Respiratory Emergencies

Cough

Causes of cough (a forced expulsive manoeuvre, usually against a closed glottis, and associated with a characteristic sound)

Acute cough (<3 weeks)
- Respiratory tract infections: viral upper respiratory tract infection, viral rhinosinusitis, common cold, acute bacterial sinusitis, acute bronchitis, pneumonia, acute exacerbation of COPD, pertussis
- Inhalation of direct irritants: dust, smoke, ozone, air pollutants
- Inhalation of specific allergen in the asthmatic: pollen, or low concentration of non-specific irritants: cigarette smoke, perfume; house dust mites
- Allergic rhinitis
- Chemical exposure: chloramines in swimming pools
- Medication: ACE inhibitors

Subacute cough (3–8 weeks)
- Post-infectious cough: prior viral upper respiratory tract infection
- Bordetella pertussis infection
- Subacute bacterial sinusitis
- Asthma
- Neoplasm

Chronic cough (>8 weeks)
- Persistent airway inflammation: COPD, asthma, bronchiectasis
- Smoking
- Neoplasm
- Interstitial lung disease: pulmonary fibrosis
- Persistent infection: tuberculosis; bronchiectasis
- Raised left atrial pressure: mitral stenosis, left ventricular failure
- Inhaled foreign body
- Iatrogenic: ACE inhibitors, radiation pneumonitis, steroid aerosols
- Aspiration syndrome: gastro-oesophageal reflux disease; bulbar dysfunction; oesophageal dysmotility
- Psychogenic
- Post-nasal drip syndrome (posterior nasal discharge and night cough): sinusitis; rhinitis (allergic; non-allergic; vasomotor)
- Primary ciliary dyskinesia

Potential origins of cough

Pharynx
- Post-nasal drip

Larynx
- Laryngitis
- Pertussis
- Croup
- Tumour

Trachea
- Tracheitis
Bronchi
• Bronchitis: acute and chronic
• Asthma
• Bronchial carcinoma
• Pneumonia
• Bronchiectasis
• Pulmonary oedema
• End-stage interstitial fibrosis

Checklist for cough
• Sudden (foreign body aspiration) or gradual onset
• Duration
• Diurnal variation
• Relation to initial upper respiratory tract infection
• Paroxysms of cough, with or without syncope
• Cough on eating or post-prandial: gastro-oesophageal reflux
• Triggers/aggravating factors: air temperature change; exercise; aerosols
• Production of sputum; haemoptysis
• Smoking
• Occupational history
• Medication history: ACE inhibitors
• Chronic lung disease: COPD, bronchiectasis
• Atopic disease

Possible presenting features of lung cancer
• Cough persisting more than 3 weeks
• Changes in character of smoker’s cough
• Unexplained haemoptysis
• Chest, shoulder or arm pain
• Dyspnoea
• Local fixed inspiratory wheeze
• Persistent or recurrent pneumonia or bronchitis
• Lobar collapse
• Pleural effusion
• Voice change (left vocal cord paralysis from recurrent laryngeal nerve involvement)
• Thoracic inlet(Pancoast) syndrome with shoulder and arm pain; Horner’s syndrome; brachial plexus lesions
• Phrenic nerve involvement
• Superior vena cava obstruction
• Weight loss; bone pain

Risk factors for lung cancer
• Tobacco smoking (cigarette, cigar, pipe)
• Exposure to asbestos, radon, chemicals (vinyl chloride, nickel chromates, coal products, diesel exhaust), radioactive ores
• Family history of lung cancer
• Passive smoking
• Radiation therapy to the chest (e.g. Hodgkin’s lymphoma, breast cancer post-mastectomy)
• Previous lung disease: COPD
• Outdoor air pollution

Chest x-ray patterns of lung cancer
Adenocarcinoma: peripheral solitary nodule
Squamous cell carcinoma: central lesion
Small cell carcinoma: hilar or perihilar mass
Broncholaveolar cell carcinoma: multicentric pneumonic pattern

Possible complications of cough
• Respiratory: subcutaneous emphysema; pneumothorax
• Cardiovascular: syncope; cardiac arrhythmias; subconjunctival haemorrhage
• CNS: headache; cerebral air embolism
• Musculoskeletal: intercostal muscle pain; rib fractures; rectus muscle rupture
• Urine incontinence

Dyspnoea
Dyspnoea is the conscious and unpleasant awareness of increased work done during breathing, and may indicate cardiac, pulmonary, cardiopulmonary or neuromuscular disease. Dyspnoea is not synonymous with respiratory distress. There are a number of underlying mechanisms, which may coexist in the same patient.

Mechanisms of shortness of breath
Increased demand
• Physiological: exercise; high altitude
• Pathological: anaemia; increased metabolism (fever, thyrotoxicosis)
Impaired performance
- Airflow obstruction: upper airway obstruction; obstructive lung disease: asthma, COPD, bronchiectasis; lower airway obstruction: foreign body
- Reduced lung volume (restrictive lung disease): pleural disease/effusion; pneumothorax; kyphoscoliosis; massive obesity; spine or chest wall deformities; interstitial lung disease
- Impaired gas exchange: alveolar lung disease: consolidation (pneumonia); pulmonary oedema
- Loss of lung compliance: interstitial lung disease
- Neuro-muscular diseases: Guillain-Barre syndrome; myasthenia gravis; diaphragmatic paralysis; poliomyelitis; spinal cord injury (cervical cord transection); muscular dystrophies
- Loss of thoracic cage (chest wall) compliance

Hyperventilation resulting from medullary respiratory centre stimulation in response to chemical or neural stimuli

Increased arterial hydrogen ion concentration, e.g. metabolic acidosis producing air hunger (Kussmaul’s breathing)
Increased arterial PaCO₂, e.g. respiratory acidosis
Decreased arterial PaO₂ via aortic, carotid and brain stem chemoreceptors, e.g. pneumonia, impaired oxygen delivery due to anaemia, shock and stroke
Increased central arousal, e.g. exertion, anxiety, thyrotoxicosis, phaeochromocytoma
Pulmonary J receptor discharge, e.g. pulmonary oedema

Auscultation findings in the presence of acute dyspnoea
- Fine end-inspiratory crackles over both lung bases: pulmonary oedema
- Medium end-inspiratory crackles: pulmonary fibrosis
- Localised crackles: pneumonia
- Expiratory wheeze: airways obstruction
- Absent breath sounds: pneumothorax

Symptoms associated with acute dyspnoea
- Chest pain: pleuritic (pulmonary embolism, pneumothorax, pneumonia); cardiac (acute coronary syndrome; mechanical complications: interventricular septal rupture, mitral regurgitation from chordae rupture; cardiac arrhythmia)
- Cough: pneumonia; asthma; COPD
- Orthopnea: congestive heart failure; bilateral diaphragmatic paralysis
- Paroxysmal nocturnal dyspnoea: congestive heart failure
- Severe sore throat: epiglottitis
- Platypnoea (dyspnoea worse on upright posture and relieved on recumbency): hepato-pulmonary syndrome with right-to-left shunt, patent foramen ovale

Clinical evaluation of dyspnoea
- Airway: airway patency
- Breathing
  Respiratory rate
  Oxygen saturation (pulse oximetry)
  Work of breathing
  1. Abnormal airway sounds: altered speech, stridor, expiratory wheeze, grunting
  2. Abnormal positioning: head bobbing; tripod posture (standing or sitting leaning forwards, supporting the upper body with the hands on the knees or another surface)
  3. Use of accessory muscles of respiration
    Retractions: supraclavicular, intercostal and substernal chest wall recession
    Flaring: nasal flaring
    Tracheal tug (abnormal downward movement of trachea during systole)
- Circulation
  Heart rate and rhythm
  Arterial blood pressure
Jugular venous pressure (elevated in congestive heart failure, pericardial tamponade, cor pulmonale)
Pulsus paradoxus (inspiratory fall in systolic blood pressure >10 mm Hg) (cardiac tamponade; severe asthma)

- Exposure
  - Barrel chest: COPD
  - Kyphoscoliosis: restrictive lung disease
  - Central obesity: obstructive sleep apnoea; deconditioning
  - Digital clubbing: bronchiectasis; interstitial lung disease; neoplasm; cystic fibrosis
  - Oedema/ascites: congestive heart failure

Objective measures of the severity of dyspnoea

- Ability to talk with ease
- Respiratory rate, especially if >30 breaths per minute
- Inability to adopt the supine position (orthopnoea)
- Increased effort of breathing
- Degree of hypoxaemia (oxygen saturation <92%)
- Central cyanosis, which equates to a concentration of deoxygenated haemoglobin greater than 50 g/L
- Peak expiratory flow rate <50% of predicted

Further investigations to be considered in evaluation of acute dyspnoea

- Peak expiratory flow
- Spirometry
- Chest x-ray (including comparison with previous films)
- 12 lead ECG
- Arterial blood gases
- Full blood count
- Inflammatory markers: C-reactive protein
- Renal and liver function tests
- Venous blood lactate
- Cardiac biomarkers
- B-type Natriuretic Peptide
- Bedside transthoracic echocardiography and pulmonary ultrasound

Chest x-ray patterns in the breathless patient

Focal density: consolidation; collapse; effusion; mass

Diffuse infiltrates

- Cardiomegaly: left ventricular failure
- Normal heart size: ARDS; acute coronary syndrome, diastolic failure; interstitial lung disease

Hyperlucency

- Focal: pneumothorax; bulla
- Generalised: airway disease

Normal CXR

- Pulmonary embolism
- Airways disease
- Acidosis; anaemia; hyperventilation
- Neuromuscular disorders

Causes of progressive shortness of breath

Congestive heart failure
Valvular heart disease
Coronary artery disease
Chronic obstructive pulmonary disease
Interstitial lung disease: idiopathic pulmonary fibrosis (age >45 years; persistent dry cough; progressive effort intolerance; dry inspiratory bi-basal “Velcro” crackles, digital clubbing)
Restrictive lung disease: pneumoconiosis, radiation fibrosis
Connective tissue diseases
Chronic thrombo-embolic pulmonary hypertension (caused by obstruction of the pulmonary vascular bed by non-resolving thromboemboli following acute or recurrent pulmonary embolism and leading to severe pulmonary hypertension and right heart failure)

Pulmonary embolism

Risk factors (related to Virchow’s triad of hypercoagulability of blood, venous stasis and vascular endothelial injury) may be temporary or reversible (provoked thromboembolism) or persistent.

Major

- Surgery: recent major abdominal or pelvic surgery; hip or knee replacement
- Obstetric: late pregnancy; Caesarean section;
puerperium
- Pelvic and lower limb fractures
- Varicose veins
- Malignancy: abdominal or pelvic malignancy; advanced or metastatic malignancy
- Reduced mobility: prolonged bed rest; hospitalization; institutional care
- Previous venous thromboembolism

Minor
- Age >60 years
- Oestrogens: oral contraceptive; oestrogen replacement therapy (HRT)
- Hypercoagulable states
- Chronic indwelling central venous catheters
- Multiple trauma
- Spinal cord injury
- Myeloproliferative disorders (hyperviscosity)
- Chronic heart failure
- Cerebrovascular accident with hemiplegia
- Acute medical illness
- Family history of venous thromboembolism
- Acquired thrombophilic syndromes: anti-phospholipid antibody syndrome; paroxysmal nocturnal haemoglobinuria; nephrotic syndrome
- Inherited thrombophilic syndromes: antithrombin III deficiency; protein C and protein S deficiency; factor V Leiden mutation (activated protein C resistance); factor II (prothrombin) gene mutation G20210A causing elevated prothrombin levels; hyperhomocystinaemia
- Mixed thrombophilic syndromes: hyperhomocystinaemia; elevated factor VIII levels
- Long distance sedentary travel (>4 h in the preceding month)
- Occult malignancy (Trousseau’s syndrome of chronic disseminated intravascular coagulation associated with migratory thrombothrombosis, multiple thrombotic events (venous and arterial), bleeding, therapeutic warfarin resistance, and non-bacterial thrombotic endocarditis, is associated with occult cancer). Unprovoked venous thromboembolism may indicate the need for testing for undiagnosed cancer by physical examination, blood tests and chest x-ray.

Syndromes suggestive of pulmonary embolism
It is important to consider the diagnosis in the following circumstances, given the non-specificity of symptoms and signs, aided by the presence of thrombo-embolic risk factors:
- Acute dyspnoea, especially when unexplained
- Cardiovascular collapse, with arterial hypotension (SBP <90 mm Hg for >15 min) and cardiogenic shock related to acute right ventricular failure (massive pulmonary embolism)
- Pleuritic chest pain
- Pulmonary infarction: pleuritic chest pain, dyspnoea and haemoptysis
- Near syncope or syncope
- Chronic progressive dyspnoea (chronic thrombo-embolic pulmonary hypertension)

Atypical presentations of pulmonary embolism
- Pneumonia
- Acute respiratory failure
- Acute abdominal pain
- New onset of atrial fibrillation
- Acute confusional state in the elderly

ECG changes in pulmonary embolism
- Sinus tachycardia
- Atrial arrhythmias, most commonly atrial fibrillation
- New onset incomplete or complete right bundle branch block
- Right axis deviation
- T wave inversion >3 mm in V1 to V3
- ST depression >0.5 mm in V1 to V3
- Q waves in III and aVF
- Right ventricular strain (S1, Q3, T3) (large S wave in I, large Q wave in III, inverted T wave in III)
Two-level Wells Score for likelihood of pulmonary embolism

| Variable                                                                 | Points |
|--------------------------------------------------------------------------|--------|
| History                                                                  |        |
| Previous DVT/PE                                                          | 1.5    |
| Surgery under GA or fracture of lower limbs within previous 4 weeks or    | 1.5    |
| immobilization >3 days                                                   |        |
| Malignancy (receiving treatment, treated in past 6 months or palliative  | 1      |
| care)                                                                    |        |
| Haemoptysis                                                              | 1      |
| Examination                                                              |        |
| Clinical signs of DVT (minimum of leg swelling and pain with palpation   | 3      |
| of the deep veins                                                        |        |
| Heart rate > 100 bpm                                                     | 1.5    |
| Alternative diagnosis less likely than PE (Respiratory disease: pneumothorax; | 3      |
| pneumonia; acute exacerbation of COPD; cardiac disease: acute coronary    |        |
| syndrome; acute congestive heart failure, aortic dissection; musculoskeletal |        |
| chest pain; gastroesophageal reflux disease; any cause for collapse)     |        |
| Clinical probability of pulmonary embolism                               | Score  |
| Likely                                                                   | 4      |
| Unlikely                                                                 | 4 points or less |

Wells PS, Anderson DR, Rodger M, et al. Excluding pulmonary embolism at the bedside without diagnostic imaging: management of patients with suspected pulmonary embolism presenting to the emergency department by using a simple clinical model and d-dimer. Ann Intern Med. 2001;135:98–107

PERC (Pulmonary Embolism Rule-Out Criteria)

- Age <50 years
- Heart rate <100 beats per minute
- Oxygen saturation >94% on room air
- No history of DVT/PE
- No recent trauma or surgery
- No haemoptysis
- No exogenous oestrogen
- No clinical signs of DVT

If all eight criteria are met, there is a less than 2% chance of pulmonary embolism

(Kline JA, Mitchell AM, Kabrhel C, et al. Clinical criteria to prevent unnecessary diagnostic testing in emergency department patients with suspected pulmonary embolism. J Thromb Haemostat. 2004;2:1244–55)

Suspect pulmonary embolism in hypotensive patients if

- There is evidence of, or predisposing risk factors for, venous thrombosis
- There is clinical evidence of acute cor pulmonale (acute right ventricular failure) such as distended neck veins, S3 gallop, or a para-sternal lift due to right ventricular pressure overload, tachycardia, tachypnoea, and especially if
- There is ECG evidence of acute cor pulmonale manifested by a new S1-Q3-T3 pattern, new incomplete right bundle branch block, or right ventricular ischaemia

Risk stratification in pulmonary embolism

- Stable, no signs of right ventricular dysfunction
- Stable, signs of right ventricular dysfunction
- Shock
- Cardiac arrest

Causes of elevated D-dimer (plasma levels of the degradation product of cross-linked fibrin formed after fibrin lysis by plasmin)

The negative predictive value of D-dimer testing is high, while the positive predictive value is low. A negative D-dimer test measured using a high-sensitivity assay excludes pulmonary embolism when the pre-test probability is low. Causes for an elevated D-dimer include

- Venous thromboembolism
- Acute coronary syndrome
- Acute aortic dissection
- Pregnancy
- Surgery
- Infection
- Cancer
- Trauma
Liver disease

Old age (age-adjusted D-dimer cut-off values have been suggested which can be calculated as age in years × 10 mcg/L, replacing a reliance on the conventional 500 mcg/L in patients aged 50 and older).

**Causes of haemoptysis** (bright red or pink, frothy, mixed in with sputum, alkaline):

Pulmonary

Tracheobronchial
- Tracheobronchitis
- Acute/chronic bronchitis
- Neoplasm: bronchogenic carcinoma; endobronchial metastases; bronchial adenoma; bronchial adenoma
- Bronchiectasis (cystic fibrosis; ciliary dyskinesia; post-lower respiratory tract infection)
- Foreign body aspiration
- Airway trauma

Pulmonary Parenchymal
- Pneumonia (bacterial: Klebsiella, Staphylococcus, Legionella; viral; parasitic)
- Fungal infections: pulmonary mycetoma; aspergilloma (in cavitary lesions)
- Parasitic causes: Paragonimus westermanii; hydatid cyst
- Pulmonary tuberculosis (mycetoma; Rasmussen’s aneurysm)
- Lung abscess
- Lung contusion (blunt trauma)
- Pulmonary vasculitis/alveolar haemorrhage syndromes

Vascular
- Pulmonary venous hypertension: congestive heart failure; severe mitral stenosis; left ventricular systolic heart failure; pulmonary embolism
- Pulmonary embolism with infarction
- Eisenmenger’s syndrome
- Arterio-venous malformations
- Arterio-bronchial fistula
- Pulmonary artery aneurysms from collagen vascular disease
- Hereditary haemorrhagic telangiectasia (telangiectasia in mouth or nose)

Systemic Coagulopathy
- Oral anticoagulation
- Von Willebrand disease: deficiency or dysfunction of von Willebrand factor characterized by mucocutaneous bleeding
- Haemophilia
- Thrombocytopenia; platelet dysfunction
- Disseminated intravascular coagulation

Non-respiratory Tract Sources (Spurious or Pseudo-haemoptysis)
- Upper airway: nasopharyngeal source of bleeding (epistaxis); oral bleeding
- Upper gastrointestinal bleeding

Miscellaneous
- Pulmonary endometriosis (catamenial haemoptysis)

**Causes of pulmonary renal syndromes** (pulmonary haemorrhage with acute kidney injury)

Systemic Vasculitis
- Anti-glomerular basement membrane disease (Goodpasture’s syndrome)
- ANCA (anti-neutrophil cytoplasmic autoantibody)-positive small vessel vasculitides: granulomatosis with polyangiitis (formerly Wegener’s granulomatosis); eosinophilic granulomatosis with polyangiitis (formerly Churg-Strauss syndrome; asthma with eosinophilia and vasculitis); drugs: penicillamine, hydralazine; microscopic polyangiitis
- Other vasculitides: cryoglobulinaemia; associated with systemic lupus erythematosus

Infection: severe bacterial pneumonia (Legionella, Mycoplasma, Leptospirosis); infective endocarditis; post-infectious glomerulonephritis
- Pulmonary oedema + acute kidney injury: volume overload; severe left ventricular failure
- Multi-organ failure: ARDS + acute kidney injury
- Miscellaneous: paraquat poisoning; renal vein or artery thrombosis

**Haemoptysis check list**

It is important to distinguish between true haemoptysis, pseudo-haemoptysis (coughing of
blood that originates from a source other than the lower respiratory tract, namely the upper airway or oral cavity), or haematemesis, based on the history. Haemoptysis is a common symptom, and can vary from streaks of blood in the sputum to massive haemorrhage.

- Post-nasal drip or epistaxis suggest pseudo-haemoptysis
- Smoking history
- Occupational exposure: asbestos; silica
- Weight loss
- Pleuritic chest pain
- Symptoms of congestive heart failure: breathlessness on exertion, orthopnoea, paroxysmal nocturnal dyspnoea
- Productive cough and fever
- Concurrent with menstruation (catamenial haemoptysis related to pulmonary endometriosis)
- History of cancer
- Anticoagulant therapy
- Exposure to tuberculosis
- Travel history
- Coexisting renal disease

Red flags in haemoptysis

- Massive haemoptysis (expectoration of >600 ml blood from below the glottis in 24 h or 150 ml blood in a 1 h period)
- Extensive smoking history
- Back pain
- Malaise, fatigue and weight loss lasting more than 3 weeks
- Dyspnoea at rest

Possible chest x-ray findings in haemoptysis

- Cavitary lesions
- Alveolar infiltrates: diffuse; localized
- Mass lesion
- Bronchiectasis
- Cardiomegaly and increased pulmonary blood flow
- Hilar adenopathy or mass
- Normal

Causes of respiratory failure
Type 1 (hypoxaemia): \( \text{paO}_2 < 8.9 \text{ kPa (60 mm Hg)} \)

- Low FiO\(_2\): high altitude (reduced barometric pressure); inadvertent hypoxic gas administration; breathing circuit disconnection during mechanical ventilation
- V/Q mismatch (oxygen responsive): COPD exacerbation; pneumonia
- Increased intra-pulmonary shunt (perfusion without ventilation) (oxygen insensitive): No gas exchange but perfused alveoli
  Alveolar filling: oedema, pus, blood (cardio-genic pulmonary oedema, ARDS, pneumonia, lung haemorrhage)
  Atelectasis
- Diffusion impairment: fibrosis (interstitial lung disease); oedema
- Venous admixture
- Anatomical right to left shunts (bypasses pulmonary circuit): pulmonary arteriovenous malformations; intra-cardiac shunts

Type 2 (hypercapnia): \( \text{paCO}_2 > 6.7 \text{ kPa (50 mm Hg)} \)

- Central respiratory depression causing alveolar hypoventilation
  Drugs: opiates; benzodiazepines; synergistic drug interactions, altered drug metabolism (hepatic or renal failure) and iatrogenic drug overdose are factors to be considered
  Cerebrovascular disease: stroke
  Raised intracranial pressure: space occupying lesion
  Central congenital hypoventilation syndrome (Ondine’s curse): results in respiratory arrest during sleep
  Uncontrolled oxygen therapy
  Trauma: head injury
- Chest wall disease: kyphoscoliosis; flail chest; thoracoplasty; ankylosing spondylitis; obesity-hypoventilation syndrome
- Pleural disease: pneumothorax; massive pleural effusion
• Neuromuscular disease affecting respiratory muscles
  Cervical spinal cord injury: trauma; neoplasm
  Neuromuscular disease:
  Neuromuscular junction disorders: myasthenia gravis, organophosphate toxicity, botulism
  Peripheral neuropathies: Gullain Barre syndrome, diphtheritic polyneuropathy, critical illness polyneuropathy
  Amyotrophic lateral sclerosis
  Anterior horn cells in spinal cord: poliomyelitis
  Myopathies: muscular dystrophy
  • Upper airway obstruction
  • Lung disease
  Obstructive: COPD
  Restrictive

**Signs of respiratory failure**

- Increased work of breathing: tachypnoea; use of accessory muscles; nasal flaring; recession (intercostal, suprasternal, supraclavicular)
- Sympathetic activation: tachycardia; hypertension; sweating
- End-organ hypoxia: confusion; altered level of consciousness; bradycardia and hypotension (late signs)
- Haemoglobin desaturation: cyanosis

**Potential sources of error with pulse oximetry**

- Poorly adherent probe
- Dark skin
- Excessive motion: motion artifact
- Low signal-noise ratio with impaired peripheral perfusion: shock; cardiac arrest
- False nails; blue, black or green nail varnish
- Lipaemia: hyperlipidaemia; propofol infusion
- Bright ambient light
- Abnormal haemoglobins: carboxyhaemoglobin; methaemoglobin
- Intravenous dyes: methylene blues
- SpO2 < 80%
- Venous pulsations: obstructed venous return; severe right heart failure; dependent limb; tourniquet constriction

**Signs of hypercapnia**

- Drowsiness
- Bounding peripheral pulses
- Flapping tremor
- Papilloedema
- Coma

**Causes of hyperventilation**

Physiological

- Fever
- Pregnancy
- High altitude

Pathological

- Hypoxaemia: asthma, left ventricular failure, pulmonary embolism, pneumonia
- Severe pain
- Metabolic acidosis: diabetic ketoacidosis
- Drugs: salicylate toxicity; withdrawal syndromes (alcohol; benzodiazepines)

**Causes of wheezing in childhood**

Intrinsic airway narrowing

- Structural anomalies: tracheobronchomalacia/stenosis; bronchopulmonary dysplasia; α-1-antitrypsin deficiency
- Bronchospasm: anaphylaxis; organophosphate toxicity
- Inflammation: asthma; bronchiolitis; episodic viral wheeze, associated with upper respiratory tract infections (in children aged 6 months
Intraluminal airway obstruction

• Foreign body aspiration (triad of unilateral monophonic wheeze, cough and unilateral reduction in breath sounds)

Extrinsic airway compression

• Congenital structural anomalies: cystic malformations of lung; vascular ring; cardiovascular enlargement
• Mediastinal tumours
• Enlarged mediastinal lymph nodes

Miscellaneous:

• Congestive heart failure
• Muco-ciliary clearance disorders: cystic fibrosis; bronchiectasis; primary ciliary dyskinesia

Types of wheeze

Polyphonic wheeze

• Asthma
• COPD
• Heart failure
• Extrinsic allergic alveolitis
• Anaphylaxis

Monophonic wheeze

• Foreign body aspiration
• Bronchial carcinoma

Features of bronchiolitis

• Affects children aged 2 years or under
• A coryzal period lasting 1–3 days is followed by persistent cough, low grade fever, tachypnoea, chest recession, and either high pitched expiratory wheezing or fine inspiratory crackles on auscultation
• Self-limiting, with symptoms peaking at 3–5 days
• Peak prevalence is in the winter months in temperate climates
• Occurs in association with viral infections (RSV in around 75% cases); also parainfluenza, adenovirus, influenza, rhinovirus, metapneumovirus

Risk factors for severe bronchiolitis

• Age <3 months
• Premature birth, especially 32 weeks or under
• Chronic lung disease (including bronchopulmonary dysplasia, cystic fibrosis)
• Congenital heart disease, which is haemodynamically significant
• Immunodeficiency (congenital or acquired)
• Neuromuscular disease: severe neurological disease

Features suggestive of asthma

• Onset in early life
• Frequent and recurrent episodic wheeze, breathlessness, chest tightness and cough, worse at night and in early mornings (diurnal and day-to-day variability), or in response to exercise, allergen exposure (including exposure to pets), cold air and emotional stress
• History of atopic disease
• Family history of asthma and/or atopic disease
• Widespread wheeze on cardiac auscultation
• Aspirin-induced asthma is associated with the triad of asthma, aspirin intolerance, and sinusitis with nasal polyps

**Historical clues which lower the probability of a diagnosis of asthma**

• Chronic productive cough in the absence of wheeze or shortness of breath
• Smoking history >20 pack years (pack year= (cigarettes smoked per day/20) × number of years smoking)
• Voice disturbance
• Symptoms with colds only, with no interval symptoms
• Normal physical examination of the chest when symptomatic
• Normal peak expiratory flow when symptomatic
• Cardiac disease

**Asthma mimics**

“All that wheezes is not asthma”

• Other obstructive lung disease: COPD; bronchiectasis; cystic fibrosis; eosinophilic bronchitis; primary ciliary dyskinesia syndromes
• Respiratory infections: recurrent viral lower respiratory tract infections, pulmonary tuberculosis, allergic bronchopulmonary aspergillosis (fever, malaise, recurrent airway obstruction, cough with expectoration of brownish mucus plugs, haemoptysis and peripheral blood eosinophilia; chest x-ray shows upper lobe infiltrates, atelectasis due to mucoid impaction, and tramline shadowing of central bronchiectasis representing thickened bronchial walls)
• Non-obstructive lung disease: diffuse parenchymal lung disease; pulmonary embolism; chronic eosinophilic pneumonia (with reverse pulmonary oedema appearance on chest x-ray)
• Gastroesophageal reflux
• Vocal cord dysfunction: paroxysmal adduction of the vocal cords on inspiration, expiration or both
• Upper airway obstruction: large airway stenosis
• Foreign body aspiration
• Primary endobronchial tumours
• Adverse drug reactions: ACE inhibitors
• Left ventricular failure (“cardiac asthma”)
• Hyperventilation syndrome and panic attacks

**Triggers for asthma**

• IgE related: allergens (animals and pets; grain; house dust mites; pollen, including trees and grass; moulds and fungi); proteolytic enzymes
• Non IgE related: hardwood dust; colophony fumes (solder); isocyanates; exercise; sex; atmospheric pollution (traffic fumes); emotion; smoking and second-hand smoke; recreational drugs; weather and changes in temperature; viral infections

**Categorisation of severity of asthma**

Mild:
• PEFR >75% predicted or best

Moderate:
• PEFR 50–75% predicted or best

Severe:
• PEFR <50% predicted or best
• Inability to complete sentences
• Respiratory rate >25/min
• Tachycardia >110/min

Life threatening

**Markers of a life threatening attack of asthma**

• Unable to talk
• Exhaustion
• Confusion
• Cyanosis of lips and tongue on room air
• Silent chest/feeble respiratory effort
• Saturation <90%
• PFR <33% of predicted or best
• No response to beta 2 agonist therapy
• Bradycardia
• Coma
• Hypotension
Warning signs of asthma exacerbation

- Increased dyspnoea
- A combination of increased wheeze, cough, or mucus secretion
- Nocturnal asthma
- Increased use of short acting sympathomimetics (reliever medication)
- Increased exercise induced asthma
- Decreased morning peak expiratory flow rate

Features suggestive of COPD

- Onset in mid-life: age greater than 35 years
- Exertional breathlessness
- Persistent progressive breathlessness
- Chronic productive cough, with regular sputum production
- Frequent winter ‘bronchitis’
- Smoker or ex-smoker

Acute exacerbation of COPD (triad of increased dyspnoea, increased sputum volume, and purulent sputum)

Precipitating factors

- Respiratory infection
  Bacteria: Haemophilus influenzae; Streptococcus pneumoniae; Staphylococcus aureus; Moraxella catarrhalis
  Viral: Rhinovirus, influenza virus, parainfluenza virus, coronavirus, adenovirus, picornavirus, parvovirus, respiratory syncytial virus
  Atypical bacteria: mycoplasma pneumoniae; chlamydia pneumoniae; legionella
- Heart failure
- Pulmonary embolism
- Pneumothorax
- Non-pulmonary infections
- Environmental: cold temperature, air pollution, cigarette smoke
- Non-compliance with medication

Causes of spontaneous pneumothorax

Primary: apical sub-pleural bleb or bulla (associated with male gender, tall stature, low body mass index, inhalant use, genetic predisposition and smoking)

Secondary:

Primary airway disease: COPD; acute severe asthma; cystic fibrosis
Lung infections: Pneumocystis jiroveci pneumonia; tuberculosis; necrotizing bacterial pneumonia; fungal pneumonia
Interstitial lung disease: sarcoidosis; idiopathic pulmonary fibrosis; lymphangioleiomyomatosis or LAM (smooth muscle proliferation around bronchioles results in air trapping and characteristic thin-walled lung cysts that are uniform in size; chest x-ray may reveal hyperinflation, chylos pleural effusion and diffuse bilateral reticulo-nodular interstitial pattern)
Connective tissue disease: rheumatoid arthritis; ankylosing spondylitis; polymyositis; dermatomyositis; systemic sclerosis; Marfan’s syndrome; Ehlers-Danlos syndrome
Cancer
Catamenial pneumothorax: thoracic endometriosis (within 72 h of menstruation)

Features of pneumothorax on supine chest x-ray

Radiolucency in lower zone
Increased air in anterior and lateral costophrenic sulcus

- Hyper-lucency of upper abdominal quadrants and lower chest
- Deep and sometimes “tongue-like” lateral costophrenic sulcus: deep sulcus sign (deep V)
- Visualization of the anterior costophrenic sulcus: double hemi-diaphragm sign, as the dome and anterior portions of the diaphragm are outlined by lung and pleural air, respectively
- Sharp diaphragmatic or mediastinal contours: increased definition of the mediastinal border (etched mediastinum)

Depression of the ipsilateral diaphragm
Double diaphragm contour
Outline of medial diaphragm under heart silhouette
Presence of a sharply defined pericardial fat pad and a distinct cardiac apex (mediastinal
structures sharply outlined by free air)-unusually clear or sharp heart border
Sub-pulmonic air which outlines the visceral pleura of the lung base

**Features of pneumothorax on bedside ultrasound**

- Absence of dynamic pleural sliding with respiration
- Loss of vertical comet-tail artefacts along the pleural interface
- Stratosphere sign or bar-code sign, where only horizontal lines are seen (continuous ocean pattern) on M-mode. The actual location where the pneumothorax begins can often be detected. This interface between normal lung and pneumothorax is known as the lung point. This can be visualised on the M-mode image, where both the seashore sign and stratosphere sign are seen on the same image

**Causes of pseudo-pneumothorax** (pneumothorax mimics) on chest x-ray

- Skin fold (straight or minimally curved; dense line sharp on one side and blurred on the other; passes outside chest cavity; does not run parallel to chest wall)
- Medial border of scapula
- Lateral edge of breast tissue
- Calcified pleural plaque
- Post-pleurectomy scarring
- Companion shadow (radiopaque line accompanying inferior rib margin, caused by extrapleural fat or visible subcostal groove, usually on 1st or 2nd rib)
- Folds of blankets or clothing
- Giant bullous emphysema (vanishing lung syndrome) (the compressed lung falls towards the costophrenic angle)

**Causes of failure of lung re-expansion after chest drain placement**

- Blocked drain
- Misplaced drain
- Persistent air leak: bronchopleural fistula

**Pleural effusion**

Causes of transudative effusion (serous) (<30 g/L)

- Right ventricular failure
- Congestive heart failure
- Constrictive pericarditis
- Superior vena caval obstruction
- Cirrhosis of the liver
- Hypoalbuminaemia: nephrotic syndrome; liver failure
- Hypothyroidism
- Meigs syndrome
- Obstructive uropathy
- End-stage chronic kidney disease
- Peritoneal dialysis

**Causes of exudative effusion**

- Malignancy: lung; breast; lymphoma; pleural mesothelioma (fixed mediastinum due to pleural encasement; progressive reduction in size of hemithorax associated with pleural thickening); metastases
- Infection: para-pneumonic effusions; tuberculosis; fungal; parasitic
- Pulmonary infarction
- Gastrointestinal: pancreatitis; oesophageal rupture (pleural fluid amylase may be elevated)
- Autoimmune: systemic lupus erythematosus
- Radiation pleuritis
- Post-myocardial infarction
- Ovarian hyperstimulation syndrome
- Asbestos-related pleural disease
- Yellow nail syndrome
- Trauma: haemothorax; chylothorax
- Fistula (ventriculo-peritoneal; bilio-peritoneal; gastro-peritoneal)
- Drugs: nitrofurantoin; methysergide

Exudates can demonstrate on bedside ultrasound the following features:

- Multiple internal echoes from floating debris
- Septations and loculation
- Consolidation of lung
- Pleural thickening
Features of pleural effusion on supine chest x-ray
Asymmetrical, diffuse, hazy opacity that increases in density within the hemithorax in a cephalo-caudad direction, due to layering of the effusion posteriorly. The hemidiaphragm is obscured and the lateral costophrenic angle is blunted. Opacity over lung apex with a concave interface inferiorly (pleural cap): the apex is the most dependent portion of the thorax tangential to the frontal x-ray beam. Absence of air bronchograms and visualization of lung vessels through the density confirms that the increased opacity is extraparenchymal in location.

Features of pleural effusion on bedside ultrasound
A pleural effusion appears as an anechoic or hypoechoic zone between the parietal and visceral pleura. The lung sliding sign is absent. A transudate is echo-free. An exudate appears as an echoic collection, with floating echogenic debris, with or without pleural thickening and loculation. Empyema and haemothorax appear homogenous and echoic. The presence of floating fragments within a pleural effusion has been referred to as the plankton sign.

Features of loculated pleural effusion
• Elliptical or oval pleural-based opacities without air bronchograms
• Located along the course of a fissure or between the visceral and parietal pleura when the pleural layers are partly adherent
• Does not shift freely within the pleural space with changing patient position
• Rapid disappearance with diuresis has led to the term vanishing lung tumour

Features of pleural thickening
• Bases: blunting of costophrenic angle, with tenting of diaphragmatic pleura
• Apices: apical pleural cap-curvedlinear density at lung apex

Malignant pleural thickening is suggested by:
• Parietal pleural thickening >1 cm
• Nodular pleural thickening >1 cm
• Mediastinal pleural thickening
• Circumferential pleural thickening with lung encasement and volume loss of involved hemithorax
• Chest wall invasion

Risk factors for community-acquired pneumonia
• Age >65 years
• Smoking
• Alcohol abuse
• Poor dental hygiene
• Chronic lung disease: COPD; asthma; cystic fibrosis
• Contact spread: nursing homes; institutions; military barracks; student dormitories
• Chronic kidney disease
• Diabetes mellitus
• Dementia
• Congestive heart failure
• Occupational dust exposure

Clinical features of pneumonia
Although conventionally pneumonia has been classified as typical and atypical, it is now recognized that clinical features correlate poorly with microbial aetiology.

Typical bacteria pneumonia with signs of lobar consolidation
• Sudden onset
• Fever with chills
• Cough with purulent sputum
• Pleuritic chest pain

Atypical pneumonia
Gradual onset, with prodrome of headache and myalgia
Dry cough
Low grade fever
Extra-pulmonary manifestations: abdominal pain, diarrhea, confusion
More protracted course
CXR findings often more severe than clinical presentation

**CURB 65 Score**

- Confusion (abbreviated mental test score 8 or less, or new disorientation in time, place and person)
- Raised blood urea nitrogen (>7 mmol/L)
- Raised respiratory rate (30 breaths per minute or more)
- Low blood pressure (systolic blood pressure <90 mm Hg, or diastolic blood pressure <60 mm Hg)
- Age 65 years or older

Home-based care can be considered for patients scoring 0 or 1, and hospital-based care for those scoring 2 or more
(Lim W, van der Eerden MM, Laing R, et al. Defining community acquired pneumonia severity on presentation to hospital: an international derivation and validation study. Thorax. 2003;58:377–82)

**Pneumonia mimics**

- Pulmonary infarction
- Non-infective, inflammatory pneumonia:
- Eosinophilic pneumonia diffuse bilateral alveolar opacities, inter-lobular septal thickening producing Kerley B lines, and bilateral small pleural effusions
- Broncho-pulmonary aspergillosis
- Cryptogenic organising pneumonia (presents with a subacute onset of dry cough, shortness of breath, anorexia, malaise, fever and weight loss; multiple bilateral patchy alveolar opacities with a peripheral sub-pleural and bronchovascular distribution, often migratory as the disease progresses; normal lung volumes; refractory to antibiotic therapy; rapid clinical and radiological improvement with steroid therapy)
- Pulmonary vasculitis: granulomatosis with polyangiitis (Wegener’s granulomatosis) (solitary or multiple pulmonary nodules, with or without cavitation)
- Eosinophilic granuloma
- Acute allergic or toxic pneumonia

**Environmental factors in pulmonary infection**

- Water cooling units: Legionella
- Military barracks: Mycoplasma
- Birds: psittacosis, histoplasmosis, aspergillosis
- Dogs, cats, rats, pigs, cattle: leptospirosis
- Goats, pigs, cattle: Q fever
- Abattoirs, veterinary practitioners: brucellosis
- Soil: blastomycosis
- Decaying wood, caves, chicken: histoplasmosis
- Florists, gardeners, plants, straw: sporotrichosis

**Radiological features of pneumonia**

- Lobar: consolidation, with air bronchogram, no volume loss
- Lobular (bronchopneumonia): multi-focal nodular or reticulonodular opacities which are patchy and/or confluent patchy
- Interstitial: diffuse bilateral reticular, nodular or reticulonodular opacities which are patchy and/or confluent
- Round pneumonia: spherical opacity simulating a parenchymal mass
- Cavitary lesions
- Pneumothorax or pneumatocele (Pneumocystis jiroveci)

**Causes of non-resolving pneumonia**

- Complications of pneumonia: empyema; lung abscess; parapneumonic effusion
- Pneumonia mimics: neoplasm (bronchogenic carcinoma, lymphoma, bronchoalveolar carcinoma); inflammatory disorders (cryptogenic organizing pneumonia-failure to improve despite several courses of antibiotics); eosinophilic pneumonia; systemic vasculitis-Wegener’s granulomatosis
- Unusual causative organism: tuberculosis; atypical mycobacteria; fungal pneumonia (aspergillosis, cryptococcosis, mucormycosis, histoplasmosis, coccidiodomycosis)
- Resistant organism: drug-resistant Streptococcus pneumoniae; MRSA
Causes of recurrent focal pneumonia

• Causes within the lumen: foreign body; endobronchial tuberculosis; tumour
• Causes affecting the wall: asthma; bronchomalacia
• Causes from external compression: enlarged hilar lymph nodes; enlarged left atrium compressing the left main bronchus
• Infected developmental lung malformations: sequestration; bronchogenic cyst; cystic adenomatoid malformation
• Other causes: bronchiectasis

Risk factors for lung abscess

• Aspiration: alcohol abuse; seizure; dental and periodontal disease; neuromuscular disorders with bulbar dysfunction; gastro-oesophageal reflux; oesophageal dysmotility
• Bronchial obstruction: neoplastic; non-neoplastic
• Immunosuppression

Causes of recurrent respiratory tract infections in children

• Cystic fibrosis
• Ciliary dysfunction: primary ciliary dyskinesia; Kartagener’s syndrome; bronchiectasis; immotile cilia syndrome
• Structural lung disease: congenital cystic adenomatoid malformation; pulmonary sequestration
• Immunodeficiency: transient hypogammaglobulinaemia of infancy; X-linked agammaglobulinemia; combined variable immunodeficiency; hyper-IgM syndrome; secondary IgA deficiency
• Oesophageal disease: achalasia; progressive systemic sclerosis; pharyngeal pouch
• Cardiovascular disease: Left to right shunts: atrial septal defect; ventricular septal defect; heart failure

Causes of cavitating lung lesions

Infection:

• Pulmonary tuberculosis
• Cavitating pneumonia/lung abscess: staphylococcus aureus; Gram negative bacteria, e.g. Klebsiella pneumoniae

• Infected post-pneumonia pneumatocele
• Fungal infection: aspergillosis; mucormycosis; histoplasmosis; pneumocystis
• Parasitic infections: hydatid cyst

Neoplasia:

• Primary malignancy: bronchogenic carcinoma, lymphoma, Kaposi’s sarcoma
• Secondary malignancy: especially squamous cell carcinoma, adenocarcinoma, sarcoma

Vascular:

• Pulmonary embolism with infarction
• Septic pulmonary emboli

Autoimmune:

• Wegener’s granulomatosis
• Rheumatoid nodules

Trauma: pulmonary contusion; lung laceration

The mnemonic CAVITY is useful: cancer, autoimmune, vascular, infection, trauma, and youth (pulmonary sequestration, bronchogenic cyst)

Risk factors for aspiration pneumonia and lung abscess

• Oropharyngeal sepsis: periodontal disease; gingivitis; dental abscess; tonsillar abscess
• Impaired level of consciousness: drugs; alcohol; coma; seizure; shock; general anaesthesia
• Impaired cough and gag reflex: vocal cord paralysis; myopathy; myelopathy
• Impaired oesophageal function: achalasia, stricture, diverticula
• Vomiting: ileus, intestinal obstruction

Key diagnoses to be recognized in chest trauma

Primary survey

• Tension pneumothorax
• Dyspnoea; tachypnoea; increasing hypoxaemia; haemodynamic instability, with hypotension and narrowing pulse pressure
• Contralateral tracheal shift
• Hyperexpanded chest
• Hyper-resonance with absent breath sounds
• Jugular venous distension
• Flail chest (anterior or lateral double fractures of three or more adjacent ribs; paradoxical segmental chest wall movement over several respiratory cycles and during coughing)
• Open sucking chest wound
• Massive haemothorax
• Cardiac tamponade

Secondary survey

• Contained rupture of thoracic aorta
• Rupture of tracheo-bronchial tree
• Oesophageal perforation
• Diaphragmatic rupture (asymmetrical chest expansion; absent breath sounds; tracheal deviation; elevated hemidiaphragm with loss of normal contour, and intra-thoracic herniation of a hollow viscus (stomach, colon or small bowel) on chest x-ray)
• Myocardial contusion
• Pulmonary contusion

Features of tracheo-bronchial tree injury

• Cervical trachea injury: hoarseness; stridor; haemoptysis; cervical subcutaneous emphysema
• Intra-thoracic tracheo-bronchial injury: massive subcutaneous emphysema, unilateral or bilateral pneumothorax/haemothorax, pneumomediastinum, mediastinal haematoma, peribronchial air and partial pulmonary atelectasis; fallen lung sign caused by atelectatic lung falling infero-laterally from the hilum in the lower medial pleural space; failure of re-expansion of the lung after chest tube placement, related to massive air leak from bronchopleural fistula; abnormal migration of tip of endotracheal tube; tension pneumothorax with positive pressure ventilation

Radiological features of traumatic aortic injury

• Mediastinal widening, with mediastinal-chest ratio >0.25 and superior mediastinal width >8 cm

• Loss of, or abnormal, aortic knob contour
• Tracheal deviation to the right
• Wide right paratracheal stripe
• Depression of left main stem bronchus
• Oesophageal deviation (nasogastric tube) deviation to the right
• Left apical pleural cap: a curvilinear opacity with mass effect on the lung apex
• Wide left paraspinal line
• Left haemothorax
• Fractures of upper ribs

Causes of mediastinal haematoma

• Aortic injury
• Venous haemorrhage
• Thoracic vertebral body fracture
• Spinal ligamentous injuries

Features of pulmonary contusion

• The commonest injury following blunt chest wall trauma in children
• Caused by alveolar capillary disruption, leading to alveolar haemorrhage and interstitial oedema
• Focal or multi-focal consolidation, crossing fissures and lobes, with sub-pleural sparing (1–2 mm region of non-opaque sub-pleural lung separating the area of consolidation from the adjacent chest wall)
• Chest x-ray signs may take 4–6 h to develop and progress over 24–48 h; resolution within 3–5 days usually

The clinical features of rib fracture include:

• Pleuritic chest pain
• Point tenderness
• Production of pain by anteroposterior or lateral chest wall compression (“springing”)
• Local crepitus
• Subcutaneous emphysema
• 1st and 2nd rib fractures may be associated with vascular injury (aorta, subclavian artery) or brachial plexus injury
• 11th and 12th rib fractures may be associated with visceral injury (liver, spleen, kidneys)
Features of sternal fractures

- Caused by direct trauma, such as anteroposterior compression from seat belt or steering wheel injuries, with posterior displacement of the distal sternal fragment, or indirect trauma, such as hyperflexion and axial compression, with posterior displacement of the proximal sternal agent. Manubrio-sternal joint dislocation may also result by these mechanisms.
- Can be associated with myocardial or pulmonary contusion
- Usually diagnosed on a lateral view of the sternum
- Isolated sternal fracture with normal 12 lead ECG and normal chest x-ray carries a very low risk of intrathoracic injury

Causes of subcutaneous emphysema in the chest wall
Air introduced from outside

- Penetrating injury
  - Air from within
- Pneumothorax
- Acute severe asthma
- Oesophageal perforation
- Tracheo-bronchial disruption (subcutaneous emphysema, persistent air leak, haemoptysis)

Predictors of difficult airway (based on oropharyngeal examination, assessment of atlanto-occipital joint mobility and of the space between the horizontal ramus of mandible and the hyoid bone-the potential displacement space)

- Small mouth
- Limited mouth opening (3 cm or less, two fingerbreadths or less); limited temporomandibular joint mobility; trismus
- Protruding upper incisors
- Dental implants
- Short inter-incisor distance
- Large tongue (macroglossia)
- Cleft or high arched narrow palate

- Mandibular hypoplasia (micrognathia)
- Short or muscular neck
- Limited neck mobility
- Obesity

In addition, current airway obstruction, inflammation and haemorrhage are also predictors of a difficult airway

Causes of hypotension with chest trauma
Bleeding

- Massive haemothorax

Reduced venous return

- Tension pneumothorax (increased intrathoracic pressure)
- Cardiac tamponade (increased intra-pleural pressure)

Direct cardiac injury (cardiogenic shock) (no blood loss)

- Myocardial contusion
- Myocardial infarction
- Conduction abnormalities
- Air embolism

Causes of cardiovascular collapse after rapid sequence induction of anaesthesia and positive-pressure ventilation following chest trauma

- Hypovolaemia
- Unrecognised oesophageal intubation, with hypoxaemia
- Tension pneumothorax
- Pericardial tamponade
- Anaphylaxis
- Systemic air embolism
- Severe blunt cardiac trauma

Causes of bilateral hilar enlargement

- Lymph node enlargement (lobulated)
- Sarcoïdosis
- Malignancy: lymphoma; carcinoma
• Infection: tuberculosis; mycoplasma; fungal infection
• Inorganic dust disease: silicosis; berylliosis
• Pulmonary artery enlargement (smooth, with peripheral decreased vascular markings-peripheral pruning)
• Pulmonary arterial hypertension (main pulmonary artery >29 mm in diameter and/or larger than adjacent ascending aorta)
• Left to right intra-cardiac shunt
• High output state
• Cystic fibrosis

**Causes of hilar lymphadenopathy**
Bilateral asymmetrical and unilateral

- Tuberculosis: primary tuberculosis
- Fungal, atypical mycobacterial, viral, tularaemia, anthrax
- Metastatic or primary hilar tumour (bronchogenic carcinoma)
- Lymphoma
- Sarcoidosis,
- Inorganic dust disease: silicosis, berylliosis
- Angio-immunoblastic lymphadenopathy
- Drug reaction

Bilateral symmetrical

- Sarcoidosis (Garland’s sign) (bilateral hilar and right paratracheal node enlargement constitutes the 1–2–3 sign)
- Viral infection (adenovirus, infectious mononucleosis)
- Other causes mentioned can be symmetrical

**Causes of diffuse confluent alveolar opacities** (widespread pulmonary consolidation) (oedema, exudate, blood, neoplasm)
- Pulmonary oedema
- Cardiogenic
- Fluid overload/acute kidney injury
- Non-cardiogenic
- Pneumonia
- Pneumocystis jiroveci

- Gram negative bacteria
- Influenza
- Fungi: histoplasmosis; aspergillosis

Pulmonary haemorrhage (Goodpasture’s syndrome)
- Neoplasm
- Bronchio-alveolar cell carcinoma
- Lymphoproliferative disorder: lymphoma
- Alveolar proteinosis
- Acute silica inhalation
- AIDS
- Reaction to drugs
- Extrinsic allergic alveolitis

**Causes of abnormal chest x-ray in immunocompromised patients**

- Infection: bacterial; mycobacterial; fungal; viral
- Neoplasm: lymphoma; leukaemia; metastases/recurrence of primary tumour
- Transfusion reaction
- Graft versus host disease after bone marrow transplantation
- Radiation pneumonitis: acute; chronic
- Adverse drug reaction: early (non-toxic); late (cytotoxic)
- Haemorrhage

**Causes of increased transradiancy of one hemithorax**
Technical-artifactual

- Rotation of patient (scoliosis; supine position)
- Off-centring of grid (grid cut-off or anode heel effect) (causes diffuse increased haziness of the hemithorax)
- Overexposure; especially in AP view

Chest wall–relative lack of overlying soft tissues

- Loss of soft tissues: mastectomy; Poland’s syndrome
Pleura: pneumothorax; especially in supine patient
Lung:
  Compensatory over-inflation
  • Post-lobectomy
  • Overlooked lobar collapse (especially left lower lobe)
  • McLeod’s syndrome: unilateral emphysemalike condition following early childhood lower respiratory tract infections

Reduced pulmonary perfusion
  • Hypoxic vasoconstriction due to hypoventilation caused by inhaled foreign body or endobronchial tumour
  • McLeod’s syndrome
  • Recurrent pulmonary emboli (rarely unilateral)

**Causes of asymmetrical lung volumes**

**Increased ipsilateral density**
  • Small lung: atelectasis; central airway obstruction; congenital venolobar syndrome (hypoplasia of the lung associated with pulmonary vascular abnormalities); diaphragmatic elevation/paresis
  • Large lung

**Decreased ipsilateral density**
  • Small lung: McLeod’s syndrome
  • Large lung: primary/secondary congenital over-inflation; central airway obstruction with ball-valve effect

**Normal density**
  • Small lung: hypoplasia; interrupted pulmonary artery

**Causes of mediastinal masses**

**Anterior mediastinum (30%)**
  (5Ts: thyroid mass; teratoma; thymic mass; terrible-lymphoma; thoracic aorta)
  • Congenital: normal thymus; thymic cyst; thymomegaly; Morgagni hernia
  • Inflammatory: mediastinitis; lymphadenopathy; sternal inflammatory disease
  • Neoplasm: lymphoma-leukaemia; teratoma and other germ cell tumours; seminoma; thymoma; thyroid or parathyroid tumour; hamartoma; mesenchymal tumour; lipoma
  • Traumatic: haematoma; sternal fracture; thymic haemorrhage
  • Vascular: aneurysm of sinus of Valsalva; anomalous vessel
  • Miscellaneous: histiocytosis; sarcoidosis
  • Thyroid (goitre or neoplasm)

**Middle mediastinum (30%)**
  • Congenital: foregut cyst (enteric, respiratory); oesophagus (hiatus hernia, achalasia); extension of normal thymus
  • Inflammatory: mediastinitis; lymphadenopathy
  • Neoplasm: lymphoma-leukaemia; bronchogenic carcinoma; metastases cardiac tumour or aneurysm
  • Traumatic: haematoma; diaphragm rupture
  • Vascular (lesions of the aorta and great vessels: aneurysm-aorta; dilated superior or inferior vena cava; anomalies of great vessels)
  • Miscellaneous: pancreatic pseudocyst
  • Dilated oesophagus and oesophageal masses: tumour: benign, malignant; oesophageal diverticulum; dilated oesophagus

**Posterior mediastinum (40%)**
  • Congenital: foregut cyst; lateral meningocele; Bochdalek hernia; ectopic thymus
  • Inflammatory: mediastinitis; spinal inflammatory disease
  • Neoplasm: neurogenic (neuroblastoma, ganglioneuroma, ganglioneuroblastoma); nerve root tumours (schwannoma, neurofibroma, malignant schwannoma); paraganglionic cell tumours (chemodectoma, phaeochromocytoma); spinal tumour (metastases, primary bone tumour); lymphoma; mesenchymal
tumour (fibroma, lipoma, leiomyoma, haemangioma, lymphangioma)

• Traumatic: haematoma; pseudo-meningoele
• Vascular: aneurysm of descending aorta
• Miscellaneous: histiocytosis; extramedullary haematopoiesis

Recognition of abnormal mediastinal widening

Widening of the mediastinum is most often due to technical factors such as patient positioning or the projection used. Rotation, incomplete inspiration, or an AP view, may all exaggerate the width of the mediastinum, as well as heart size.

• Mediastinal width >6 cm in erect PA film
• Mediastinal width >8 cm in supine AP chest film
• Mediastinal width >7.5 cm at aortic knob
• Ratio of mediastinal width to chest width >0.25 at aortic knob

Causes of diffuse mediastinal widening

Smooth

• Mediastinal lipomatosis
• Malignant infiltration: lymphoma; squamous cell carcinoma; adenocarcinoma
• Mediastinal haemorrhage: traumatic arch/great vessel laceration; arterial bleeding; venous bleeding; SVC/right atrial laceration
• Mediastinitis: acute (suppurative); chronic (sclerosing): histoplasmosis, tuberculosis, idiopathic

Lobulated

• Lymph node enlargement
• Thymic mass
• Germ cell neoplasm
• Neurofibromatosis
• Vascular lesions: tortuous great vessels; SVC occlusion

Pneumomediastinum

Pneumomediastinum indicates perforation of some portion of either the respiratory or gastrointestinal tracts

Features of pneumomediastinum

• Free mediastinal air, causing streaky radiolucencies in the mediastinum and outlining the heart borders, the central diaphragm (causing visualisation of the entire diaphragm) and the thymus, and extending to the neck
• Subcutaneous emphysema
• Ring shaped radiolucencies around the aorta and pulmonary artery

Causes of pneumomediastinum

• Alveolar rupture: spontaneous; positive pressure ventilation; compressive trauma to the chest; rupture of lung by rib fracture with tracking of air into the mediastinum by way of the chest wall and neck; asthma
• Traumatic laceration of trachea or a central bronchus (associated with double wall sign, caused by intramural air in proximal airways leading to visualisation of both sides of the bronchial wall, and the fallen lung sign of collapse of the inferior lung)
• Spontaneous or iatrogenic perforation of pharynx, oesophagus, duodenum, colon, or rectum, with tracking of air into the mediastinum: Boerhaave syndrome; endoscopic intervention (biopsy, dilatation, sclerotherapy)
• Following placement of an intercostal tube or mediastinoscopy or similar invasive procedures

Defining features of solitary pulmonary nodule

• Single lung lesion
• Less than 3 cm in diameter
• Surrounded on all sides by lung parenchyma
• Benign patterns of calcification: central; diffuse; popcorn; laminated

Causes of solitary pulmonary nodule

• Neoplastic: malignant (bronchogenic carcinoma; solitary metastasis; lymphoma; carcinoid tumour); benign (hamartoma; adenoma; benign connective tissue and neural tumours)
- Inflammatory: granuloma; lung abscess; rheumatoid nodule; inflammatory pseudotumour (plasma cell granuloma)
- Congenital: arteriovenous malformation; lung cyst; broncho-pulmonary sequestration; bronchial atresia with mucoid impaction
- Miscellaneous: pulmonary infarct; intrapulmonary lymph node; mucoid impaction; haematoama; amyloidosis; normal confluence of pulmonary veins; conglomerate mass of silicosis

Mimics of solitary pulmonary nodule
- Nipple shadow
- Cutaneous lesion
- Rib or other bone lesion
- Vanishing pseudotumour of congestive cardiac failure (loculated pleural effusion)

Features suggestive of malignancy
- Clinical: older age; COPD; previous malignancy; environmental exposures; haemoptysis; lung cancer in first degree relatives
- Radiological: speculated appearance; eccentric or speculated calcification; thick walled cavitation

Causes of multiple pulmonary nodules
Neoplastic
- Metastases
- Malignant lymphoma/lymphoproliferative disorders

Inflammatory
- Granulomas
- Fungal and opportunistic infections
- Septic emboli
- Rheumatoid nodules
- Granulomatosis with polyangiitis (Wegener’s granulomatosis)
- Sarcoidosis
- Langerhans’ cell histiocytosis

Congenital
- Arteriovenous malformations (Osler-Weber-Rendu disease)

Miscellaneous
- Haematomas
- Pulmonary infarcts
- Occupational (silicosis)

Causes of an opaque hemithorax
Ipsilateral pathology
  With mediastinum central or same side (ipsilateral volume loss)
  - Lung aplasia/agenesis
  - Lung hypoplasia
  - Collapse/consolidation of lung/lobe
  - Complete left lung collapse secondary to central obstructing tumour
  - Central mucus plug(postoperative)
  - Empyema with collapse/consolidation
  - Post-pneumonectomy: elevated gastric bubble; leftward shift of mediastinum; surgical clips in the left hemithorax in the vicinity of the left main stem bronchus

With mediastinum pushed to opposite side
  - Pleural fluid: large pleural effusion (often malignant); chylothorax; haemothorax; empyema
  - Mesothelioma
  - Diaphragmatic hernia (fluid-filled)
  - Thoracic meningocoele
  - Tumours

Contralateral pathology with mediastinal shift +/− compression
  - Emphysema- congenital lobar; obstructive-secondary to tumour; foreign body
  - Cystic adenomatoid malformation
  - Tension pneumothorax
  - Diaphragmatic hernia(air-filled)
Causes of unilateral elevation of the diaphragm

a. Causes above the diaphragm

Diminished pulmonary volume:
- Atelectasis
- Lobectomy

Phrenic nerve paralysis (paradoxical movement on bedside ultrasound)

Splinting of diaphragm, with rib fractures

Sub-pulmonic effusion
- Blunted posterior costo-phrenic sulcus
- A measurable distance between the stomach bubble and the inferior surface of the lung
- Usually demonstrable on a decubitus film

b. Diaphragmatic causes

Eventration of the diaphragm

Diaphragmatic masses

c. Causes below the diaphragm

Intra-abdominal process:
- Organomegaly
- Gaseous distension of the stomach or splenic flexure
- Subphrenic inflammatory disease: subphrenic abscess; liver or splenic abscess

Causes of focal contour abnormality of the diaphragm

- Hernia (hiatus hernia; foramen of Morgagni; foramen of Bochdalek; traumatic diaphragmatic rupture)
- Partial eventration
- Diaphragmatic tumours
- Basal pleural tumour
- Loculated sub-pulmonic effusion

Patterns of interstitial lung disease on chest x-ray

- Linear (due to thickening of interlobular septa)

Causes of diffuse interstitial lung disease

- Granulomatous lung disease: sarcoidosis; with vasculitis: eosinophilic granulomatosis with polyangiitis
• Connective tissue disease with lung involvement: rheumatoid arthritis; SLE; scleroderma; dermatomyositis and polymyositis

• Pulmonary infiltration with eosinophilia: allergic bronchopulmonary aspergillosis; tropical pulmonary eosinophilia; helminth infections

• Idiopathic pulmonary fibrosis

• Idiopathic interstitial pneumonias: idiopathic; non-specific; lymphoid; cryptogenic organizing pneumonia

• Drug induced pulmonary disease: antimicrobial agents (nitrofurantoin); cardiovascular drugs (amiodarone; ACE inhibitors; statins); disease-modifying anti-rheumatic drugs (methotrexate; sulphasalazine); cancer chemotherapeutic agents (bleomycin; selective EGFR inhibitors); recreational drugs (heroin; methadone)

• Inhalation secondary to occupational or environmental exposure: inorganic dusts (pneumoconioses-e.g. asbestosis; silicosis; berylliosis; coal dust); animal protein (bird proteins-exotic birds, chickens, pigeons); grain dust (farming); mould (showers; indoor hot tubs)

**Causes of rib notching**, on Inferior Surface

**Arterial obstruction**

• Coarctation of the aorta: 4th–8th ribs bilaterally

• Aortic thrombosis

• Subclavian obstruction: after Blalock-Taussig shunt for Tetralogy of Fallot

• Pulmonary oligaemia

• Takayasu’s arteritis

**Venous obstruction**

• Superior vena caval obstruction

**Arteriovenous**

• Pulmonary arteriovenous malformation

• Chest wall arteriovenous malformation

**Neurogenic**

• Neurofibromatosis

• Normal variant