pneumonia; useful to exclude infection
- Transbronchial biopsy: useful in sarcoid and differential of malignancy or infection
- Bronchial biopsy: occasionally useful in sarcoid

Video-assisted thoracoscopic lung biopsy (in selected cases)

- Allows pathological classification: presence of asbestos bodies may suggest asbestosis; silica in occupational fibrosing lung disease

Others

- Liver biopsy: may be useful in sarcoidosis
- Urinary calcium excretion: may be useful in sarcoidosis

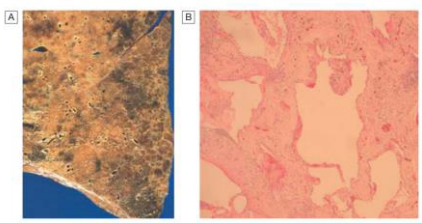


Fig. 17.57 Pathology of usual interstitial pneumonia. A Lung tissue showing subpleural scarring, most prominently down the posterior edge of the lower lobe. This distribution of fibrosis is typical of usual interstitial pneumonitis. The fibrosis may be associated with prominent cystic change known as 'honeycomb lung'. B Histology showing severe interstitial fibrosis with loss of the normal alveolar architecture and the development of 'honeycomb' cysts. *Courtesy of Dr William Wallace, Department of Pathology, Royal Infirmary of Edinburgh.*

i 17.72 Presentation of sarcoidosis

- Asymptomatic: abnormal routine chest X-ray (~30%) or abnormal liver function tests
- Respiratory and constitutional symptoms (20–30%)
- Erythema nodosum and arthralgia (20–30%)
- Ocular symptoms (5–10%)
- Skin sarcoid (including lupus pernio) (5%)
- Superficial lymphadenopathy (5%)
- Other (1%), e.g. hypercalcaemia, diabetes insipidus, cranial nerve palsies, cardiac arrhythmias, nephrocalcinosis

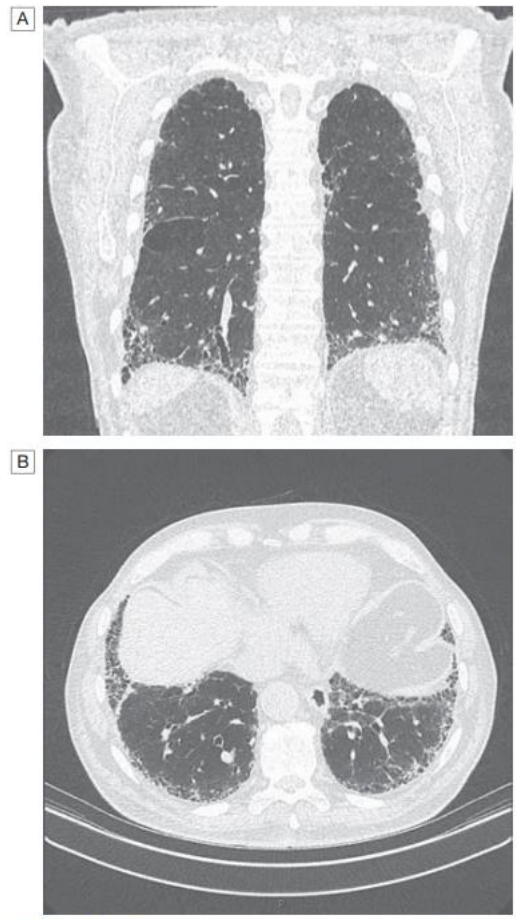


Fig. 17.56 Idiopathic pulmonary fibrosis. Typical high-resolution CT images demonstrate the bilateral, predominantly basal and peripheral reticular opacities, accompanied by honeycombing in the later stages. A Anteroposterior view. B Transverse section. *Courtesy of Dr Andrew Baird, Consultant Radiologist, NHS Lothian, Edinburgh, UK.*

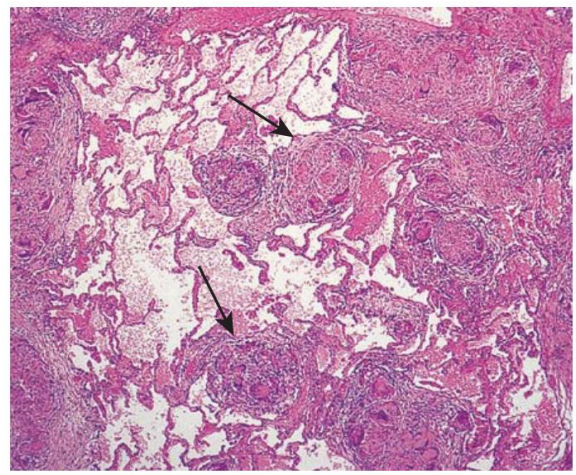


Fig. 17.58 Sarcoidosis of the lung. Histology showing non-caseating granulomas (arrows). *Courtesy of Dr William Wallace, Department of Pathology, Royal Infirmary of Edinburgh.*

i 17.74 Respiratory complications of connective tissue disorders

Disorder	Airways	Parenchyma	Pleura	Diaphragm and chest wall
Rheumatoid arthritis	Bronchitis, obliterative bronchiolitis, bronchiectasis, crico-arytenoid arthritis, stridor	Pulmonary fibrosis, nodules, upper lobe fibrosis, infections	Pleurisy, effusion, pneumothorax	Poor healing of intercostal drain sites
Systemic lupus erythematosus	–	Pulmonary fibrosis, 'vasculitic' infarcts	Pleurisy, effusion	Diaphragmatic weakness (shrinking lungs)
Systemic sclerosis	Bronchiectasis	Pulmonary fibrosis, aspiration pneumonia	–	Cutaneous thoracic restriction (hidebound chest)
Dermatomyositis/ polymyositis	Lung cancer	Pulmonary fibrosis	–	Intercostal and diaphragmatic myopathy
Granulomatosis with polyangiitis	Epistaxis, nasal discharge crusting, subglottic stenosis	Pulmonary nodules that may cavitate	Pleurisy, effusion	–

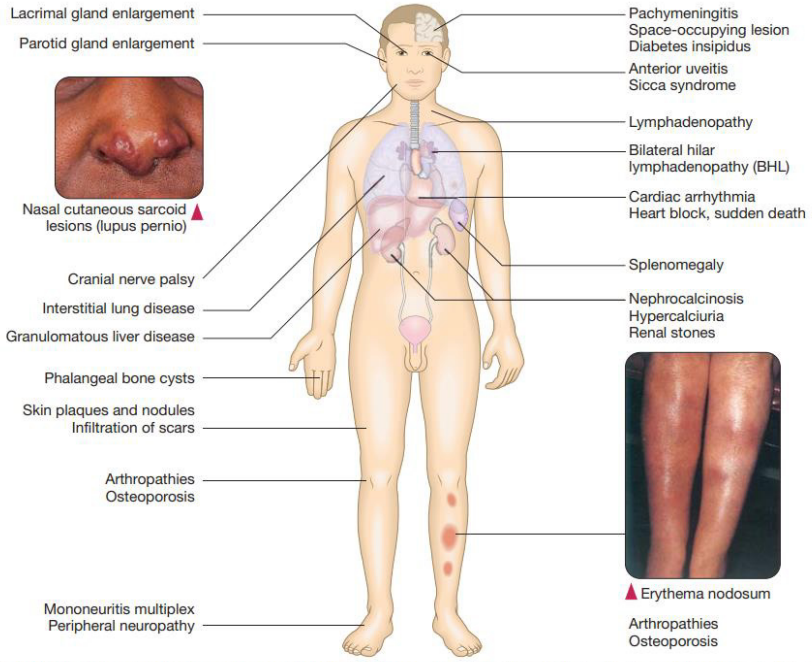


Fig. 17.59 Possible systemic involvement in sarcoidosis. *Inset (Erythema nodosum): From Savin JA, Hunter JAA, Hepburn NC. Skin signs in clinical medicine. London: Mosby–Wolfe; 1997.*

i 17.73 Chest X-ray changes in sarcoidosis

Stage I: BHL (usually symmetrical); paratracheal nodes often enlarged

- Often asymptomatic but may be associated with erythema nodosum and arthralgia. The majority of cases resolve spontaneously within 1 year

Stage II: BHL and parenchymal infiltrates

- Patients may present with breathlessness or cough. The majority of cases resolve spontaneously

Stage III: parenchymal infiltrates without BHL

- Disease less likely to resolve spontaneously

Stage IV: pulmonary fibrosis

- Can cause progression to ventilatory failure, pulmonary hypertension and cor pulmonale

(BHL = bilateral hilar lymphadenopathy)

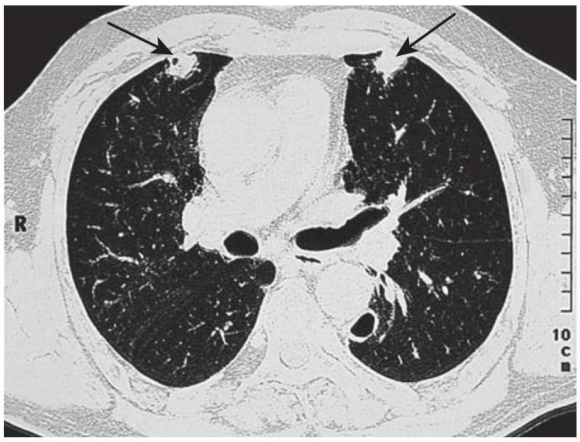


Fig. 17.60 Rheumatoid (necrobiotic) nodules. Thoracic CT just below the level of the main carina, showing the typical appearance of peripheral pleural-based nodules (arrows). The nodule in the left lower lobe shows characteristic cavitation.

i 17.75 Pulmonary eosinophilia

Extrinsic (cause known)

- Helminths: e.g. *Ascaris*, *Toxocara*, *Filaria*
- Drugs: nitrofurantoin, para-aminosalicylic acid (PAS), sulfasalazine, imipramine, chlorpropamide, phenylbutazone
- Fungi: e.g. *Aspergillus fumigatus* causing allergic bronchopulmonary aspergillosis (p. 596)

Intrinsic (cause unknown)

- Cryptogenic eosinophilic pneumonia
- Eosinophilic granulomatosis with polyangiitis (formerly Churg–Strauss syndrome), diagnosed on the basis of four or more of the following features:
  - Asthma
  - Peripheral blood eosinophilia >1.5×10^9/L (or >10% of a total white cell count)
  - Mononeuropathy or polyneuropathy
  - Pulmonary infiltrates
  - Paranasal sinus disease
  - Eosinophilic vasculitis on biopsy of an affected site
- Hypereosinophilic syndrome
- Polyarteritis nodosa (p. 1042; rare)

i 17.76 Drug-induced respiratory disease

Non-cardiogenic pulmonary oedema (ARDS)

- Hydrochlorothiazide
- Thrombolytics (streptokinase)
- Intravenous β-adrenoceptor agonists (e.g. for premature labour)
- Aspirin and opiates (in overdose)

Non-eosinophilic alveolitis

- Amiodarone, flecainide, gold, nitrofurantoin, cytotoxic agents – especially bleomycin, busulfan, mitomycin C, methotrexate, sulfasalazine

Pulmonary eosinophilia

- Antimicrobials (nitrofurantoin, penicillin, tetracyclines, sulphonamides, nalidixic acid)
- Drugs used in joint disease (gold, aspirin, penicillamine, naproxen)
- Cytotoxic drugs (bleomycin, methotrexate, procarbazine)
- Psychotropic drugs (chlorpromazine, dosulepin, imipramine)
- Anticonvulsants (carbamazepine, phenytoin)
- Others (sulfasalazine, nadolol)

Pleural disease

- Bromocriptine, amiodarone, methotrexate, methysergide
- Induction of systemic lupus erythematosus – phenytoin, hydralazine, isoniazid

Asthma

- Pharmacological mechanisms (β-blockers, cholinergic agonists, aspirin and NSAIDs)
- Idiosyncratic reactions (tamoxifen, dipyridamole)

(ARDS = acute respiratory distress syndrome; NSAIDs = non-steroidal anti-inflammatory drugs)

17.77 Interstitial lung disease in old age

- **Idiopathic pulmonary fibrosis**: the most common interstitial lung disease, with a poor prognosis.
- **Chronic aspiration pneumonitis**: must always be considered in elderly patients presenting with bilateral basal shadowing on a chest X-ray.
- **Granulomatosis with polyangiitis (Wegener's granulomatosis)**: a rare condition but more common in old age. Renal involvement is more common at presentation and upper respiratory problems are fewer.
- **Asbestosis**: symptoms may appear only in old age because of the prolonged latent period between exposure and disease.
- **Drug-induced interstitial lung disease**: more common, presumably because of the increased chance of exposure to multiple drugs.
- **Rarer interstitial disease**: sarcoidosis, idiopathic pulmonary haemosiderosis, alveolar proteinosis and eosinophilic pneumonia rarely present.
- **Increased dyspnoea**: coexistent muscle weakness, chest wall deformity (e.g. thoracic kyphosis) and deconditioning may all exacerbate dyspnoea associated with interstitial lung disease.
- **Surgical lung biopsy**: often inappropriate in the very frail. A diagnosis therefore frequently depends on clinical and high-resolution computed tomography findings alone.

17.78 Rare interstitial lung diseases

Disease	Presentation	Chest X-ray	Course
Idiopathic pulmonary haemosiderosis	Haemoptysis, breathlessness, anaemia	Bilateral infiltrates, often perihilar Diffuse pulmonary fibrosis	Rapidly progressive in children Slow progression or remission in adults Death from massive pulmonary haemorrhage or cor pulmonale and respiratory failure
Alveolar proteinosis	Breathlessness and cough Occasionally fever, chest pain and haemoptysis	Diffuse bilateral shadowing, often more pronounced in the hilar regions Air bronchogram	Spontaneous remission in one-third Whole-lung lavage or granulocyte–macrophage colony-stimulating factor (GM–CSF) therapy may be effective
Langerhans cell histiocytosis (histiocytosis X)	Breathlessness, cough, pneumothorax	Diffuse interstitial shadowing progressing to honeycombing	Course unpredictable but may progress to respiratory failure Smoking cessation may be followed by significant improvement Poor response to immunosuppressive treatment
Neurofibromatosis	Breathlessness and cough in a patient with multiple organ involvement with neurofibromas, including skin	Bilateral reticulonodular shadowing of diffuse interstitial fibrosis	Slow progression to death from respiratory failure Poor response to glucocorticoid therapy
Alveolar microlithiasis	May be asymptomatic Breathlessness and cough	Diffuse calcified micronodular shadowing more pronounced in the lower zones	Slowly progressive to cor pulmonale and respiratory failure May stabilise in some
Lymphangioleiomyomatosis	Haemoptysis, breathlessness, pneumothorax and chylous effusion in females	Diffuse bilateral shadowing CT shows characteristic thin-walled cysts with well-defined walls throughout both lungs	Progressive to death within 10 years Oestrogen ablation and progesterone therapy of doubtful value Consider lung transplantation
Pulmonary tuberous sclerosis	Very similar to lymphangioleiomyomatosis, except occasionally occurs in men		

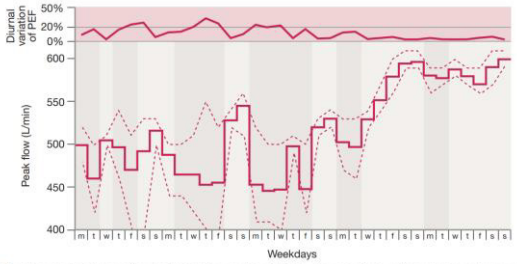


Fig. 17.61 Peak flow readings in occupational asthma. In this example, an individual with suspected occupational asthma has performed serial peak flow recording both at and away from work. The maximum, mean and minimum values are plotted daily. Days at work are indicated by the shaded areas. The diurnal variation is displayed at the top. Here, a period away from work is followed by a marked improvement in peak flow readings and a reduction in diurnal variation. (PEF = peak expiratory flow)

17.79 Occupational asthma

Most frequently reported causative agents

- Isocyanates
- Flour and grain dust
- Colophony and fluxes
- Latex
- Animals
- Aldehydes
- Wood dust

Workers most commonly reported to occupational asthma schemes

- Paint sprayers
- Bakers and pastry-makers
- Nurses
- Chemical workers

17.80 Lung diseases caused by exposure to inorganic dusts

Cause	Occupation	Description	Characteristic pathological features
Coal dust **Silica**	Coal mining Mining, quarrying, stone dressing, metal grinding, pottery, boiler scaling	Coal worker's pneumoconiosis Silicosis	Focal and interstitial fibrosis, centrilobular emphysema, progressive massive fibrosis
Asbestos	Demolition, ship breaking, manufacture of fireproof insulating materials, pipe and boiler lagging	Asbestos-related disease	Pleural plaques, diffuse pleural thickening, acute benign pleurisy, carcinoma of lung, interstitial fibrosis, mesothelioma
Iron oxide	Arc welding	Siderosis	Mineral deposition only
Tin oxide	Tin mining	Stannosis	Tin-laden macrophages
Beryllium	Aircraft, atomic energy and electronics industries	Berylliosis	Granulomas, interstitial fibrosis

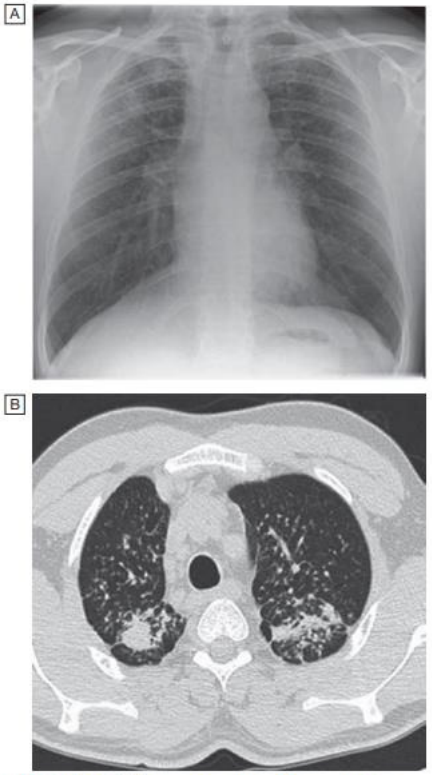


Fig. 17.62 Silicosis. A A chest X-ray from a patient with silicosis, showing the presence of small rounded nodules, predominantly seen in the upper zones. B High-resolution computed tomogram from the same patient, demonstrating conglomeration of nodules with posterior bias.

17.81 Examples of lung diseases caused by organic dusts

Disorder	Source	Antigen/agent
Farmer's lung*	Mouldy hay, straw, grain	*Saccharopolyspora rectivirgula* (formerly *Micropolyspora faeni*) *Aspergillus fumigatus*
Bird fancier's lung*	Avian excreta, proteins and feathers	Avian serum proteins
Malt worker's lung*	Mouldy maltings	*Aspergillus clavatus*
Cheese worker's lung*	Mouldy cheese	*Aspergillus clavatus* *Penicillium casei*
Maple bark stripper's lung*	Bark from stored maple	*Cryptostroma corticale*
Saxophone player's lung*	Reed of any wind instrument	*Fusarium* spp. *Penicillium* spp. *Cladosporium* spp.
Byssinosis	Textile industries	Cotton, flax, hemp dust
Inhalation ('humidifier') fever	Contamination of air conditioning	Thermophilic actinomycetes

*Presents as hypersensitivity pneumonitis.

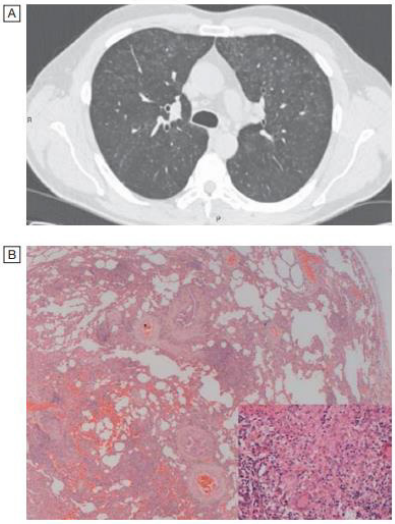


Fig. 17.63 **Hypersensitivity pneumonitis.** A High-resolution computed tomogram showing typical patchy ground-glass opacification. B Histology shows evidence of an interstitial inflammatory infiltrate in the lung, and expanding alveolar walls, with a peribronchial distribution. Within the infiltrate, there are foci of small, poorly defined non-caseating granulomas (inset), which often lie adjacent to the airways. In this case, there is little in the way of established lung fibrosis but this can be marked. *A, Courtesy of Dr S. Jackson, Western General Hospital, Edinburgh. B, Courtesy of Dr William Wallace, Dept of Pathology, Royal Infirmary of Edinburgh.*

i 17.82 Predictive factors in the identification of hypersensitivity pneumonitis

- Exposure to a known offending antigen
- Positive precipitating antibodies to offending antigen
- Recurrent episodes of symptoms
- Inspiratory crackles on examination
- Symptoms occurring 4–8 hours after exposure
- Weight loss

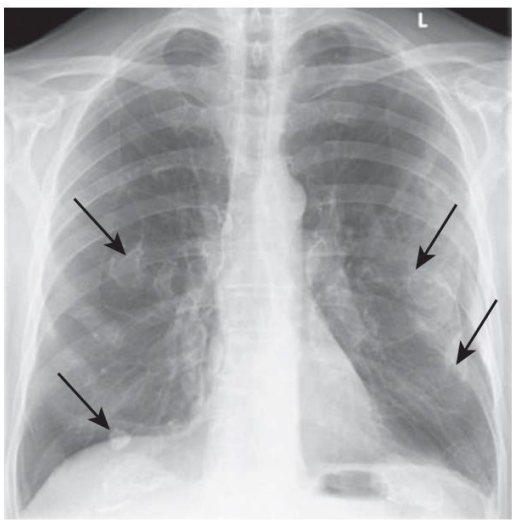


Fig. 17.64 Asbestos-related benign pleural plaques. Chest X-ray showing extensive calcified pleural plaques ('candle wax' appearance – arrows), particularly marked on the diaphragm and lateral pleural surfaces.

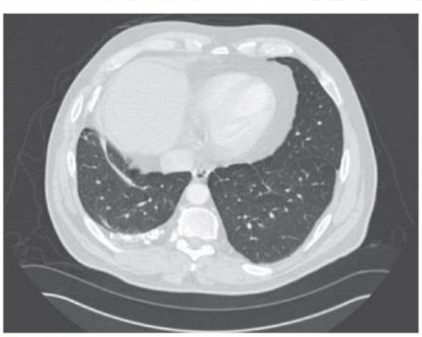


Fig. 17.65 Thoracic CT scan showing right-sided pleural thickening and an associated parenchymal band.

i 17.83 Features of pulmonary thromboemboli

	Acute massive PE	Acute small/medium PE	Chronic PE
Pathophysiology	Major haemodynamic effects: ↓cardiac output; acute right heart failure	Occlusion of segmental pulmonary artery → infarction ± effusion	Chronic occlusion of pulmonary microvasculature, right heart failure
Symptoms	Faintness or collapse, crushing central chest pain, apprehension, severe dyspnoea	Pleuritic chest pain, restricted breathing, haemoptysis	Exertional dyspnoea Late: symptoms of pulmonary hypertension or right heart failure
Signs	Major circulatory collapse: tachycardia, hypotension, ↑JVP, RV gallop rhythm, loud P_2, severe cyanosis, ↓urinary output	Tachycardia, pleural rub, raised hemidiaphragm, crackles, effusion (often blood-stained), low-grade fever	Early: may be minimal Later: RV heave, loud P_2 Terminal: signs of right heart failure
Chest X-ray	Usually normal; may be subtle oligaemia	Pleuropulmonary opacities, pleural effusion, linear shadows, raised hemidiaphragm	Enlarged pulmonary artery trunk, enlarged heart, prominent right ventricle
Electrocardiogram	$S_1Q_3T_3$ anterior T-wave inversion, RBBB	Sinus tachycardia	RV hypertrophy and strain
Arterial blood gases	Markedly abnormal with ↓PaO_2 and ↓$PaCO_2$, metabolic acidosis	May be normal or ↓PaO_2 or ↓$PaCO_2$	Exertional ↓PaO_2 or desaturation on formal exercise testing
Alternative diagnoses	Myocardial infarction, pericardial tamponade, aortic dissection	Pneumonia, pneumothorax, musculoskeletal chest pain	Other causes of pulmonary hypertension

(JVP = jugular venous pressure; PE = pulmonary embolism; RBBB = right bundle branch block; RV = right ventricular)

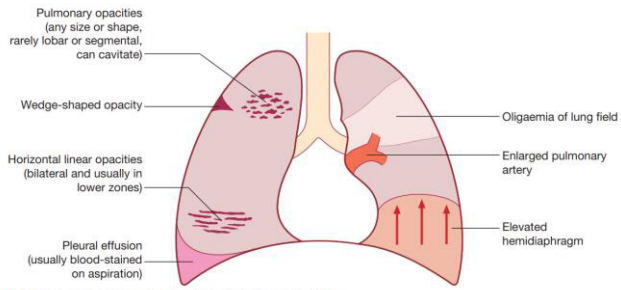


Fig. 17.66 Features of pulmonary thromboembolism/infarction on chest X-ray.

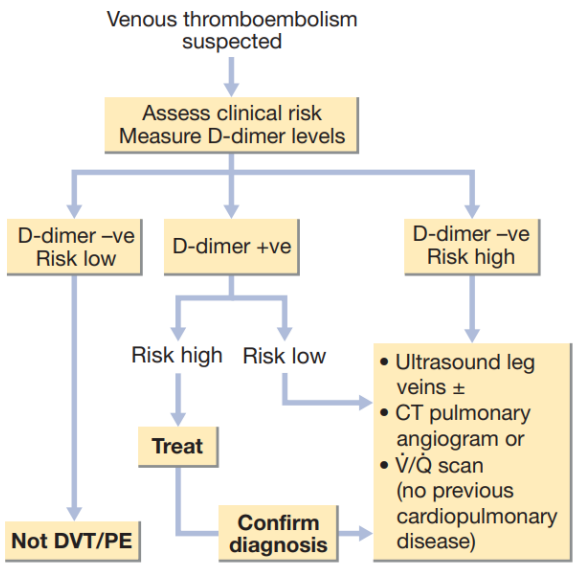


Fig. 17.67 Algorithm for the investigation of patients with suspected pulmonary thromboembolism. Clinical risk is based on the presence of risk factors for venous thromboembolism and the probability of another diagnosis. (DVT = deep vein thrombosis; PE = pulmonary embolism)

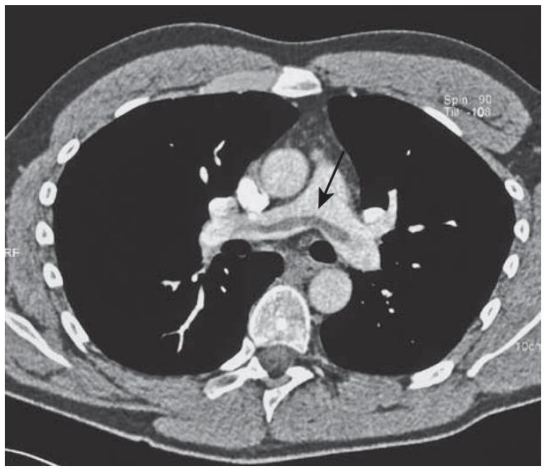


Fig. 17.68 CT pulmonary angiogram. The arrow points to a saddle embolism in the bifurcation of the pulmonary artery.

17.84 Pulmonary embolism in pregnancy

- **Maternal mortality**: venous thromboembolism is the leading direct cause in the UK.
- **CT pulmonary angiography**: may be performed safely (0.01–0.06 mGy). It is important to consider the risk of radiation to breast tissue (particularly if there is a family history of breast carcinoma).
- **V̇/Q̇ scanning**: greater radiation dose to fetus (0.11–0.22 mGy) but significantly less to maternal breast tissue.
- **In utero radiation exposure**: estimated incidence of childhood malignancy is about 1 in 16 000 per mGy.
- **Warfarin**: teratogenic, so pulmonary embolism should be treated with low-molecular-weight heparin during pregnancy.

17.85 Classification of pulmonary hypertension

Pulmonary arterial hypertension

- Primary pulmonary hypertension: sporadic and familial
- Secondary to: connective tissue disease (limited cutaneous systemic sclerosis), congenital systemic to pulmonary shunts, portal hypertension, HIV infection, exposure to various drugs or toxins, and persistent pulmonary hypertension of the newborn

Pulmonary venous hypertension

- Left-sided atrial or ventricular heart disease
- Left-sided valvular heart disease
- Pulmonary veno-occlusive disease
- Pulmonary capillary haemangiomatosis

Pulmonary hypertension associated with disorders of the respiratory system and/or hypoxaemia

- Chronic obstructive pulmonary disease
- Diffuse parenchymal lung disease
- Sleep-disordered breathing
- Alveolar hypoventilation disorders
- Chronic exposure to high altitude
- Neonatal lung disease
- Alveolar capillary dysplasia
- Severe kyphoscoliosis

Pulmonary hypertension caused by chronic thromboembolic disease

- Thromboembolic obstruction of the proximal pulmonary arteries
- In situ thrombosis
- Sickle-cell disease

Miscellaneous

- Inflammatory conditions
- Extrinsic compression of central pulmonary veins

Adapted from Dana Point 2008. Simonneau G, Robbins IM, Beghetti M, et al. Updated clinical classification of pulmonary hypertension. J Am Coll Cardiol 2009; 54:S43–S54.

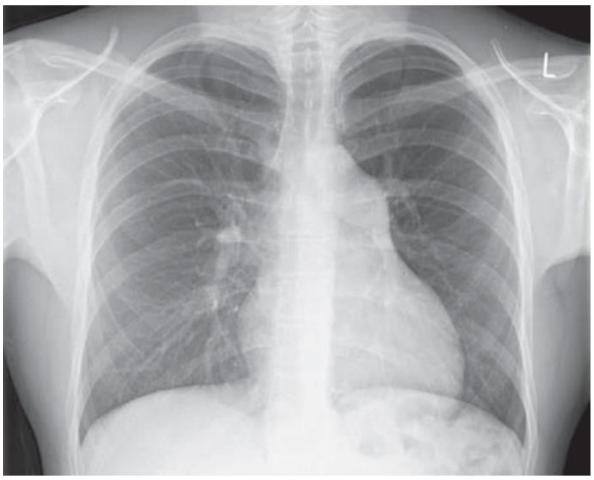


Fig. 17.69 Chest X-ray showing the typical appearance in pulmonary hypertension.

17.86 Epworth sleepiness scale

How likely are you to doze off or fall asleep in the situations described below? Choose the most appropriate number for each situation from the following scale:

0 = would never doze
1 = slight chance of dozing
2 = moderate chance of dozing
3 = high chance of dozing

- Sitting and reading
- Watching TV
- Sitting inactive in a public place (e.g. a theatre or a meeting)
- As a passenger in a car for an hour without a break
- Lying down to rest in the afternoon when circumstances permit
- Sitting and talking to someone
- Sitting quietly after a lunch without alcohol
- In a car, while stopped for a few minutes in the traffic

Normal subjects average 5.9 (SD 2.2) and patients with severe obstructive sleep apnoea average 16.0 (SD 4.4).

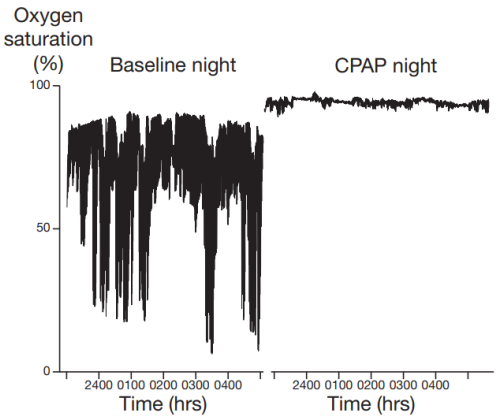


Fig. 17.70 **Sleep apnoea/hypopnoea syndrome: overnight oxygen saturation trace.** The left-hand panel shows the trace of a patient who had 53 apnoeas plus hypopnoeas/hour, 55 brief awakenings/hour and marked oxygen desaturation. The right-hand panel shows the effect of continuous positive airway pressure (CPAP) of 10 cm H_2O delivered through a tight-fitting nasal mask: it abolished his breathing irregularity and awakenings, and improved oxygenation. *Courtesy of Professor N.J. Douglas.*

17.87 Differential diagnosis of persistent sleepiness

Lack of sleep

- Inadequate time in bed
- Extraneous sleep disruption (e.g. babies/children)
- Shift work
- Excessive caffeine intake
- Physical illness (e.g. pain)

Sleep disruption

- Sleep apnoea/hypopnoea syndrome
- Periodic limb movement disorder (recurrent limb movements during non-REM sleep, frequent nocturnal awakenings; p. 1106)

Sleepiness with relatively normal sleep

- Narcolepsy
- Idiopathic hypersomnolence (rare)
- Neurological lesions (e.g. hypothalamic or upper brainstem infarcts or tumours)
- Drugs

Psychological/psychiatric

- Depression

17.88 Causes of chronic laryngitis

- Repeated attacks of acute laryngitis
- Excessive use of the voice, especially in dusty atmospheres
- Heavy tobacco smoking
- Mouth-breathing from nasal obstruction
- Chronic infection of nasal sinuses

17.89 Causes of laryngeal obstruction

- Inflammatory or allergic oedema, or exudate
- Spasm of laryngeal muscles
- Inhaled foreign body
- Inhaled blood clot or vomitus in an unconscious patient
- Tumours of the larynx
- Bilateral vocal cord paralysis
- Fixation of both cords in rheumatoid disease

17.90 Classification of pneumothorax

Spontaneous

Primary

- No evidence of overt lung disease; air escapes from the lung into the pleural space through rupture of a small pleural bleb, or the pulmonary end of a pleural adhesion

Secondary

- Underlying lung disease, most commonly chronic obstructive pulmonary disease and tuberculosis; also seen in asthma, lung abscess, pulmonary infarcts, lung cancer and all forms of fibrotic and cystic lung disease

Traumatic

- Iatrogenic (e.g. following thoracic surgery or biopsy) or chest wall injury

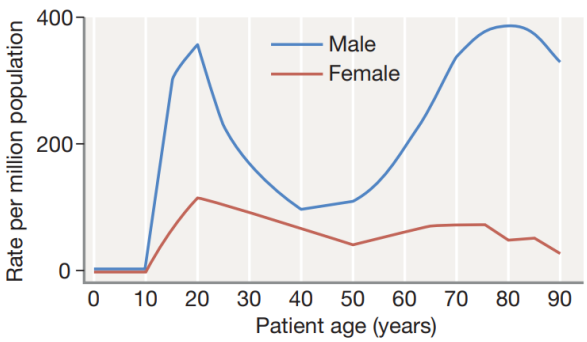


Fig. 17.71 Bimodal age distribution for hospital admissions for pneumothorax in England. The incidence of primary spontaneous pneumothorax peaks in males aged 15–30 years. Secondary spontaneous pneumothorax occurs mainly in males over 55 years.

17.91 Pleural disease in old age

- **Spontaneous pneumothorax**: invariably associated with underlying lung disease in old age and has a significant mortality. Surgical or chemical pleurodesis is advised in all such patients.
- **Rib fracture**: common cause of pleural-type pain; may be spontaneous (due to coughing), traumatic or pathological. Underlying osteomalacia may contribute to poor healing, especially in the housebound with no exposure to sunlight.
- **Tuberculosis**: should always be considered and actively excluded in any elderly patient presenting with a unilateral pleural effusion.
- **Mesothelioma**: more common in older individuals than younger people due to a long latency period between asbestos exposure (often more than 20 years) and the development of disease.
- **Analgesia**: frail older people are particularly sensitive to the respiratory depressant effects of opiate-based analgesia and careful monitoring is required when using these agents for pleural pain.

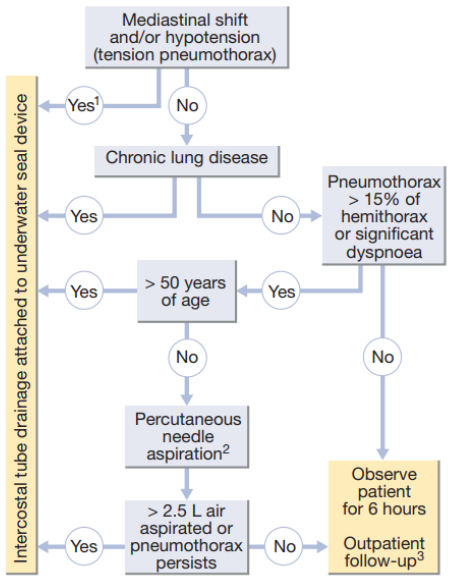


Fig. 17.73 Management of spontaneous pneumothorax. (1) Immediate decompression prior to insertion of the intercostal drain. (2) Aspirate in the second intercostal space anteriorly in the mid-clavicular line using a 16F cannula; discontinue if resistance is felt, the patient coughs excessively, or more than 2.5 L of air are removed. (3) The post-aspiration chest X-ray is not a reliable indicator of whether a pleural leak remains, and all patients should be told to attend again immediately in the event of deterioration.

17.92 Causes of elevation of a hemidiaphragm

- Phrenic nerve paralysis
- Eventration of the diaphragm
- Decrease in volume of one lung (e.g. lobectomy, unilateral pulmonary fibrosis)
- Severe pleuritic pain
- Pulmonary infarction
- Subphrenic abscess
- Large volume of gas in the stomach or colon
- Large tumours or cysts of the liver

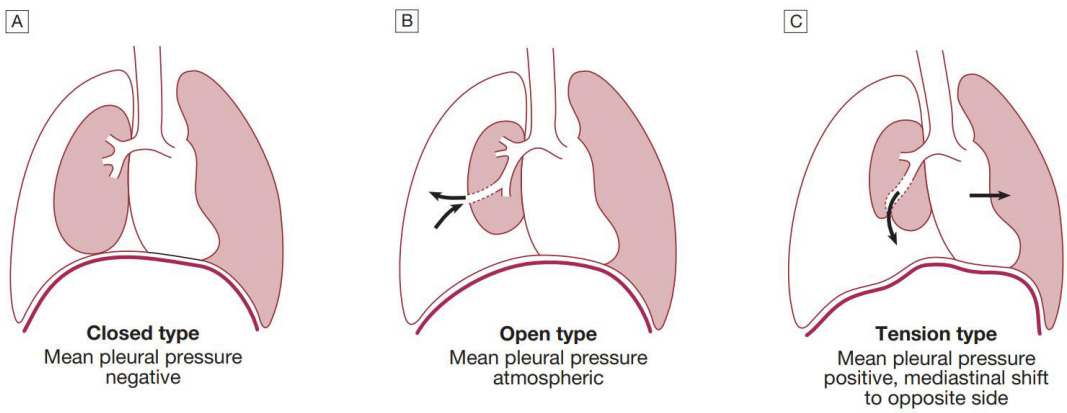


Fig. 17.72 Types of spontaneous pneumothorax. A Closed type. B Open type. C Tension (valvular) type.

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