Respiratory

1. Dyspnoea
2. Cough
3. Obstructive vs. restrictive
4. Emphysema
5. Chronic bronchitis
6. Asthma
7. Bronchiectasis
8. Pneumonia
9. Pleural effusion
10. Pulmonary embolism
11. Interstitial lung disease
12. Lung cancer
13. Tuberculosis
14. Interpreting chest x-rays

Editorial team
Katie Newman, Rebecca Ng, Ashleigh Spanjers

Peer reviewed in 2011 by
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Respiratory Physician
**Dyspnoea**

**Definition**
Subjective sensation of SOB. An abnormal, uncomfortable awareness of respiration.

### Types of Dyspnoea

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute dyspnoea</td>
<td>$\leq$ 1 month</td>
</tr>
<tr>
<td>Chronic dyspnoea</td>
<td>$&gt;1$ month</td>
</tr>
<tr>
<td>Exertional dyspnoea</td>
<td>Dyspnoea on physical exertion</td>
</tr>
<tr>
<td>Orthopnoea</td>
<td>Dyspnoea when supine due to redistribution of fluid in lung. Patient may need to be upright or propped on a number of pillows to sleep.</td>
</tr>
</tbody>
</table>

### Differentials for acute dyspnoea

**Respiratory**
- Asthma
- Bronchitis
- Pneumonia
- Pneumothorax
- Acute pulmonary oedema
- Pulmonary embolism
- ARDS
- Allergen exposure
- Foreign body obstruction

**Cardiac**
- Cardiac tamponade
- Shock

**Other**
- Psychogenic
- Haemolysis
- Rib fracture
- CO poisoning
- Metabolic acidosis

### Differentials for chronic dyspnoea

**Respiratory**
- Bronchiectasis
- COPD
- Chronic anaemia
- Infiltrative tumour
- Interstitial lung disease
- Pleural effusion
- Pulmonary hypertension

**Cardiac**
- Heart failure
- Pericardial effusion
- Restrictive pericarditis

**Other**
- Severe obesity
- Ankylosing spondylitis
- Kyphoscoliosis
- Neuromuscular disease

### Examination

**Inspection**
- Respiratory rate (brady <8bpm, tachy >25bpm)
- Cyanosis (peripheral in hands, central in tongue)
- Use of accessory muscles of respiration (sternocleidomastoids, scalene)
- Pursed lips breathing
  - COPD
- Increased anterioposterior diameter/barrel chest
  - COPD
- Elevated JVP (>5cm)
  - Heart failure
- Tracheal shift from midline
  - Pneumothorax, pleural effusion

**Percussion**
- Dull note
  - Consolidation (pneumonia)
- Stony dull note
  - Fluid (pleural effusion)
- Hyperresonant note
  - Air trapping (COPD)

**Auscultation**
- Absent unilateral breath sounds
  - Pneumothorax
- Fine crackles
  - Interstitial LD
- Coarse crackles
  - Heart failure
- Inspiratory and expiratory crackles
  - Bronchiectasis
- Wheeze
  - Asthma
- Stridor
  - Upper airway obstruction
- S3 gallop
  - Heart failure
- Fixed S2 split
  - Pulmonary hypertension

### Questions to ask on history

**Onset?**
- Sudden or gradual, sporadic or in certain circumstances such as on exertion or exposure to an allergen or at rest

**Duration?**
- Acute or chronic

**Exercise tolerance?**
- Steps climbed/distance walked?

**Effect on function?**
- NYHA classification scale

**Exacerbating and relieving factors?**
- Use of puffers, resting, change of setting

**Diurnal variation?**
- Asthma
- Worse when lying flat?

**Orthopnoea**
- How many pillows does the patient sleep with?

**Paroxysmal nocturnal dyspnoea**
- Does it ever wake patient from sleep gasping for breath?

**Associated symptoms?**
- Chest pain, swelling of ankles, panic or anxiety, cough

### Possible associated symptoms/signs

**Wheeze**
- Airway disease - asthma, COPD, anaphylaxis

**Stridor**
- Obstruction - foreign body, tumour, acute epiglottitis, anaphylaxis, trauma

**Chest pain**
- Cardiac event, pericarditis, pneumothorax, PE

**Crackles**
- Heart failure with pulmonary oedema, pneumonia, bronchiectasis, fibrosis

**Cough with sputum production**
- Pneumonia, bronchitis

**Cough with haemoptysis**
- Pneumonia, bronchitis, PE, malignancy

**Oedema of ankles, sacrum**
Pathophysiology

Respiration is regulated through the CNS. The respiratory centre is composed of
- Dorsal respiratory group of the medulla (inspiration)
- Ventral respiratory group of the medulla (expiration)
- Pneumotaxic centre in the pons (rate and depth of breathing).

The respiratory centre transmits these efferent signals to muscles of respiration.

The ultimate goal of respiration is maintenance of O2, CO2 and H+ in the blood which occurs via afferent signals from
- Chemosensitive area of the medulla (CO2 and H+)
- Peripheral chemoreceptors of the carotid and aortic bodies (O2)

The sensation of dyspnoea arises from a mismatch between afferent and efferent signals. Factors that enter into the development of the sensation of dyspnoea
1. Abnormality of respiratory gases in the bodily fluids (primarily hypercapnia, secondarily hypoxia)
2. Amount of work that must be performed by respiratory muscles to provide adequate ventilation
3. State of mind (neurogenic/psychogenic)

Acute management
Oxygen
- If hypoxic (oxygen not given for dyspnoea alone)
Address underlying cause

NYHA Functional Classification Scale

Class I (asymptomatic left ventricular dysfunction)
- No limitations, ordinary physical activity does not cause undue fatigue, dyspnoea or palpitations
Class II (mild CHF)
- Slight limitation of physical activity, ordinary physical activity results in fatigue, dyspnoea, palpations or angina
Class III (moderate CHF)
- Marked limitation of physical activity, less than ordinary activity leads to symptoms
Class IV (severe CHF)
- Unable to carry on any physical activity without discomfort, symptoms of CHF present at rest

Spirometry

<table>
<thead>
<tr>
<th>DDx</th>
<th>FVC</th>
<th>FEV1</th>
<th>TLC</th>
<th>RV</th>
<th>DLCO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emphysema</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Chronic bronchitis</td>
<td>↓</td>
<td>↓</td>
<td>=</td>
<td>↑</td>
<td>=</td>
</tr>
<tr>
<td>Asthma</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
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<tr>
<td>Intertstitial LD</td>
<td>↓</td>
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<td>↓</td>
<td>↓</td>
<td></td>
</tr>
<tr>
<td>Kyphoscoliosis</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>=</td>
<td>=</td>
</tr>
<tr>
<td>HF (early, ↑blood flow)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>HF (late, pulmonary oedema)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>PE</td>
<td>-</td>
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<td>↓</td>
</tr>
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**Cough**

**Definition**: Cough is deep inspiration followed by explosive expiration and is a defence mechanism which enables the airways to be cleared of secretions and foreign bodies. A common presenting symptom.

**Classification**:

<table>
<thead>
<tr>
<th></th>
<th>Acute cough: &lt;3 weeks</th>
<th>Sub-acute cough: 3-8 weeks</th>
<th>Chronic cough: &gt;8 weeks</th>
</tr>
</thead>
</table>

**History**

**Cough**

**Onset and duration**:

<table>
<thead>
<tr>
<th>Acute cough</th>
<th>Chronic cough with wheezing</th>
<th>Chronic dry, irritating cough</th>
<th>Chronic dry cough</th>
<th>Paroxysmal nocturnal cough</th>
<th>Chronic cough productive of large volumes of purulent sputum</th>
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</thead>
<tbody>
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<td>Acute cough with fever and symptoms of respiratory tract infection</td>
<td>Chronic cough with wheezing</td>
<td>Chronic dry, irritating cough</td>
<td>Chronic dry cough</td>
<td>Paroxysmal nocturnal cough</td>
<td>Chronic cough productive of large volumes of purulent sputum</td>
</tr>
<tr>
<td>Pneumonia, acute bronchitis</td>
<td>Asthma</td>
<td>Oesophageal reflux (acid irritation of lungs)</td>
<td>ACE inhibitors (build-up of bradykinin)</td>
<td>Cardiac failure, acid reflux (positional fluid shift)</td>
<td>Bronchiectasis</td>
</tr>
</tbody>
</table>

**Temporal changes in cough**

- Cough worse at night: Asthma, cardiac failure
- Cough worse after food or drink: Oesophageal reflux, tracheo-oesophageal fistula

**Character**:

- Barking: Inflammation, epiglottitis
- Loud, brassy: Tracheal compression
- Hollow, bovine: Recurrent laryngeal nerve palsy (inability of vocal cords to completely close)
- Loose, productive: Chronic bronchitis, bronchiectasis, pneumonia (excessive bronchial secretions), post nasal drip
- Dry, irritating: Chest infection, asthma, bronchial carcinoma, cardiac failure, interstitial lung disease, ACE inhibitor

**Sputum**

- Enquire about volume, colour and character:
  - Large volume purulent (yellow or green): Bronchiectasis, lobar pneumonia
  - Foul-smelling dark-coloured sputum: Lung abscess with anaerobic organisms
  - Pink, frothy: Pulmonary oedema (NOT sputum, originates from trachea)

**Haemoptysis**

Coughing up of blood.

May indicate serious underlying disease (e.g. malignancy) and always requires further investigation.

**Other associated symptoms and signs**

- Dyspnoea, wheeze, chest pain, fever, hoarseness, night sweats

**Complete full history, pertinently:**

- Past medical history (especially respiratory diseases), medications (ACE inhibitors), allergies (atopy), smoking history (pack years), environmental and occupational exposures (chemicals, dusts), travel history

**Examination**

- Respiratory examination
  - Inspection
    - Sputum cup
  - Auscultation
    - Crackles
    - Wheeze
    - Consolidation
  - Other
    - Sinus tenderness
    - Rhinitis

**Investigations**

**Sputum MC&S**

- Gram stain for infectious causes

**CXR**

- If Hx and Ex do not clearly elucidate aetiology

**Further testing to rule in/rule out diagnoses**:

- Bronchodilator test (reversible airway obstruction)
- Lung function tests (reveal obstructive/restrictive/other defects)
- CT (lesions, masses in airway)
- Bronchoscopy

**Common aetiologies**

- Post nasal drip (allergic, perennial non-allergic and vasomotor rhinitis, acute nasopharyngitis, sinusitis)
- URI (pharyngitis, tracheitis)
- LRTI (bronchitis, pneumonia)
- Asthma
- Gastrooesophageal reflux
- Laryngopharyngeal reflux
- ACE inhibitors
- Structural changes (bronchiectasis, tumours, interstitial lung disease)
- Occupational or environmental exposures (smoke, pollen, dusts, chemicals)
Pathophysiology

Coughing
- Deep inspiration is followed by explosive expiration.
- Increased flow rates of air (may approach the speed of sound)
- Defense mechanism (clearance of foreign bodies/secretions from airways)

Pathway
- Chemical cough receptors
  - Located in epithelium of the respiratory tracts, pericardium, oesophagus, diaphragm, stomach.
  - Stimuli include temperature, acid, other chemical irritants
  - Stimulation activates of cough reflex through transient receptor potential vanilloid type 1 (TRPV1) and transient receptor potential ankyrin type 1 (TRPA1) ion channel classes.
- Mechanical cough receptors
  - Located in larynx, trachea, bronchial tree
  - Stimuli include touch and displacement
- Reflex arc
  - Stimulation of cough receptor → afferent impulse → vagus n → medullary cough centre → efferent impulse → vagus, phrenic, spinal motor n → expiratory musculature → cough
  - There is also some descending input from higher cortical centres

Acute management
Empiric treatment is directed at common causes of cough.

Removal of stimuli
- Avoidance of stimuli (smoking, occupational exposures, environmental pollutants), cessation of ACE inhibitors

Antibiotics
- If infective aetiology is suspected
- Empirical treatment according to TGA

Anti-histamines and decongestants
- First generation combination anti-histamine and decongestant
- Where post-nasal drip is suspected

Inhaled glucocorticoids
- Where chronic inflammation is suspected or obstructive defect is present

Anti-cholinergics
- Ipratropium bromide
- Blocks efferent limb of cough reflex and decreases cough receptor stimulus

Bronchodilators
- If obstructive defect is found on LFTs

Protein pump inhibitor
- Where GORD is suspected

Anti-tussives
- Symptomatic relief only where aetiology cannot be identified
- Peripherally acting antitussives (work on peripheral cough receptors) such as Benzonatate.
- Centrally acting antitussives (↑ threshold of impulse required to activate medullary cough centre) which may be opioid (e.g. Codeine) or non-opioid (Dextromethorphan)

Algorithm for sub-acute and chronic cough

![Algorithm for sub-acute and chronic cough](image)

# Obstructive vs. restrictive lung disease

## Obstructive lung disease

### Pathophysiology \(^{1,5,6,9}\)
Increased resistance to airflow due to partial or complete obstruction of the airways at any level of the respiratory tract resulting in decrease in maximal expiratory air flow.

### Aetiology \(^{5,6}\)
- Asthma
- Emphysema (COPD)
- Chronic bronchitis (COPD)
- Bronchiectasis

### Presentation \(^{1,2,5}\)
*Typical presentation:* dyspnoea, productive cough, wheeze
*Fever and systemic signs (if infective exacerbation)*
*Typical history:* smoking (COPD), past medical history (respiratory tract infections - bronchiectasis, atopy – asthma)

### Examination \(^{1,2,5}\)
*Inspection:* Barrel chest, pursed lips breathing, use of accessory muscles of inspiration and indrawing of intercostal muscles, cachexia and weight loss, no clubbing
*Palpation:* reduced chest expansion
*Percussion:* hyperresonant percussion note
*Auscultation:* reduced air entry, wheeze
*Other:* signs of RHF

### Investigations \(^{2,5,6}\)
*CXR:*
- Hyperinflation, decreased peripheral vascular markings, bullae in lung parenchyma

*Lung function tests:*
- Obstructive defect
  - FEV1 <80% of predicted, FEV1/FVC: 0.7
  - Bronchodilator test
    - Reversible: asthma
    - Irreversible: COPD
    - Often mixed component present
- Increased lung volumes
  - ↑TLC, ↑RV
- DLCO
  - ↓DLCO = emphysema
  - Normal DLCO = chronic bronchitis

## Restrictive lung disease

### Pathophysiology \(^{5,6,9,10}\)
Decreased expansion of lung parenchyma due to chronic inflammation of the lung resulting in damage to alveolar wall and surrounding structures. Leads to decreased viable lung for gas exchange and tissue scarring and fibrosis resulting in restriction of movement of the lung.

### Aetiology \(^{5}\)
*Interstitial lung disease:*
  - Idiopathic pulmonary fibrosis: sarcoidosis, vasculitides, haemorrhagic syndromes, auto-immune disorders
  - Exposures: silicates, carbon, metals, dusts, birds
  - Medications: antibiotics, anti-inflammatories, anti-arrhythmics, chemotherapeutic agents
  - Chest wall disorders: kyphoscoliosis, obesity, polio, pleural disease

### Presentation \(^{5}\)
*Typical presentation:* dyspnoea and non-productive cough
*May also present with:* haemoptysis, wheezing, extra-pulmonary signs (reflecting underlying aetiology)
*Typical history:* smoking, occupational/environmental exposures (dusts, chemicals)

### Examination \(^{2,5}\)
*Inspection:* clubbing
*Palpation:* reduced chest expansion
*Percussion:* normal
*Auscultation:* fine or late pan-inspiratory crackles
*Other:* signs of RHF (cor pulmonale from pulmonary hypertension), associated extrapulmonary signs (reflecting underlying aetiology)

### Investigations \(^{5}\)
*CXR:*
- Reticular or reticular nodular infiltrates, diminished lung volumes, hilar and mediastinal lymphadenopathy, (sarcoid), pleural disease, honeycomb lung (IPF)

*Lung function tests:*
- Restrictive defect
  - ↓FEV1, ↓FVC, normal/↑FEV1/FVC
  - Decreased lung volumes
  - ↓VC, ↓TLC
  - Decreased DLCO
  - ↓SaO2 (decreases with walking)

### Spirometry

\[ FEV1/FVC <70% \]
- \[ FEV1 <80\% \text{ of predicted} \]
- \[ FEV1 <60\% \text{ of predicted} \]
- \[ FEV1 <50\% \text{ of predicted} \]

\[ FEV1 >80\% \text{ of predicted} \]
- \[ FEV1 >60\% \text{ of predicted} \]
- \[ FEV1 >50\% \text{ of predicted} \]

Obstruction plus low vital capacity

### Spirometry

\[ FVC <80\% \text{ of predicted} \]
- \[ FEV1/FVC <70\% \]
- \[ FEV1 >80\% \text{ of predicted} \]

\[ FVC >80\% \text{ of predicted} \]
- \[ FEV1/FVC >70\% \]

Obstructive pattern

### References
**Emphysema**

**Definition** \(^1,2,5\) Histological diagnosis of pathological and permanent dilatation (increase in size) of the air spaces distal to terminal bronchioles with destruction of the alveolar walls. A subtype of chronic obstructive pulmonary disease (COPD).

**Presentation** \(^1,2,5\)

Typical presentation:
- Dyspnoea (persistent and exertional)
- Cough (intermittently or daily)
- Sputum production (absent or scant)
- No haemoptysis

History of presentation complaint:
- Dyspnoea: gradual onset (years), ask about exertion required to precipitate dyspnea, rate on NYHA scale
- Cough: ask about onset and duration, character, sputum production, haemoptysis

Acute exacerbation:
- Ask about recent changes in symptoms from normal day-to-day symptoms
- Ask about any identifiable precipitants

Respiratory history:
- Smoking history: age of initiation, amount, pack years, high risk if heavy smoker especially if >70 pack years
- Past medical history: frequent respiratory infections
- Personal history or family medical history: alpha1-antitrypsin deficiency, emphysema or other respiratory diseases

**Examination** \(^1,2,5\)

“Pink puffers”

**Inspection:**
- Dyspnoea
- Barrel chest (increased AP diameter, hyperinflation)
- Pursed lip breathing (increases end expiratory pressure to open airways to minimize air trapping)
- Use of accessory muscles of inspiration (SCM, scalenes)
- In-drawing of lower intercostal muscles in inspiration
- May show signs of cachexia and weight loss
- No clubbing

**Palpation:**
- Reduced chest expansion

**Percussion:**
- Hyperresonant percussion note

**Auscultation:**
- Decreased breath sounds/air entry
- ↑ Forced expiratory time

Acute exacerbation may show:
- Fever
- Tachypnoea
- Cough
- Sputum production
- Early inspiratory crackles

In pre-terminal state, signs of right heart failure may be present:
- Elevated JVP
- Peripheral oedema
- Increased P2, splitting of S2
- Hepatosplenomegaly
- Early inspiratory crackles

**Investigations** \(^2,5,6\)

**Lung function tests:**
- Obstructive defect:
  - FEV1 <80% of predicted
  - FEV1/FVC: 0.7
  - Irreversible (some reversibility may be present on bronchodilator test)
- ↑ TLC, ↑RV
- ↓ DLCO

**CXR:**
- Hyperinflation: >6 anterior ribs seen above diaphragm in mid-clavicular line, flat hemidiaphragms, narrow cardiac shadow
- Large central pulmonary arteries
- Decreased peripheral vascular markings
- Increased radiolucency of lungs
- Bullae in lung parenchyma (radiolucent areas >1cm diameter surrounded by arcuate hairline shadows)
- Cardiomegaly (if cor pulmonale)

**CT:**
- Loss of markings of alveolar walls
- Pancinar: lung bases, genealized paucity of vascular structures
- Centrilobular: upper lobes, holes seen in centre of secondary pulmonary lobules

**ABGs:**
- Low PaO2
- May have high PaCo2 (CO2 retention)

**V/Q Scan:**
- V/Q mismatch
  - High V/Q (ventilatory compensation of undamaged lung)

**Management** \(^5,7\)

Pharmacotherapy:
- Long acting B2 agonists (e.g. salmeterol, eformoterol)
- Inhaled anticholinergics (e.g. tiotropium bromide)
- Combination inhalers (ICS and LABA, e.g.salmeterol/beclometasone, eformoterol/fluticasone)
- Theophylline (very occasionally used)

Steroids:
- 2 week high oral steroid trial to assess reversibility.
- >15% in FEV1 indicates clinical benefit from steroids.
- Avoid long term steroid use

Non-invasive positive pressure ventilation (PPV)

Pulmonary rehabilitation program:
- 6 week exercise training and education

Home oxygen therapy
- Oxygen must be given with care in hypoxia as CO2 retention results in insensitivity of respiratory centres to CO2 = dependence on hypoxia for respiratory drive.
- Supplementary oxygen may therefore result in suppression of respiratory drive and respiratory failure

Reduction of risk factors:
- Smoking cessation, exercise, nutrition, obesity

Preventative medicine:
- Influenza vaccination, pneumococcal vaccination

Acute exacerbations:
- Antibiotics if infective exacerbation
- May require hospitalization
Aetiology\textsuperscript{1,3,6}

Development of COPD:
- A complex process that is not completely understood but is thought to be multifactorial (genetic, biological, behavioural).

Smoking:
- Most common cause of COPD in developed world is exposure to tobacco smoke. \( 50\% \) of chronic smokers develop COPD and almost all COPD patients have significant smoke exposure recorded.

Environmental exposures:
- Occupational dusts, chemicals, air pollution

Genetic alpha1-antitrypsin deficiency:
- Alpha1-antitrypsin is a protease inhibitor. Its deficiency results in loss of inhibition of proteases (such as elastase) which are then able to digest alveolar walls resulting in alveolar destruction as seen in emphysema. Alpha1-antitrypsin deficiency shows a histologically distinct pattern of emphysema (panacinar rather than centrilobular).

Exacerbations of COPD:
- An acute exacerbation is diagnosed on signs and symptoms and may be supported by spirometry showing decreases in FEV1, FVC and PEF due at least in part to airway inflammation.
- Triggered primarily by infection (viral and bacterial) and airborne pollutants.
- Bacterial pathogens are responsible for 50-70\% of acute exacerbations.
  - Most common organisms: Haemophilus influenzae, Streptococcus pneumoniae, Moxarella catarrhalis.
  - Other pathogens: atypical bacteria Mycoplasma and Chlamydia pneumoniae, respiratory viruses rhinovirus, influenza, respiratory syncytial virus, parainfluenza virus and human metapneumovirus.
- Other precipitants are environmental pollutants: Smoke, particular matter, sulfur dioxide, nitrogen dioxide, ozone

Pathophysiology\textsuperscript{5,6,8,9}

Smoking:
- Smoke results in
  - Impaired integrity of normally tight junctions between epithelial cells of the lung
  - Inflammation including action of neutrophil elastase (protease which digests CT)
- Results in
  - Destruction of the alveolar walls
  - Loss of alveolar surface area for gas exchange and decreased elastic recoil
  - Increased tendency of the airways to collapse in expiration
  - Air outflow limitation and hyperinflation and dyspnoea.
- Smoking typically displays centrilobular emphysema
  - Most affects respiratory bronchioles and alveolar ducts
  - Typically seen in the upper lobes of the lung.

Alpha1-antitrypsin deficiency:
- Alpha1-antitrypsin is a protease inhibitor
- Genetic deficiency in alpha1-antitrypsin protease inhibitor
- Unchecked action of proteases on connective tissue
- Alveolar destruction
- Alpha1-antitrypsin deficiency typically displays panacinar emphysema
  - Destruction through the acinus
  - Seen typically in the lower lobes of the lung.

Acute exacerbation:
- “Acute on chronic” inflammatory response
- Increases in airway inflammatory cells and proteins
- Exacerbated obstructive defect and airflow limitation to expiration
- Worsening of dyspnoea, cough, sputum production beyond normal baseline
- Acute exacerbations become more frequent and severe as COPD progresses and may of themselves accelerate COPD progression.

Complications\textsuperscript{5,6,8,9}

Pulmonary hypertension
- Blood vessel constriction from hypoxia
- Blood vessel loss from alveolar destruction

Cor pulmonale
- Right heart failure from pulmonary hypertension

Abnormal ventilatory response
- CO2 retention results in blunted response to hypercapnia
- Switch to hypoxia driven respiratory drive.

CXR: Emphysema
- Hyperinflation
- Reduced vascular markings
- Prominent pulmonary vessels (pulmonary hypertension)

Epidemiology\textsuperscript{10}

Prevalence of COPD:
- 2.9\% of population (591,000) in 2004-05
- F (1.6\%) >M (1.3\%) in 2004-05
- M > F in >85 age group due to dramatic increase in male prevalence rates >75 years
- Disease of older age groups

Mortality from COPD:
- Mortality from COPD most marked >75 years
- 4\% (4,761) deaths in 2006

Morbidity from COPD:
- 34\% of patients with COPD reported some disability due to the condition in 2003

Services use from COPD:
- <1\% of GP encounters for COPD in 2007-08
- 52,560 hospitalisations for COPD in 2006-07
**Chronic bronchitis**

**Definition** A clinical diagnosis of daily sputum production for three months of the year for two consecutive years. A subtype of chronic obstructive pulmonary disease (COPD).

**Presentation**

*Typical presentation:*
- Chronic loose cough
- Chronic sputum production (mucoid or muco-purulent)
- Dyspnoea
- Wheeze
- No haemoptysis
- History of recurrent respiratory infection

**History of presentation complaint:**
- Dyspnoea: ask about exertion required to precipitate dyspnoea, rate on NYHA scale
- Cough: ask about onset and duration, character
- Sputum production: ask about frequency, volume, character, colour, smell
- Impact on function: ask about mobility, communication, activities of daily living, occupational

**Acute exacerbation:**
- Ask about recent changes in symptoms from normal day-to-day symptoms
- Ask about any identifiable precipitants (exposure to illness, environmental exposures, etc.)

**Respiratory history**
- Smoking history: age of initiation, amount, high risk if heavy smoker especially if >70 pack years
- Exposures: dusts, chemicals, air pollution
- Past medical history: respiratory infections
- Family history: COPD, other respiratory diseases

**Investigations**

*Lung function tests:*
- Obstructive defect:
  - FEV1: <80% of predicted
  - FEV1/FVC: 0.7
  - Irreversible (some reversibility may be present on bronchodilator test)
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*ABGs:*
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*V/Q Scan:*
- V/Q mismatch
  - High V/Q (ventilatory compensation) undamaged lung

**Examination**

*“Blue bloaters”*

**Inspection:**
- Cyanosis
- Oedema (from right ventricular failure)
- No clubbing

**Palpation:**
- Reduced chest expansion

**Percussion:**
- Hyperresonant percussion note

**Auscultation:**
- Reduced breath sounds/air entry
- End expiratory or low pitched wheeze
- Early inspiratory crackles

**Acute exacerbation may show:**
- Fever
- Tachypnoea
- Cough
- Sputum production (change in volume/character)

**In pre-terminal state may have signs of right heart failure:**
- Elevated JVP
- Peripheral oedema
- Increased P2, splitting of S2
- Hepatosplenomegaly

**Management**

*Pharmacotherapy:*
- Long acting B2 agonists (e.g. salmeterol, eformoterol)
- Inhaled anticholinergics (e.g. tiotropium bromide)
- Combination inhalers (ICS and LABA, e.g. salmeterol/beclometasone, eformoterol/fluticasone)
- Theophylline (very occasionally used)

*Steroids:*
- 2 week high oral steroid trial to assess reversibility.
- >15% in FEV1 indicates clinical benefit
- Avoid long term steroid use

*Non-invasive positive pressure ventilation (PPV)*

*Pulmonary rehabilitation program*
- 6 week exercise training and education

*Home oxygen therapy*
- Oxygen must be given with care in hypoxia as CO2 retention results in insensitivity of respiratory centres to CO2 = dependence on hypoxia for respiratory drive.
- Supplementary oxygen may therefore result in suppression of respiratory drive and respiratory failure

*Reduction of risk factors:*
- Smoking cessation, exercise, nutrition, obesity

*Preventative medicine:*
- Influenza vaccination, pneumococcal vaccination

*Acute exacerbations:*
- Antibiotics if infective exacerbation
- May require hospitalization
### Aetiology

#### Development of COPD
- A complex process that is not completely understood but is thought to be multifactorial (genetic, biological, behavioural).
- Smoking:
  - Most common cause of COPD in developed world is exposure to tobacco smoke. 50% of chronic smokers develop COPD and almost all COPD patients have significant smoke exposure recorded.
- Environmental exposures:
  - Occupational dusts, chemicals, air pollution
- Recurrent bronchial infection

#### Exacerbations of COPD
- An acute exacerbation is diagnosed on signs and symptoms and may be supported by spirometry showing decreases in FEV1, FVC and PEF due at least in part to airway inflammation.
- Triggered primarily by infection (viral and bacterial) and airborne pollutants.
- Bacterial pathogens are responsible for 50-70% of acute exacerbations.
  - Most common organisms: Haemophilus influenzae, Streptococcus pneumoniae, Moxarella catarrhalis.
  - Other pathogens: atypical bacteria Mycoplasma and Chlamydia pneumoniae, respiratory viruses rhinovirus, influenza, respiratory syncytial virus, parainfluenza virus and human metapneumovirus.
- Other precipitants are environmental pollutants: Smoke, particular matter, sulfur dioxide, nitrogen dioxide, ozone

### Pathophysiology

#### Chronic bronchitis:
- Hypertrophy and hyperplasia of airway mucous glands and increased numbers of goblet cells and hypersecretion of mucous into the bronchial tree
  - Cough and excessive sputum production
  - Mucous plugging in the lumen of the airways
- Chronic mucosal and submucosal inflammation
  - Smooth muscle hypertrophy
- Airway obstruction
  - Obstructive defect: ↓FEV1 and ↓FEV1/FVC
- Loss of ventilation in regions distal to the obstruction
  - Dyspnoea
- Decreased mucociliary clearance
  - Increased risk for pathogens to stimulate the lower respiratory tract
  - Increased risk of infection
- Infection further initiates inflammation
  - Inflammatory oedema
  - Mucous gland activity
  - Exacerbates obstructive defect and baseline symptomatology.

#### Acute exacerbation:
- “Acute on chronic” inflammatory response
- Increases in airway inflammatory cells and proteins
- Exacerbated obstructive defect and airflow limitation to expiration.
- Worsening of dyspnoea, cough, sputum production beyond normal baseline symptomatology.
- Acute exacerbations become more frequent and severe as COPD progresses and may of themselves accelerate COPD progression.
Asthma

**Definition**

A disease characterized by recurrent episodes of reversible airway obstruction due to bronchial hyperresponsiveness to stimuli, and contributed to by underlying chronic processes of mucosal inflammation and excess mucous production.

**History**

Typical history:
- Intermittent episodes of dyspnoea, wheeze, chest tightness, cough, sputum production, nocturnal waking

Time course
- Episodic with duration minutes-hours

Relieving factors
- Rest, removing self from situation
- Use of bronchodilators

Exacerbating factors
- Symptoms in relation to work (ask about exposure to allergens, chemicals, ask if symptoms better at weekends or holidays)
- Symptoms in relation to home (ask about carpets, pets, dust, feather pillows, clutter)

Precipitating factors
- Cold air, exercise, emotion, allergens (dust mites, pollen, animals), infection, smoking, pollution, NSAIDs, beta-blockers

Character
- Diurnal variation (decreased peak flow in morning which can precipitate attack despite normal peak flow at other times of day)

Functional capacity
- Exercise tolerance, disturbed sleep (nights per week), days off per week from work or school

Past medical history
- Atopic disease (eczema, hay fever, allergies)

Medication history and adherence
- Asthma drugs, NSAIDs, beta-blockers

Family history
- Asthma, other atopic disease

Smoking history

**Examination**

**Signs of asthma attack**
- Wheezing
- Dry or productive cough
- Tachypnoea, tachycardia
- Use of accessory muscles of expiration (rectus abdominus, external obliques, internal obliques)
- Hyperinflated chest (increased AP diameter, high shoulders, decreased liver dullness)
- Inspiratory and expiratory wheeze
- Decreased chest wall movement symmetrically
- Hyperresonance on percussion
- Reduced air entry
- Added sound wheeze

Bedside spirometry:
- Prolonged expiration (↓PEFR, ↓FEV1)

**Additional signs of severe asthma attack**
- Inability to speak due to dyspnoea, drowsiness (hypercapnia), cyanosis, tachycardia (>130bpm), pulsus paradoxus (>20mmHg), tachypnoea (>25breaths/min) reduced breath sounds

**Differentials of acute asthma attack**
- Pulmonary oedema (“cardiac asthma”)
- Bronchitis
- Pulmonary embolism
- Upper airway obstruction
- Pneumonia
- COPD
- Pneumothorax

**Management**

**Acute management of asthma attack**
- Oxygen (elevated CO2 means severe disease requiring intubation)
- SABA
- Evaluate if adrenalin is indicated
- Initiate treatment with other agents as indicated by response to initial treatment and severity

**Long term management of asthma**

Asthma Action Plan (individualized treatment algorithm)

Classes of medications:
- Relievers: direct bronchodilators taken for relief of acute attack
  - Short acting beta-2 agonists (SABA)
  - Long acting beta-2 agonists (LABA)
- Preventers: anti-inflammatories taken regularly to reduce symptoms and prevent exacerbations
  - Inhaled glucocorticoids (ICS)
  - Leukotriene receptor antagonists (LTRA)
  - Cromones
  - Anti-IgE
**Aetiology**

**Risk factors**
- Atopy (strongest risk factor, genetic predisposition to develop IgE-mediated response to aeroallergens, as indicated by positive skin prick test)
- Wheezing before 3 years of age
- Allergic rhinitis
- Environmental tobacco exposure
- Residential exposure (pets, gas cooking, damp housing, mold exposure)
- Perinatal risk factors (preterm delivery, maternal smoking, antenatal chemical exposure)
- Occupational risk factors (cleaning, farming)
- Respiratory infections before 1 year of age (pneumonia, RSV, otitis media, croup)
- Medications (acetaminophen, aspirin, oestrogen, beta-blockers)
- Genetic (possible associations with genes encoding GPRA protein, PTGDR receptor, ADAM33, CH13L1, CHIT1)

**Pathophysiology**

**Acute asthma**
Constriction of smooth muscles of bronchioles causing obstruction and acute difficulty in breathing.
Bronchoconstriction caused by hypersensitivity to stimuli (younger people typically exhibit allergic hypersensitivity to pollen, etc where older people have nonallergic hypersensitivity such as irritants in pollution)

Allergic asthma
- Atopic individual has tendency to form excessive IgE antibodies which are attached to mast cells of lung near bronchioles and small bronchi to cause allergic reaction upon interaction with antigen
- Exposure to antigen results in IgE cross-linking resulting in mast cell activation and degranulation
- Degranulation releases histamine, leukotrienes, eosinophilic chemotactic factor and bradykinin
- Pro-inflammatory markers produces oedema in small bronchiolar walls, mucous secretion into lumen of bronchioles and contraction of bronchiole smooth muscle combining to cause airway resistance.
Bronchioles have the tendency to dilate in inspiration and collapse in expiration. The reduced bronchiolar diameter in asthma thus results in further bronchiolar occlusion in expiration which results in
- Decreased PEFR and FEV1

**Chronic asthma**
Persistent changes from asthma can include
- Accumulation of leukotrienes and prostaglandins
- Over time, smooth muscle hypertrrophy and hyperplasia
- Vascular congestion and oedema
- Mucous gland hyperplasia and hypersecretion
- Epithelial cell injury
- Accumulation of mucous (mucous gland hyperplasia)
- Angiogenesis
- Sub-basement fibrosis

Results in persistent chronic inflammatory state and hyperproduction of mucous.

**Natural history of asthma**
- Most childhood asthmatics either remit or greatly improve in adulthood.
- Some childhood asthma will progress to chronic asthma in adulthood.

**Classification of asthma**

<table>
<thead>
<tr>
<th>Daytime asthma symptoms</th>
<th>Night-time asthma symptoms</th>
<th>Exacerbations</th>
<th>Spirometry</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intermittent</td>
<td>Less than weekly</td>
<td>Infrequent</td>
<td>FEV1 ≤ 80% predicted</td>
</tr>
<tr>
<td>Mild persistent</td>
<td>More than weekly and less than daily</td>
<td>Occasional</td>
<td>FEV1 ≤ 80% predicted</td>
</tr>
<tr>
<td>Moderate persistent</td>
<td>More than weekly and more than daily</td>
<td>May affect</td>
<td>FEV1 ≤ 80% predicted</td>
</tr>
<tr>
<td>Severe persistent</td>
<td>Daily</td>
<td>Frequent</td>
<td>FEV1 ≤ 80% predicted</td>
</tr>
</tbody>
</table>

**Epidemiology**

**Prevalence**
- 10.2% prevalence in Australia
- Most common reported long term condition in those aged 0-14 yrs.
- 12% prevalence in age group 0-14 yrs.
- 9% prevalence in age group ≥25 yrs
- M>F (13%>10%) prevalence 0-14 yrs
- F>M (12%>8%) prevalence ≥15 yrs
- 15% prevalence in Indigenous

**Mortality**
- 402 deaths attributable to asthma in 2006 (0.3% of deaths in 2006)

**Health service use**
- $606 million health expenditure 2004-5 (1.2% of all health care)
**Definition**
Pathological permanent dilatation and distortion of the bronchi resulting in impaired clearance of mucous characterised by chronic cough and persistent infection.

**Presentation**
Ask about history of:
- Chronic cough and purulent sputum (often since childhood, quantify amount when well vs. unwell)
- Severe bacterial infections (pneumonia, TB, pertussis, measles)
- History of recurrent infections (pneumonia, sinusitis)
- Past admissions to hospital
- Past medical history of respiratory conditions (cystic fibrosis, asthma, COPD)
- Medication use (bronchodilators, ICS, etc)

Ask specifically about symptoms of:
- Cough (chronic, productive)
- Sputum (voluminous, purulent, foul-smelling sputum)
- Haemoptysis
- Pleuritic chest pain
- Dyspnoea
- Systemic symptoms of infection: fever, LOW and LOA

Other
- Family history of respiratory tract disease
- Smoking history

Effects on function and activities of daily living

**Examination**

*Vital signs:*
- Fever

*General inspection:*
- Moist cough
- Sputum cup (voluminous, purulent, foul-smelling, blood)
- Cachexia

*Inspection:*
- Clubbing
- Cyanosis (if severe disease)

*Auscultation:*
- Coarse late inspiratory or pan-inspiratory crackles (localized or diffuse)
- Wheeze

If very severe, clinical signs of cor pulmonale may be present.

**Investigations**

*CXR*
- Cystic shadows (dilated bronchi)
- Thickened bronchial walls (tram-tracking)

*Sputum culture*
- Haemophilus influenzae
- Streptococcus pneumoniae
- Staphylococcus aureus
- Pseudomonas aeruginosa

*Bronchoscopy*
- Tumour, foreign body, bronchial stenosis

*spirometry*
- Obstructive pattern (common)
  - FEV1 <80% of predicted, FEV1/FVC: 0.7
  - Assess reversibility (bronchodilator test)
- Restrictive
  - ↓FEV1, ↓FVC, normal FEV1/FVC

*CT chest*
- To confirm diagnosis and extent of disease
- Tests to confirm aetiology
- Genetic studies for CF

**Differentials**

| Chronic bronchitis | Acute bronchitis |

**Management**

*Antibiotics*
- Sensitivities as taken from sputum culture.

*Bronchodilators and ICS*
- If co-existing COPD, asthma, CF
- If reversible component identified in spirometry
- Inhaled mannitol

*Chest physiotherapy*
- Postural drainage (aid mucous drainage and sputum expectoration)
- Pulmonary rehabilitation (improve exercise tolerance)

*Surgical excision*
- If localised disease or severe haemoptysis (embolization)

*Prophylaxis*
- Longterm antibiotics have little proven efficacy
- Predisposes to individual and population resistance and other side effects such as antibiotic associated diarrhea, etc.

*Vaccinations*
- Yearly influenza, pneumococcal, Haemophilus
- Cessation of smoking

*Management of any associated complications*

**CXR:** Central bronchiectasis from allergic bronchopulmonary aspergillosis

*CT:** Bronchiectasis

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*References*

PA view showing dilation and thickening of airways RUL (arrow). Cellular debris and mucous seen in airways of LUL.

Typical “tree in bud” linear branch markings of small airways (A) and dilated and thickened airways (B).
Aetiology\textsuperscript{5,6,10}

**Causes:**
- Congenital
  - Cystic fibrosis, Young’s syndrome, primary ciliary dyskinesia, Kartagener’s syndrome.
- Post-infection
  - Measles, pertussis, bronchiolitis, pneumonia, tuberculosis, HIV.
- Other
  - Bronchial obstruction (retained foreign body, tumour, anatomical obstruction, recurrent aspiration), immune deficiency, allergic bronchopulmonary aspergillosis, hypogammaglobulinaemia, rheumatoid arthritis, ulcerative colitis
  - Idiopathic.

**Risk factors:**
- Congenital cystic disease of the lung
- Bronchial stenosis (tracheobronchomalacia)
- Compression of bronchi
- Subglottic hemangioma

**Associated conditions:**
- Congenital conditions
  - Marfan’s syndrome, pulmonary sequestration, cartilage deficiency, tracheobronchomegaly, cystic fibrosis, primary ciliary dyskinesia
- Post-infectious
  - Pseudomonas aeruginosa, Haemophilus influenzae, Mycobacterium tuberculosis, Aspergillus, measles virus, influenza virus, adenovirus, HIV.
- Sequelae of toxic aspiration
  - Chlorine, foreign body, heroin overdose
- Rheumatic conditions
  - SLE, rheumatoid arthritis, Sjogren’s syndrome, relapsing polychondritis
- Immunodeficiency
  - Hypogammaglobulinemia, chemotherapy, malignancy, immune modulation
- Other
  - Inflammatory bowel disease, Young’s syndrome (secondary ciliary dyskinesia), yellow nail syndrome

Pathophysiology\textsuperscript{5,6,15}

- Chronic, recurrent or severe infection in the airways
- Destruction of bronchial wall → bronchial dilatation and impaired mucociliary function.
- Impaired clearance of secretions → accumulate and predispose to bacterial infection.
- Inflammation from infection → further mucous production and damage to bronchial walls → self-propagating cycle.

**Causes of trauma to the airway**
- Chronic or recurrent infection in the airways (especially childhood)
- Severe infection (pneumonia, tuberculosis, pertussis, measles), in particular suppurative infection in an obstructed bronchus.
- Tumour, foreign body in airway
- Congenital

**Complications**\textsuperscript{6,10}
- Pneumonia
- Pleural effusion
- Pneumothorax
- Haemoptysis
- Cerebral abscess
- Amyloidosis

Epidemiology\textsuperscript{10,15}

- Not usually a primary condition but a consequence of other respiratory disease
- More common in F>M
- More common in older age groups
- More common in Indigenous Australians (14/1,000 Indigenous children)
- Few deaths directly attributed to bronchiectasis (80 males and 153 females in 2006)
- More deaths with bronchiectasis as an associated cause (120 males and 188 females in 2006)
- 80% of deaths from bronchiectasis >70 years, average age 77 years

Mnemonic for aetiology: “BRONCHIECTASIS”
- Bronchial cyst
- Repeated gastric acid aspiration
- Or due to foreign bodies
- Necrotizing pneumonia
- Chemical corrosive substances
- Hypogammaglobulinemia
- Immotile cilia syndrome
- Eosinophilia (pulmonary)
- Cystic fibrosis
- Tuberculosis (primary)
- Atopic bronchial asthma
- Streptococcal pneumonia
- In Young’s syndrome
- Staphylococcal pneumonia
### Pneumonia

**Definition**
A lower respiratory tract infection characterized by inflammation of the lung and exudation into the alveoli and manifested clinically with systemic and respiratory signs and symptoms and radiological changes on chest x-ray.

### Classifications/subtypes

- **By setting:**
  - Community acquired (presents in community) or nosocomial (presents >48 hours after admission to hospital)
- **By host:**
  - Normal immunity vs. immunocompromised, normal lung vs. abnormal lung (COPD, bronchiectasis, etc)
- **By anatomy:**
  - Lobar pneumonia, segmental pneumonia or lobular pneumonia
- **By organism:**
  - Typical (S. pneumoniae, H influenzae, S. aureus, GAS, Moraxella catarrhalis, anaerobes, aerobic GNB)
  - Atypical (Legionella, M. pneumoniae, C. pneumoanae, C psitataci)
- **Other:**
  - Aspiration pneumonia (high risk in those with stroke, myasthenia, bulbar palsies, decreased consciousness - drunk, post-ictal), oesophageal disease (achalasia, reflux)

### Presentation

**Typical symptoms (sudden onset - days):**
- Fevers and rigors
- Malaise
- Anorexia
- Dyspnoea
- Cough
- Sputum production
- Haemoptysis
- Pleuritic chest pain

**Obtain history:**
- Past respiratory history (underlying CF, COPD, etc.)
- Past medical history (immunocompromisation, Haemophilus and Pneumococcus immunization history if elderly)
- Medications and allergies
- Smoking history
- Recent travel (unexpected pathogens)
- Recent exposure to illness

### Examination

**Vital signs:**
- Fever
- Tachypnea
- Tachycardia
- Hypotension

**Inspection:**
- Cyanosis
- Confusion or altered mental state (elderly)

**Palpation:**
- Increased tactile fremitus
- Reduced chest expansion

**Percussion:**
- Dull percussion note

**Auscultation:**
- Bronchial breathing
- Medium, late or pan-inspiratory crackles
- Pleural rub
- Increased vocal fremitus

### Investigations

**CXR**
- Consolidation (radiopaque density, typically sharply demarcated in lobar pneumonia)
  - RML pneumonia (loss of R cardiac border)
  - RLL pneumonia (loss of R hemidiaphragm)
  - LLL pneumonia (loss of L hemidiaphragm)
- Interstitial infiltrates (poorly defined opacities)
- Cavitations (radiolucent shadow)

**Negative CXR** may be present in:
- Initial stages of infection
- PCP
- Neutropenia
- Dehydration

**Bloods (FBC, U&E, LFTs, CRP)**
- Raised WCC, raised CRP may be seen

**Blood culture**
- MC&S (5-10% yield)
- IgM/IgG serology for Mycoplasma, Legionella, Chlamydia

**Sputum culture**
- MC&S (40% yield)

**Urinary antigen**
- Pneumococcal, Legionella

**ABGs**
- Indicated if oxygen saturation <92%

**Bronchoscopy and bronchoalveolar lavage (BAL)**
- If patient is immunocompromised, high risk or unresolving

### Management

**Supportive therapy**
- Oxygen (maintain oxygen saturation ≥94%)
- IV fluids (as required)
- Analgesia (as required)

**Antibiotics**

**Empirical therapy:**
- Streptococcus pneumonia, Haemophilus influenzae, Mycoplasma pneumoniae
  - Amoxicillin, Clarithromycin
- Legionella
  - Amoxicillin, Clarithromycin and Rifampicin
- PCP
  - Co-trimoxazole
- Pseudomonas
  - Anti-pseudomonal penicillin (Ticarcillin or Piperacillin) or 3rd generation cephalosporin

**Targeted therapy:**
- According to culture and sensitivities

**Prevention:**
- High risk patients (elderly, immunocompromised, respiratory disease)
- Vaccines:
  - Influenza, Pneumococcus, Haemophilus
Aetiology

Causes:

<table>
<thead>
<tr>
<th>Community acquired</th>
<th>Nosocomial</th>
<th>Immunocompromised</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacteria</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Most common</td>
<td>Most common</td>
<td></td>
</tr>
<tr>
<td>• Streptococcus pneumonia</td>
<td>• Gram negative enterobacteriaceae</td>
<td></td>
</tr>
<tr>
<td>• Haemophilus influenza</td>
<td>• Staphylococcus aureus</td>
<td></td>
</tr>
<tr>
<td>• Mycoplasma pneumonia</td>
<td>Others</td>
<td></td>
</tr>
<tr>
<td>• Staphylococcus aureus</td>
<td>• Pseudomonas</td>
<td></td>
</tr>
<tr>
<td>• Legionella</td>
<td>• Klebsiella</td>
<td></td>
</tr>
<tr>
<td>• Moraxella catarrhalis</td>
<td>• Bacteroides</td>
<td></td>
</tr>
<tr>
<td>• Chlamyphila</td>
<td>• Clostridia</td>
<td></td>
</tr>
<tr>
<td>Rare</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Gram negative bacilli</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Coxiella burnetti</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Anaerobes</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| Viruses              |            |                   |
|• Influenza           |            |                   |
|• Parainfluenza       |            |                   |
|• Adenovirus          |            |                   |

| Fungi                |            |                   |
|• CMV                 |            |                   |
|• HSV                 |            |                   |

|• Pneumocystis jeroveci (formerly carinii) |            |                   |

Epidemiology

Mortality
- 2% (2,715) of deaths in Australia from influenza or pneumonia in 2006
- 14.1% death rate in males
- 10.2% death rate in females
- Death most likely in COPD and elderly

Hospitalizations
- >50 years age group showed highest rate of hospitalizations.
- Rise in pneumonia hospitalizations in seasonal flu period (late autumn to late spring)

Pneumonia severity index (PSI)
Online access: http://www.debug.net.au/pharmacy/calculator.html
Divides patients into classes:
- I: oral antibiotics, outpatient
- II-III: IV antibiotics, outpatient or 24 hour admission
- IV-V: antibiotics inpatient, may require ICU

CURB-65
Confusion (abbreviated mental test ≤8)
Urea (>7mmol/L)
Respiratory rate (≥30 breaths/min)
Blood pressure <90mmHg systolic and/or <60mmHg diastolic
65 years or older

Scoring
- 0-1: outpatient,
- 2: hospital admission,
- 3-5: hospital admission, consider ICU

Pathophysiology

Lower respiratory tract is sterile despite day to day exposure to pathogens and particulate matter due to
- Innate (non-specific) immune function
- Acquired (specific) immune function

Pneumonia occurs when the virulence of an organism is able to overcome the host immune system due to
- Host factors (e.g. immunocompromisation)
- Pathogen factors (e.g. high virulence factors).

Transmission to lung
- Microaspiration (most common)
- Haematogenous spread
- Direct local spread
- Macroaspiration

Differentials
Infectious:
- URTI, sinusitis, pharyngitis, acute bronchitis
Non-infectious:
- Pulmonary embolism, chronic HF, bronchial carcinoma, inflammatory lung disease

Unresolving pneumonia
Pneumonia should improve within 24 hours of Rx
- Subjectively “feeling better”, resolving fever
If no improvement, consider
- Wrong antibiotic (e.g. different organism, poor compliance, poor absorption)
- Wrong diagnosis (e.g. cancer, pulmonary embolism)
- Complication (e.g. empyema)
**Pleural effusion**

**Definition**
A collection of fluid in the pleural space (between the parietal and visceral pleura). Fluid may consist of blood (haemothorax), lymph (chylothorax) or pus (empyema).

<table>
<thead>
<tr>
<th>Classification</th>
<th>Transudate: &lt;30g protein per litre of fluid</th>
<th>Exudate: &gt;30g protein per litre of fluid</th>
</tr>
</thead>
</table>

**Presentation**
- Often asymptomatic
- If symptomatic:
  - Dyspnoea
  - Pleuritic chest pain

Associated clinical features of pleural effusion are important in determining likely aetiology:
- Cough, sputum production
- Haemoptysis
- Fever
- Night sweats, weight loss (signs of malignancy)

Take a full respiratory history considering possible risk factors for pleural effusion:
- Occupational exposures
- Smoking
- Personal history of malignancy
- Family history of malignancy
- Medication history

**Examination**
- Trachea displaced away from effusion
- Apex beat displaced away from effusion
- Reduced chest expansion on affected side
- Stony dull percussion note over fluid
- Reduced or absent breath sounds.
- Bronchial breath sounds may be present above the level of the effusion (compression of lung)
- Pleural friction rub
- Reduced vocal resonance

**Investigations**

**CXR**
- Blunt costophrenic angle (loss of adjacent aerated lung for contrast)
- Water dense shadows with curved concave upper border (“meniscus sign”)
- Trachea and heart border may be deviated away from effusion

**Pleural fluid aspiration (thoracentesis)**
- Diagnostic (may also be therapeutic)
- Performed under ultrasound guidance
- Needle with syringe inserted 1-2 intercostal spaces below upper border of pleural effusion (as percussed)

**MC&S**
- Pleural fluid sent to laboratory for:
  - Clinical chemistry (protein, glucose, pH, LDH, amylase)
  - Bacteriology (microscopy, culture, staining)
  - Cytology
  - Immunology (rheumatoid factor, ANA, complement)

**Pleural biopsy**
- If inconclusive pleural fluid analysis
- Performed under CT or thorascopic guidance

**Classification**

**Transudate:**
- <30g protein per litre of fluid

**Exudate:**
- >30g protein per litre of fluid

<table>
<thead>
<tr>
<th>Colour of fluid</th>
<th>Palae yellow (straw)</th>
<th>Transudate, some exudates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red (bloody)</td>
<td>Malignancy, benign asbestos pleural effusion, postcardiac injury syndrome, pulmonary infarction in absence of trauma</td>
<td></td>
</tr>
<tr>
<td>White (milky)</td>
<td>Chylothorax, cholesterol effusion</td>
<td></td>
</tr>
<tr>
<td>Brown</td>
<td>Long-standing blood effusion, rupture of amoebic liver abscess</td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>Aspergillosis</td>
<td></td>
</tr>
<tr>
<td>Yellow-green</td>
<td>Rheumatoid pleurisy</td>
<td></td>
</tr>
<tr>
<td>Dark green</td>
<td>Biliothorax</td>
<td></td>
</tr>
</tbody>
</table>

**Colour of enteral tube feeding**
- Feeding tube has entered pleural space

**Colour of central venous catheter infusate**
- Extravascular catheter migration

**Character of fluid**
- Pus
- Empyema
- Viscous
- Mesothelioma
- Debris
- Rheumatoid pleurisy
- Turbid
- Inflammatory exudate, lipid effusion
- Anchovy paste
- Amoebic liver abscess

**Odour of fluid**
- Putrid
- Anaerobic empyema
- Ammonia
- Urinothorax

**Pleural fluid analysis**

**Normal pleural fluid**
- pH 7.60-7.64
- <1000 WBC/mm³
- LDH <50% of plasma
- Glucose similar to that of plasma

**Diagnostic yield from pleural fluid analysis**

<table>
<thead>
<tr>
<th>Empyema</th>
<th>Observation (pus, putrid odour); culture</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malignancy</td>
<td>Positive cytology</td>
</tr>
<tr>
<td>Lupus pleuritis</td>
<td>LE cells present; pleural fluid serum ANA &gt;1.0</td>
</tr>
<tr>
<td>Tuberculosis pleurisy</td>
<td>Positive AFB stain, culture</td>
</tr>
<tr>
<td>Oesophageal rupture</td>
<td>High salivary amylase, pleural fluid acidosis (can be as low as 6.0)</td>
</tr>
<tr>
<td>Fungal pleurisy</td>
<td>Positive KOH stain, culture</td>
</tr>
<tr>
<td>Chylorrax</td>
<td>Triglycerides (&gt;100mg/dL); lipoprotein electrophoresis (chylomicrons)</td>
</tr>
<tr>
<td>Haemothorax</td>
<td>Haematocrit (pleural fluid/blood &gt;0.5)</td>
</tr>
<tr>
<td>Urinothorax</td>
<td>Creatinine (pleural fluid/serum &gt;1.0)</td>
</tr>
<tr>
<td>Peritoneal dialysis</td>
<td>Protein (&lt;1g/dL); glucose (300-400mg/dL)</td>
</tr>
<tr>
<td>Extravascular migration of central venous catheter</td>
<td>Observation (milky if lipid infusion); pleural fluid/serum glucose &gt;1.0</td>
</tr>
<tr>
<td>Rheumatoid pleurisy</td>
<td>Characteristic cytology</td>
</tr>
</tbody>
</table>

**Pleural fluid aspiration (thoracentesis)**

**Diagnosis**

- Observation (pus, putrid odour); culture
- Positive cytology
- LE cells present; pleural fluid serum ANA >1.0
- Positive AFB stain, culture
- High salivary amylase, pleural fluid acidosis (can be as low as 6.0)
- Positive KOH stain, culture
- Triglycerides (>100mg/dL); lipoprotein electrophoresis (chylomicrons)
- Haematocrit (pleural fluid/blood >0.5)
- Creatinine (pleural fluid/serum >1.0)
- Protein (<1g/dL); glucose (300-400mg/dL)
- Observation (milky if lipid infusion); pleural fluid/serum glucose >1.0
- Characteristic cytology
Management

Therapeutic drainage
- Pleural fluid aspiration under U/S guidance
- Intercostal drain (alternative)
- Repeated drainage may be necessary

Pleurodesis
- Indicated in recurrent effusions
- Obliteration of pleural space by adhesion of pleural surfaces
- Chemical pleurodesis
  - Talc, tetracyclines or bleomycin
- Surgical pleurodesis
  - If persistent collections
- Pleural catheter (tunneled)

Aetiology

Pleural effusion is a clinical manifestation that is indicative of underlying disease.

Causes of transudate (<30g protein/L) pleural effusion:
- Increased venous pressure (cardiac failure, fluid overload, constrictive pericarditis)
- Hypoproteinaemia (nephrotic syndrome, chronic liver disease, malabsorption)
- Hypothyroidism
- Meigs syndrome (ovarian fibroma which causes pleural effusion and ascites)

Causes of exudate (>30g protein/L) pleural effusion:
- Pneumonia
- Malignancy (bronchial carcinoma, metastatic carcinoma, mesothelioma)
- Tuberculosis
- Pulmonary infarction
- Subphrenic abscess
- Acute pancreatitis
- Connective tissue disorders (rheumatoid arthritis, SLE)
- Drugs (methysergide, cytotoxics)
- Irradiation
- Trauma

Causes of haemotherax:
- Chest trauma
- Rupture of pleural adhesion with blood vessel

Causes of chylothorax:
- Trauma to thoracic duct
- Surgical instrumentation of thoracic duct
- Carcinoma or lymphoma of thoracic duct

Causes of empyema:
- Pneumonia
- Lung abscess
- Bronchiectasis
- Tuberculosis
- Penetrating chest trauma

Pathophysiology

A small amount of fluid (0.13ml/kg of body mass) is usually present in the pleural space to allow frictionless movement of two pleural surfaces (visceral and parietal) against each other during respiration. This volume is maintained through the balance of oncotic and hydrostatic pressures and lymphatic drainage.

The interplay of several mechanisms can result in the formation of a pleural effusion:
- Change in permeability of pleura
- ↓ intravascular oncotic pressure (e.g. hypoproteinaemia)
- ↑ permeability of capillaries or disruption in vascular integrity of capillaries
- ↑ hydrostatic pressure in capillaries of systemic or pulmonary circulation
- ↓ pressure in pleural space resulting in decreased expansion
- ↓ or absent lymphatic drainage due to obstruction or rupture of vessel, typically of thoracic duct
- ↑ amount and migration of peritoneal fluid across the diaphragm
- Presence of pulmonary oedema resulting in migration of fluid across visceral pleura
- ↑ pleural fluid oncotic pressure

This can result in increased pleural fluid formation and/or decreased pleural fluid clearance resulting in collection of fluid in the pleural space and pleural effusion.
### Pulmonary embolism

<table>
<thead>
<tr>
<th>Definition</th>
<th>Pulmonary embolism is the obstruction of a pulmonary artery or one of its branches by a material that has originated from elsewhere in the body. Emboli are most commonly formed by blood clots, but may also be due to fat, air or amniotic fluid embolism.</th>
</tr>
</thead>
</table>

### Classification

**Acute** (patient develops clinical features immediately following obstruction of pulmonary vessel) or **chronic** (patient develops clinical features, typically progressive dyspnoea, over years). Acute pulmonary embolism can be subclassified as **massive** (causing hypotension <90mmHg systolic or <40mmHg diastolic for >15 minutes, medical emergency) or **sub-massive** (all other acute pulmonary embolisms not meeting criteria for massive acute pulmonary embolism).

### Presentation

Pulmonary embolism is often asymptomatic

Common symptoms reported:
- Dyspnoea (severe, sudden onset)
- Pleuritic chest pain
- Cough
- Wheezing
- Orthopnoea
- Calf or thigh pain
- Calf or thigh swelling
- Dizziness
- Syncope
- Haemoptysis

Note
- Pulmonary embolism is often asymptomatic
- Clinical features PE are **nonspecific**

Ask about
- Risk factors
- Past history of thromboembolism
- Family history of thromboembolism

### Examination

**Signs on general inspection**
- Tachycardia
- Tachypnoea
- Cyanosis
- Hypotension
- Fever (if infarction)

**Signs on auscultation**
- Decreased breath sounds
- Crackles (rales)
- Pleural friction rub (if infarction)

**Signs of massive pulmonary embolism**
- Elevated JVP
- Right ventricular gallop
- Right ventricular heave
- Tricuspid regurgitation murmur (pansystolic)
- Loud P2 in second heart sound

**Signs of deep vein thrombosis**
- Tenderness
- Oedema
- Erythema

### Investigations

Essential as PE cannot be diagnosed on Hx and Ex alone.

**Bloods**
- FBC, U&E, coagulation picture
- D-dimer
- High sensitivity (useful to rule out PE if negative)
- Low specificity (not useful to rule in PE even if positive)
- Detects fibrin degradation product in blood
- Positive in: PE, inflammation, thrombosis, post-op, infection, malignancy

**ABG**
- Hyperventilation (low PaO2 and PaCo2)

**CXR**
- May be normal
- May show oligaemia, dilated pulmonary vessels, linear atelectasis (collapse), pleural effusion, opacities (wedge-shaped, cavitation)

**ECG**
- May be normal
- Tachycardia (most common)
- RBBB
- Right ventricular strain
- Right axis deviation
- AF
- Classical “SI QII TIII” pattern (deep S waves in I, Q waves in III, inverted T waves in III)

**CT pulmonary angiography (CTPA)**
- High sensitivity and specificity

**V/Q scan**
- V/Q Mismatch
  - Decreased perfusion, normal ventilation
  - Useful only if previously normal lung (indeterminate in lung disease)

**Echocardiogram**
- Right heart strain

### Management

**Immediate management**
- Oxygen 100%
- Analgesia (morphine)
- Anti-emetic
- Establish IV access
- Assess haemodynamic stability
- Colloid infusion +/- adrenalin if hypotensive (systolic <90mmHg)

**Anti-coagulation**
- To prevent further blood clot
- Warfarin if systolic >90mmHg
- IV heparin (LMW or unfractioned), bolus first then infusion, infusion as guided by APTT

**Thrombolysis**
- Dissolve existing blood clot
- High risk patients (large or unstable PE)
- Streptokinase or recombinant tissue plasminogen activator (rTPA)

**Inferior vena cava filter**
- Limited indications
- Should be used in co-therapy with anti-coagulation

**Prevention**
- Early mobilization post-op
- TED stockings (anti-thromboembolic)
- LMW Heparin prophylaxis
- Anticoagulation if recurrent PE
### Aetiology

**Causes**
- Thrombus
  - Deep venous thrombosis (most common cause)
  - 50-80% from distal vein below the popliteal veins
  - Others from proximal iliac, femoral and popliteal veins
- Air
- Fat
- Amniotic fluid
- Malignant cells
- Parasites

**Risk factors**
- Deep vein thrombosis (50%)
- Immobilization (decreased mobility, bedbound, stroke, paresis, paralysis)
- Recent surgery (<3 months, particularly if abdominal or pelvic, hip or knee replacement)
- Thrombophilia
- Malignancy
- Recent central venous instrumentation (<3 months)
- Hormonal risk factors (pregnancy, post partum, oral contraceptive pill, hormone replacement therapy)
- Previous pulmonary embolism
- Chronic heart disease
- Obesity (BMI >29)
- Smoking (>25 cigarettes/day)
- Hypertension

### Pathophysiology

- Spontaneous emboli (most typically thrombus from the deep venous system of the lower limbs)
- Venous system → right heart → pulmonary vessels
- Large thrombi lodge at bifurcation of the pulmonary artery or its branches
- Results in obstruction to blood flow
- Infarction in 10% (especially patients with pre-existing respiratory disease)

**Symptoms**
- Pleuritic chest pain
  - Inflammatory response of parietal pleura to thrombus
- Dyspnoea
  - Atelectasis (pulmonary collapse) from obstruction by thrombus and release of inflammatory mediators
  - Impaired gas exchange from functional intrapulmonary shunting and changes in surfactant function
- Right ventricular failure
  - ↑ pulmonary pressure
- Hypotension
  - ↓ cardiac output due to increase pulmonary resistance = ↓ right ventricular outflow = ↓ left ventricular inflow.

### Epidemiology

**Incidence**
- Likely underestimated (commonly asymptomatic and undiagnosed)

**Mortality**
- 0.2% of all deaths in Australia in 2008 (ABS)
- Untreated mortality rate is 30%
- Treated mortality rate is 2-8%
- Recurrent embolism is most common cause of death

**Morbidity**
- Morbidity is common amongst survivors
- Pulmonary hypertension (acute PE)

### Differentials

- AMI
- Pneumonia
- Aortic dissection
- Pneumothorax
- Cardiac tamponade
- Septicaemia

### Mnemonics/extra notes

**Virchow’s triad**
- Factors contributing to venous thrombosis:
  1. Hypercoagulability (thrombophilia, hormonal factors)
  2. Haemodynamic changes (stasis, turbulence, other changes to blood flow)
  3. Endothelial injury or dysfunction (hypertension, etc)
Interstitial Lung Disease

**Definition**
A heterogenous group of diffuse parenchymal lung diseases that are classified together because of similar clinical, radiographic or pathological features.

**Classification**
Diffuse parenchymal lung diseases

**Diagnosis**
A heterogenous group of diffuse parenchymal lung diseases that are classified together because of similar clinical, radiographic or pathological features.

**Examinations**
- **Inspection**
  - Clubbing
- **Auscultation**
  - Fine late or pan-inspiratory crackles
- **Signs of RHF**
  - Advanced pulmonary fibrosis → pulmonary HTN → cor pulmonale
  - Accentuated P2, right sided heave, congestive hepatomegaly, ankle and sacral oedema, raised JVP
- **Associated signs**
  - May be present due to associated illness

**Epidemiology**
Prevalence and incidence
- Idiopathic pulmonary fibrosis and sarcoidosis are the most common ILDs

Mortality
- Lung diseases due to external agents accounted for 0.9% of deaths in Australia in 2008

**Investigations**
Bloods and serology
- Findings may include:
  - Leukopenia, leukocytosis, eosinophilia, thrombocytopenia, haemolytic anaemia, normocytic anaemia, hypercalcemia, elevated LDH hypogammaglobulinaemia, hypergammaglobulinaemia, anti-GBM antibody, RF, ANA

CXR
- Normal in 10% (false negative rate) of ILD diagnosed on biopsy
- Findings may include:
  - Reticular or reticulonodular infiltrates (nodular densities and shadowing), diminished lung volume, alveolar infiltrates, hilar and mediastinal lymphadenopathy, pneumothorax, pleural disease, miliary disease, honeycomb lung

Anatomical location may provide hint to aetiology:
- Upper zones: Sarcoidosis, silicosis, berylliosis, coal miner’s pneumoconiosis, histiocytosis X, chronic hypersensitivity pneumonitis, tuberculosis
- Lower zones: rheumatoid arthritis, asbestosis, scleroderma, radiation, drugs (busulphan, bleomycin, nitrofurantoin, methotrexate, amiodarone), idiopathic

HRCT (high resolution computerized tomography)
- Greater sensitivity and specificity
- Can help stage ILD
- Findings may include:
  - Air space opacities, reticular opacities, nodules, isolated lung cysts

**Lung function tests**
- Decreased lung volume (decreased IC, VC, TLC)
- Decreased DLCO
- Restrictive defect (decreased FEV1 and FVC, normal FEV1/FVC)
- Variable obstructive defect may be seen

**Lung biopsy**
- Gold standard diagnostic tool in ILD
- Types:
  - Transbronchial lung biopsy during bronchoscopy (sarcoid), thoracoscopic biopsy or open lung biopsy (IPF)
  - Bronchoscopy with bronchoalveolar lavage
  - May reveal
    - Infectious agents, antigens, antibodies, small molecules (dusts, particles), malignant cells, inflammatory cells (eosinophils, macrophages)
  - Inflammatory cell differential can suggest aetiology

**Differentials**
- Pneumonia CCF Asthma COPD
Management

Aetiology
- Identify aetiology through Ix and eliminate aetiology as appropriate (e.g., removal of agent in exposures, immunosuppressive therapy in autoimmune diseases)

Corticosteroids
- High dose and taper for response
- If unresponsive to aetiological removal/ if not possible
- Prevent progression
- Typically does not alter existing disease
- Decline in respiratory function in absence

Supplemental oxygen
Lung transplantation

Pathophysiology

Aetiological factors → chronic inflammation of the lung with polymorphonuclear leukocytes, B lymphocytes, T lymphocytes, macrophages → inflammatory damage to alveolar wall and surrounding structures → scarring and fibrosis → decreased viable lung for gas exchange, restriction of movement of the lung (restrictive defect) and decreased lung volumes and may result in a variable obstructive pattern.

Specific mechanism and pattern of defect depends on aetiology.

Algorithm to approach patient with interstitial lung disease

Aetiology

Primary diseases associated with ILD:

<table>
<thead>
<tr>
<th>Disease</th>
<th>Aetiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sarcoidosis</td>
<td>Amyloidosis</td>
</tr>
<tr>
<td>Pulmonary Langerhans cell histiocytosis</td>
<td>Vasculitides (Wegener’s granulomatosis, Churg-Strauss syndrome)</td>
</tr>
<tr>
<td>Lymphangio-leiomyomatosis</td>
<td>Neurofibromatosis</td>
</tr>
<tr>
<td>Chronic pulmonary oedema</td>
<td>Chronic uraemia</td>
</tr>
<tr>
<td>Alveolar proteinosis</td>
<td>Pulmonary veno-occlusive syndrome</td>
</tr>
<tr>
<td>Gaucher’s disease</td>
<td>Neimann-Pick disease</td>
</tr>
</tbody>
</table>

Occupational/environmental exposures associated with ILD:

<table>
<thead>
<tr>
<th>Silicates</th>
<th>Carbon</th>
<th>Metals</th>
<th>Organic inhaled agents</th>
<th>Other inhaled agents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Silica (silicosis)</td>
<td>Coal dust (coal worker’s pneumoconiosis)</td>
<td>Tin (stannosis)</td>
<td>Thermophilic fungi (Macropolyspora faenia, Thermactinomyces vulgaris, Thermactinomyces sacchari)</td>
<td>Synthetic fibres (orlon, polyester, nylon, acrylic)</td>
</tr>
<tr>
<td>Asbestos (asbestosis)</td>
<td>Graphite (carbon pneumoconiosis)</td>
<td>Aluminium</td>
<td>True fungi (Aspergillus, Cryptostroma corticale, Aureobasidium pullulans, Penicillium)</td>
<td>Vinyl and polivinyl chloride</td>
</tr>
<tr>
<td>Talc (talcosis)</td>
<td>Hard metal dusts</td>
<td>Bacteria (Bacillus subtilis, Bacillus cereus)</td>
<td>Gases (oxygen, nitrogen oxide, sulphur dioxide, chlorine, methyl isocyanate)</td>
<td></td>
</tr>
<tr>
<td>Beryllium (berylliosis)</td>
<td>Iron (“siderosis”, “arc welder’s lung”)</td>
<td>Animal proteins</td>
<td>Fumes (zinc, copper, manganese, cadmium, iron, magnesium, nickel, brass, selenium, tin, antimony oxides)</td>
<td></td>
</tr>
<tr>
<td>Barium (baritis)</td>
<td></td>
<td></td>
<td>Vapours (hydrocarbons, toluene disocyanate, mercury)</td>
<td></td>
</tr>
<tr>
<td>Antimony</td>
<td></td>
<td></td>
<td>Aerosols (oils, fats, pyrethrum)</td>
<td></td>
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<tr>
<td>Hematite (“sidersilicosis”)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed dusts of silver and iron oxide (“argyrosiderosis”)</td>
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</tbody>
</table>

Drugs associated with ILD:

<table>
<thead>
<tr>
<th>Antibiotics</th>
<th>Anti-inflammatories</th>
<th>Anti-arrhythmics</th>
<th>I illicit drugs</th>
<th>Chemotherapeutic agents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitrofurantoin</td>
<td>Gold</td>
<td>Tocainide</td>
<td>Heroin</td>
<td>Antibiotics (Bleomycin, Mitomycin C)</td>
</tr>
<tr>
<td>Sulfasalazine</td>
<td>Penicillamine</td>
<td>Amiodarone</td>
<td>Cocaine</td>
<td>Alkylating agents (Busulfan, Cyclophosphamide, Chlorambucil, Melphalan)</td>
</tr>
<tr>
<td>Minocycline</td>
<td>NSAIDs</td>
<td>Methadone</td>
<td>Anti-metabolites (Azathioprine, Cytoxine arabinoside, Nethotrexate)</td>
<td></td>
</tr>
<tr>
<td>Ethambutol</td>
<td>Lefluonomide</td>
<td>Hydrochloride</td>
<td>Eposide</td>
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<td></td>
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<td></td>
<td>Propoxyphene</td>
<td>Paclitaxel</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hydrochloride</td>
<td>Thalidomide</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Talc</td>
<td>Alpha interferon</td>
</tr>
</tbody>
</table>

Drugs associated with SLE:

| Procainamide hydrochloride | Isoniazid | Hydralazine hydrochloride | Hydantoit | Penicillamine |
## Lung cancer

### Definition
Malignancy that originates in the airways or pulmonary parenchyma

### Classifications/subtypes
Broad clinical classification into:
- Small cell lung cancer (SCLC) and non-small cell lung cancer (NSCLC)

### Examination
Many patients have no signs on examination.

- **Inspection**
  - Cachexia
  - Haemoptysis in sputum cup
  - Clubbing
  - Hypertrophic pulmonary osteoarthropathy (not SCLC)

- **Palpation**
  - Lymphadenopathy (supraclavicular, axillary)

- **Auscultation**
  - Fixed inspiratory wheeze

### Other
- Pleural effusion
- Pneumonia
- Less commonly: signs of focal emphysema, atelectasis, bronchitis, bronchiectasis

### Signs of metastases
- Bony tenderness of ribs (bone), hepatomegaly (liver), confusion, fits, focal neurological signs (brain)

### Presentation
**Typical presentation:** Absence of symptoms until local spread or metastases is common

- Advanced disease seen in majority of clinical presentations

  - 75% of patients have ≥1 symptom at diagnosis
    - Cough (45-75%)
    - Weight loss (46-68%)
    - Dyspnoea (37-58%)
    - Chest pain (27-49%)
    - Haemoptysis (27-29%)
    - Bone pain (20-21%)
    - Hoarseness (8-18%)

### Other features in presentation may include:
- Pleural effusion
- Recurrent pneumonia
- Superior vena cava syndrome (fullness in head, dyspnoea commonly, may have cough, pain, dysphagia)
- Extrathoracic metastases (liver, bone, adrenal gland, brain)
- Paraneoplastic syndromes (hypercalcaemia, SIADH, neurological manifestations, haematological manifestations, hypertrophic osteoarthropathy, dermatomyositis and polymyositis, Cushing’s syndrome)

### History taking:
- Smoking history: pack years, current/past smoker
- Exposure history: occupational and environmental (asbestos, dusts, chemicals, metals)
- Past medical history: radiation, past malignancy, lung conditions and infections
- Family medical history: lung cancer, other malignancies

### Investigations
- **CXR** (raises suspicion of lung cancer)
  - Mass or nodule
  - Hilar and mediastinal adenopathy
  - May also see: cavitations (rare), lobar atelectasis, pleural effusion

- **Tissue diagnosis** (required to confirm Dx and determine histology)
  - FNA under CT or fluoroscopic guidance (transbronchial needle aspiration)
  - Resection of lesion
  - Thoracentesis (if pleural effusion)
  - Bronchial washings or brushings
  - Sputum cytology

- **Lymph node biopsy** (diagnose SCLC vs. NSCLC)
  - Transbronchial biopsy
  - Thorascopy
  - Mediastinoscopy or mediastinotomy

- **CT** (staging – more sensitive than CXR)
  - Lung mass
  - Adenopathy

- **Bone scan**
  - Bony metastases (can assist in staging)

### Differentials
- Lung mass: tuberculosis, granulomatous (sarcoidosis, Wegener’s), fungal (histoplasmosis, coccidiomycosis, Cryptococcus)

---

### Management
**NSCLC**

- **Stage I and II**
  - Surgical resection offers best long term survival rate and cure
  - Suitability according to pre-operative staging (resectability), performance status regarding comorbidities, pulmonary function (operability).
  - Post-operative adjuvant chemotherapy improves survival (NSCLC stage II)

- **Radiotherapy** can be provided for non-surgical candidates and may include stereotactic radiosurgery, radiofrequency ablation, photodynamic therapy (primary treatment in superficial airway lesions)

- **Stage III**
  - Combined radiotherapy and chemotherapy with some role for surgical resection

- **Stage IV**
  - Palliative symptomatic treatment (not curative). Types may include chemotherapy, molecular targeted therapy, radiotherapy, surgery

**SCLC**

- **Limited stage disease**
  - Combination chemotherapy and radiotherapy
  - Usually not surgical resection unless solitary pulmonary nodule with no lymph node involvement or metastases

- **Extensive stage disease**
  - Chemotherapy alone (initial)
  - Prophylactic radiation therapy
  - Both limited and extensive stage disease
  - ↓ incidence of brain metastases and ↑ survival
**Aetiology**

**Risk factors**

- **Smoking**
  - Primary risk factor, accounts for 90% of lung cancers, 20x ↑ risk for a patient with 40 pack years compared to non-smoker
  - Passive smoking also ↑ risk.

- **Radiation**
  - Radiation therapy ↑ the risk of a primary lung cancer in those being treated for other malignancies (especially ipsilateral lung)

- **Environmental toxins** (act as carcinogens)
  - Second hand smoke, asbestos, radon, metals (arsenic, chromium, nickel), ionizing radiation, polycyclic aromatic hydrocarbons

- **Pulmonary fibrosis**
  - 7x risk

- **Genetic factors**
  - Familial risk clearly established
  - Specific genetic markers (oncogenes – EML4-ALK fusion gene, K-ras oncogene, HER2 oncogene, Bcl-2 gene, tumour suppressor genes – p53) have been implicated but are still being investigated

**Pathophysiology**

**WHO classification for primary lung cancer:**

*Histological types (WHO)*

- Small cell carcinoma (13%)
- Adenocarcinoma (including bronchioalveolar carcinoma) (38%)
- Squamous cell carcinoma (20%)
- Large cell carcinoma (5%)
- Other non-small cell carcinomas that cannot be further classified (18%)
- Other (6%)

Clinically, NSCLC is made up of adenocarcinoma, squamous cell carcinoma and large cell carcinoma.

- 95% of lung cancers are SCLC or NSCLC.

**Symptomatology**

- Direct effects of the tumour (chest pain, haemoptysis),
- Local effects of tumour (phrenic nerve irritation causing cough, mass effects causing hoarse voice)
- Local spread
- Metastatic spread (bony metastases causing bone pain)

**Staging: TNM staging for lung cancer** (see online reference for full-sized copy)

---

**Epidemiology**

**Prevalence and incidence**

- Most commonly Dx cancer in Australia
- Dramatic ↑ in relative incidence of adenocarcinoma and corresponding ↓ in incidence of other types of NSCLC and SCLC

**Mortality**

- Most common cause of cancer death worldwide
- Most common cause of death in M
- Third most common cause of death in F
- ATSI>non-ATSI rates of mortality from lung cancer
- 5th most common premature cause of death in Australia
- 4th leading cause of all deaths in Australia in 2009
- 5 year survival rate: 10% males, 12% females

---

**Approach to possible lung cancer**

<table>
<thead>
<tr>
<th><strong>Ask yourself</strong></th>
<th><strong>Ask the patient</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Is it lung cancer? (Biopsy)</td>
<td>What do they think is going on?</td>
</tr>
<tr>
<td>What type of lung cancer is it? (Pathology)</td>
<td>What would they like to happen?</td>
</tr>
<tr>
<td>Has it spread? Local? Distant? Paraeoplastic? (Consider investigations – LFTs, U&amp;Es, Ca)</td>
<td>What are they scared of? Prognosis? Cause of death?</td>
</tr>
<tr>
<td>What is the best treatment? Curative? Palliative?</td>
<td></td>
</tr>
<tr>
<td>Are they fit for their treatment? Heart? Lungs?</td>
<td></td>
</tr>
<tr>
<td><strong>Classification</strong></td>
<td><strong>Differentials</strong>&lt;sup&gt;5&lt;/sup&gt; Sarcoidosis, Malignancy, Histoplasmosis, Coccidiosis (USA)</td>
</tr>
<tr>
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<tr>
<td><strong>Active disease:</strong>&lt;br&gt; - Uncontrolled disease by Mycobacterium tuberculosis causing clinical features and infectivity.</td>
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<tr>
<td><strong>Latent disease:</strong>&lt;br&gt; - Absence of active disease through control by cell mediated immunity but persisting infection with Mycobacterium tuberculosis bacilli.&lt;br&gt; - Clinical features are absent and patient is not infectious.</td>
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<tr>
<td><strong>Primary disease:</strong>&lt;br&gt; - Active disease upon infection with M. tuberculosis</td>
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<tr>
<td><strong>Reactivation disease:</strong>&lt;br&gt; - Active disease years after infection with M. tuberculosis</td>
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<tr>
<td><strong>Disseminated disease:</strong>&lt;br&gt; - Dissemination of bacilli</td>
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<tr>
<th><strong>Presentation</strong></th>
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<tr>
<td><strong>Primary tuberculosis</strong>&lt;br&gt; - Most common presentation of primary disease: fever (low grade, typically 14-21 days duration)</td>
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<tr>
<td><strong>Other symptoms in primary disease (&lt;25%): chest pain, pleuritic chest pain, bronchial lymphadenopathy, arthralgia, pharyngitis</strong></td>
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<tr>
<td><strong>Reactivation tuberculosis</strong>&lt;br&gt; - Classical symptoms of reactivation disease: night sweats, malaise, cough (non-productive or scant productive, ↑ in morning, progresses to productive of yellow-green sputum and continuous), haemoptysis (due to caseous sloughing, endobronchial erosion, blood typically in small amounts), weight loss</td>
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<tr>
<td><strong>Recovery disease may also have: chest pain, dyspnoea</strong></td>
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<tr>
<th><strong>Ask about</strong></th>
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<tr>
<td>Recent travel to places where tuberculosis is endemic</td>
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<tr>
<td>Contact with people with known tuberculosis</td>
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<tr>
<td>Past tuberculosis infection or BCG vaccination</td>
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<tr>
<td>HIV/AIDS status</td>
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<tr>
<th><strong>Management</strong></th>
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<tr>
<td><strong>Antibiotic treatment</strong>&lt;br&gt; - Long term, combination therapy:</td>
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<tr>
<td>- Therapeutic Guidelines: Antibiotics&lt;br&gt; Isoniazid 300mg, po, daily for 6/12 PLUS&lt;br&gt; Rifampcin 600mg, po, daily for 6/12 PLUS&lt;br&gt; Ethambutol 15mg/kg, po, daily for 2/12 PLUS&lt;br&gt; Pyrazinamide 25-40mg/kg up to 2mg, po, daily for 2/12</td>
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<tr>
<td>- Consider susceptibilities when prescribing regimen.</td>
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<tr>
<th><strong>Side effects:</strong></th>
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<tr>
<td>- Isoniazid: Hepatitis, neuropathy, pyridoxine deficit, agranulocytosis.</td>
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<tr>
<td>- Rifampcin: Hepatitis, orange discolouration of urine and tears, inactivation of oral contraceptive pill, flu-like symptoms. Cease if rise in bilirubin.</td>
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<tr>
<td>- Ethambutol: Optic neuritis</td>
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<tr>
<td>- Pyrazinamide: Hepatitis, arthralgia. Contraindicated in acute gout and porphyria.</td>
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<tr>
<th><strong>Prevention</strong></th>
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<tr>
<td>- Bacillus Calmette-Guerin (BCG) vaccine (live attenuated)</td>
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<th><strong>Examination</strong></th>
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<td><strong>Physical findings are non-specific and often absent in mild or moderate disease.</strong></td>
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<tr>
<th><strong>Inspection</strong></th>
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<tr>
<td>- Febrile, finger clubbing</td>
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<tr>
<th><strong>Chest examination</strong></th>
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<td>- Typically no abnormal findings on chest examination</td>
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<tr>
<td>- Signs of pleural effusion may be present&lt;br&gt; o Displaced trachea and apex beat away from the effusion, reduced chest expansion, stony dull percussion note, reduced or absent breath sounds, reduced vocal resonance</td>
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<tr>
<td>- Signs of pleural thickening may be present&lt;br&gt; o Dull percussion note and decreased fremitus</td>
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<td>- Inspiratory or post-tussive (post-cough) crackles</td>
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<tr>
<td>- Consolidation if large area of lung involved&lt;br&gt; o Dull percussion, ↓ expansion, bronchial breathing</td>
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<tr>
<th><strong>Disseminated disease</strong></th>
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<tr>
<td>- May have abnormal findings according to site of military tuberculosis if disseminated disease&lt;br&gt; o E.g. hepatosplenomegaly, meningitis, lymphadenopathy, dyspnoea, pleural effusion</td>
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<tr>
<th><strong>Investigations</strong></th>
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<tr>
<td><strong>Bloods</strong></td>
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<tr>
<td>- FBC typically shows no changes</td>
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<td>- Advanced disease: normocytic anaemia, leukocytosis, monocytosis</td>
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<tr>
<th><strong>CXR</strong></th>
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<td>Primary tuberculosis:</td>
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<tr>
<td>- Hilar adenopathy (seen within 1/52-8/52)</td>
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<tr>
<td>- Pleural effusion (1/3 of patients within first 3-4/12)</td>
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<tr>
<td>- Pulmonary infiltrates (peri-hilar, pleural effusion)</td>
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<td>- Right middle lobe collapse</td>
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<td>- Focal shadowing</td>
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<td>- Solitary nodules</td>
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| **Reactivation tuberculosis:** | |
| - 80-90% → apical-posterior segment of upper lobes | |
| - Pulmonary infiltrates | |
| - Cavitations (unlike primary disease) | |
| - No lymphadenopathy (unlike primary disease) | |
| - Air fluid level may be visible | |
| - Fibrosis and calcification may be seen | |

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<tr>
<th><strong>CT scan</strong></th>
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<td>- More sensitive than CXR (esp. for small apical lesions)</td>
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<td>- May visualize cavities, centrilobular lesions, nodules, branching linear densities (“tree in bud” appearance )</td>
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<th><strong>MCAS</strong></th>
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<td>- Clinical samples (spumtum, pleural fluid, as indicated urine, pus, peritoneal fluid, bone marrow, CSF) should be tested for M, tuberculosis acid fast bacilli.</td>
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<td>- Caseating granulomata is classical of disease.</td>
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<tr>
<th><strong>Mantoux test (tuberculin skin test)</strong></th>
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<td>- Intradermal injection of TB antigen with recording of cell-mediated response after 48-72 hours.</td>
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<td>- Positive test indicates immunity (previous infection or BCG vacc, ↑ positive indicates active infection).</td>
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<tr>
<th><strong>Interferon gamma testing (Quantiferon-TB/T-spot-TB)</strong></th>
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<td>- Measures delayed hypersensitivity reaction to exposure to Mycobacterium tuberculosis.</td>
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**Aetiology**

*Cause*
- Mycobacterium tuberculosis
- Anaerobic, slow growing pathogen (20-24 hours), difficult to identify (acid fast bacilli) due to mycolic acid surface coating with no true outer membrane of cell envelope which makes it difficult for gram staining (stains gram positive). Acid fast stain (Ziehl-Neelsen stain) used instead.
- Virulence factors include mycolic acid glycolipids and trehalose dimycolate ‘cord factor’ (form granulomas), catalase-peroxidase and lipoarabinomannan (resist oxidative stress response from host, induce cytokines).

*Risk factors*
- Immunosuppression (HIV, AIDS, end stage renal disease, diabetes mellitus, malignant lymphoma, corticosteroids, TNF-alpha inhibitors, old age due to decreased cell mediated immunity)
- Low socioeconomic status, overcrowding, poor access to healthcare
- Family history of tuberculosis

**Pathophysiology**

Inhalation of Mycobacterium tuberculosis bacilli and deposition in the lungs can result in several outcomes
- Clearance of the organism
- Chronic latent infection
- Rapidly progressive active disease (primary disease)
- Active diseases years after infection (reactivation disease)

*Primary disease*
- Uncommon (5-10%), high risk in patients with AIDs
- Bacilli are deposited in the alveoli → evade the innate immune system → proliferate inside alveolar macrophages → kill the cells
- Infected alveolar macrophages produce cytokines, chemokines → recruit phagocytes, macrophages, neutrophils → form nodular granuloma (tuberculoma)
- Uncontrolled replication → infection of lymph nodes → lymphadenopathy
- Ghon’s complex = infection from expansion of tubercle from alveoli to lung parenchyma and lymph nodes
- Primary infection occurs until cell mediated immune response occurs (typically 2-6 weeks following infection)
- If no CMI → progressive lung destruction → haematogenous spread → dissemination (spleen, liver, kidneys, brain, joints) → military tuberculosis (millet seed appearance)
- If caseating lesions invade into the airway → host is infectious to others.
- Resolution of disease → healing by fibrosis around tuberculous lesions
- Complete eradication of Mycobacterium tuberculosis is rare and latency most commonly occurs

*Reactivation disease*
- Proliferation of latent bacteria
- Most commonly in immunocompromised patients.
- Reactivation disease is typically more localized (apex of lung with disseminated disease uncommon) with less involvement of lymph nodes and less caseation.

**Epidemiology**

*Worldwide*
- 2nd most common infectious cause of death worldwide
- 8 million new cases of active TB/year
- 1.7 million TB deaths/year
- Magnified by concurrent epidemic of HIV

*Australia*
- 1000 notifications/year
- Most cases due to latent re-activation in patients infected in birth countries (migrants or refugees) or in childhood (Australia)
- 85% of notifications for TB in overseas-born Australians
- Reactivation tuberculosis is the most common type of TB infection seen = 90% of non-HIV adult cases

*Trends*
- Decline observed in 20th century however resurgence in 1990s due to rise in HIV co-infection, drug resistance and poor management of control programs.
Interpreting chest x-rays

**XR**
X-ray is a radiological imaging technique which is painless, fast and easy.
5 Roentgen densities seen:
From most black (exposed) to most white (blocked)
1. Gas
2. Fat
3. Soft tissue
4. Bone
5. Metal

Radiation dose
- X-ray: 0.2mSv
- Annual background radiation dose: 2.6mSv

Process
- X-ray source and x-ray receiving plate
- Stand with chest against x-ray plate (PA) or if unable to stand lie on a table (AP).
- Patient takes a deep breath and holds inspiration whilst x-ray is taken.

**Indications for CXR**
- Chest pain
- Cough
- Dyspnoea
- Other cardiovascular complaints
- Other respiratory complaints
- Septic screen
- Abdominal pain (if suspected cardiac/thoracic origin)
- Rib fractures
- Diagnostic
- Monitoring (resolution of pneumonia, etc)

**Signs on CXR**
- Silhouette sign
  - Loss of lung/soft tissue interface
  - Implies two areas of similar radiodensity
  - Caused by pathology where normal air in lung is displaced/replaced
  - E.g. Silhouette sign seen in right middle lobe pneumonia where consolidation results in loss of right heart border

**Systematic interpretation of CXRs**
- ABCDE
  - A: Airways (bronchi, lung, pleura)
  - B: Bones (ribs, clavicles, scapula)
  - C: Circulation (heart, vessels, mediastinum)
  - D: Diaphragm
  - E: Soft tissue (breast) and other (lines, tubes, artefacts)
- Outside chest to inside chest

**General points for interpretation of CXR**
- Refer to lung zones (e.g. left upper zone of lung) rather than lobes
- Compare left to right side

**Normal CXR**

Visible structures
1. Trachea
2. Hilum
3. Lung
4. Hemi-diaphragm
5. Heart
6. Aortic knuckle
7. Ribs
8. Scapula
9. Breast
10. Stomach

Obscured or invisible structures (typically only visible on CXR when abnormal)
- Sternum
- Oesophagus
- Spine
- Pleural
- Lung fissures
- Aorta

---

Interpreting a CXR

1. Identify the CXR
   - Correct patient name, date of birth, UMRN, gender.
   - Correct date and time of CXR.

2. Technical aspects
   - Rotation
     - Centred CXR will have symmetrical distance between L and R sternoclavicular joint and central spinous process of vertebrae
   - Penetrance
     - Optimal penetration: vertebral bodies are just visible
     - Under-penetration: vertebral bodies cannot be visualized
     - Over-penetration: vertebral bodies are distinctly visible, lung markings are poorly seen and lungs are very black
   - Patient position
     - Label should denote PA/AP/lateral and erect/supine/decubitus/sitting
     - PA (posterior-anterior)
       - Usual position
       - X-ray source posterior to patient and receiving x-ray plate anterior to patient (patient stands hugging plate against chest)
     - Scapulae are clear of lungs on PA
     - All are erect CXR
     - AP
       - May be taken if patient is unable (too ill, etc) to stand for PA view
       - All supine are AP, AP may also be done sitting or standing
     - Lung volume
       - CXR requires full inspiration to be held whilst film is being taken to visualize lung abnormalities
       - Normal inspiration should see diaphragm at 6th rib anteriorly or 8-10th rib posteriorly

3. Skin and soft tissue
   - Body habitus
     - Is patient obese or very thin?
   - Breast
     - Can breast shadow be seen?
     - Mastectomy?
   - Breast

4. Pleura
   - Thick or thin?
   - Fluid or air in pleural space?
   - Mass or nodules in pleural space?
   - Asbestosis/mesothelioma?

5. Bones
   - Consider: ribs, clavicles, scapula, vertebrae
   - Symmetrical? (scoliosis, chest deformity)
   - Dislocations, fractures? (rib fracture: “arrowhead”)

6. Heart
   - Cardiothoracic ratio
     - Width of heart: width of thorax
     - <50%: normal
     - >50%: cardiomegaly
     - Cardiomegaly can only be Dx on PA film as AP film magnifies heart due to divergence. Only assessment that can be made from AP film is cardiothoracic ratio <50% is normal.

7. Lungs
   - ↑ opacification
     - Pulmonary oedema (diffuse opacification)
     - Interstitial lung disease (reticular white line markings)
     - Nodular (small, white, round markings)
   - ↓ opacification
     - Emphysema (↓ of lung markings, very black lung)
   - ↑ lung volume
     - Hyperinflation (COPD)
   - ↓ lung volume
     - Atelectasis
   - Fluid level
     - Pulmonary effusion (meniscus seen)
   - Peripheral lung markings
     - Should be visible to chest wall
     - If not visible (pneumothorax)
   - Hila
     - Left hilum should be higher than right (heart)
     - Hilar lymphadenopathy?
   - Fissures
     - Right lung
       - 3 lobes (U, M, L)
       - Horizontal fissure (U/M lobes)
       - Oblique fissure (M/L lobes)
     - Left lung
       - 2 lobes (U, L)
       - Oblique fissure (U/L lobes)
     - Horizontal fissures seen on frontal view
     - Oblique fissures seen on lateral view

8. Hemidiaphragms
   - Right hemidiaphragm should be higher than left hemidiaphragm (due to liver on right side)
   - Costophrenic angles
     - Should be sharp and well-defined
     - Abnormalities
       - Blunt/flattened hemi-diaphragm (pleural effusion, hyperinflation)
       - Hemidiaphragm lower than expected (hyperinflation in COPD)
       - Hemidiaphragm higher than expected (poor inspiration on x-ray)
   - Free gas under diaphragm
     - Perforated hollow viscous (e.g. small bowel perforation)

9. Mediastinum
   - Consider: tracheal, oesophagus
   - Deviated from midline
     - Tension pneumothorax (deviated away from affected lung)
     - Atelectasis (deviated towards affected lung)

10. Abdomen
    - Stomach and bowel
      - Gas?

11. Other
    - Lines
      - Chest drain
      - Central line (to lower superior vena cava)
      - Endotracheal tubes
      - Gastric tubes
Normal Trachea and bronchi

Normal Pulmonary arteries

Normal hila

Normal lung zones

Normal pleura and pleural spaces

Normal costophrenic angle and recess

Normal hemidiaphragms

Normal heart size

Normal cardiac contours

Normal bony landmarks

Left pneumothorax (traumatic injury)⁹
Right pleural thickening (mesothelioma)⁹
Bilateral pleural plaques (asbestos)⁹
Left pleural effusion (lung cancer)⁹

Left middle zone consolidation (pneumonia)⁹
Bilateral lung nodules (pulmonary metastases)⁹
Left hyperinflation (COPD)⁹
Left pneumothorax (traumatic injury)⁹

Mediastinal mass (Hodgkin’s lymphoma)⁹
Pneumoperitoneum (ruptured peptic ulcer)⁹
Left diaphragmatic rupture (trauma)⁹
Cardiomegaly (heart failure)⁹

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