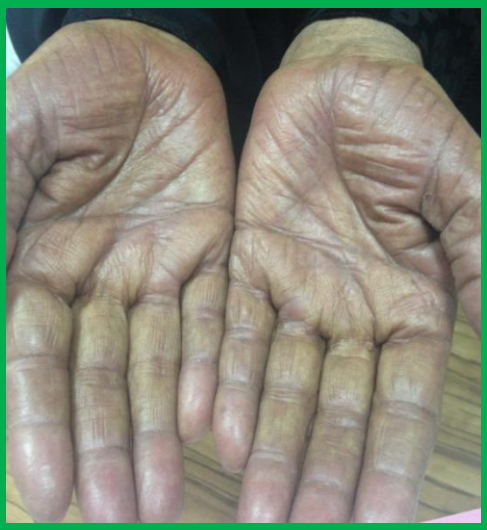
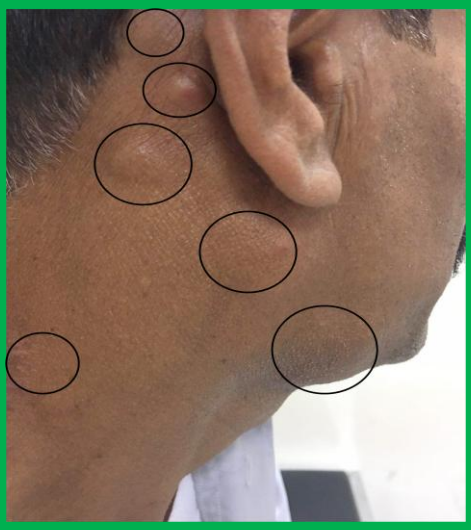
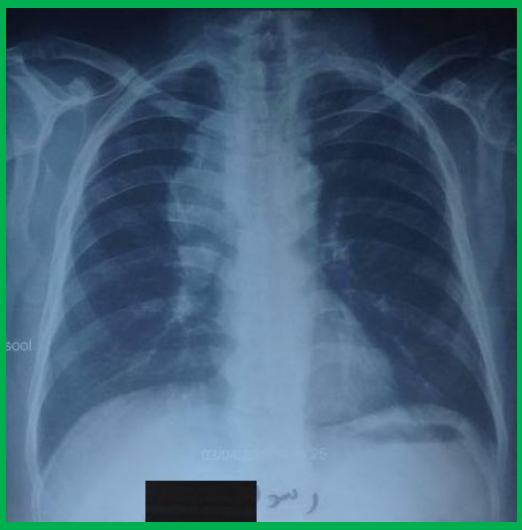
Short Window

on Medicine

2017

Section Four **Respiratory System**

Prof. Nasser Ghaly Yousif
Prof. Kathy A. Shwaan

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Prof. Nasser Ghaly Yousif
MD, MSc (Germany), MRCP(UK), Postdoctorate/Fellowship(US)
Medical Oncology/University of Colorado School of Medicine
Professor Lecturer/Hemato-Oncology
Muthanna Medical College

Prof. Kathy A. Shwaan
MD, ABIM, Postdoctorate
Professor Lecturer/Internal Medicine
University of Colorado School of Medicine

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Preface

History taking and physical examination are an art and cannot be acquired simply by reading books. Nonetheless, a book such as this can help you organize your approach to each symptom, select a battery of appropriate questions, interpret the information received, and narrow down the diagnostic hypotheses. It is important that students learn a structured approach from the very beginning during their clinical attachments, but it is never too late for postgraduate students to adapt and develop it. There are 6–8 principal symptoms in each system and students should consult a book on clinical skills and master a battery of questions for each symptom, and then practice the art of asking these questions at every opportunity during their clinical training. Remembering the questions is easier than the art of asking them, which can be improved by constant practice, self-criticism and helpful comments from a good teacher. A famous neurologist once said, during a teaching session, that diagnosing the cause of headache the most common symptom in medicine is like completing a jigsaw puzzle of asking 13 questions. Those who can only count 12 questions should consider asking the final question to the patient as to what he or she thinks are the cause of the headache. The same can be said about any other symptom such as chest pain or palpitations. It is important to explore the presenting symptoms fully before going on to other aspects of the history taking. The examiners take a dim view of any candidate who skates back and forth from the presenting complaints to past or family history. It becomes easier to identify the chief areas of concern in other parts of the history, and the possible risk factors, only after adequately exploring the presenting complaint (s). Besides, it is imperative to let the patient ventilate fully his or her major concerns both in clinical practice and in the exam. A systems review will be necessary to find out if the patient has any other complaint which he or she has not mentioned. During your clinical attachments, foundation programme and core training appointments, you should get into the habit of going over your notes each time you take a history, and judging whether you have covered all aspects of the history and then assembled the appropriate differential diagnoses. Once you have done that you should then prepare a summary of the problem(s) and the possible management plan and articulate it vocally to yourself. This habit will serve you well for any examination. As you prepare for the clinical exam, you should act out each history scenario from this book with a fellow candidate and discuss the conclusions and management plans. This will tighten up your history-taking technique and your presentation skills. Remember, the examiners do not know you are clever; you have to demonstrate it. The exercise will also help to make you a methodical and articulate clinician.

Section Four

Respiratory System

- ✓ Lectures Note
- ✓ Clinical Approach
- ✓ Communication Skills
- ✓ Questions

Lectures Note

Physiology of respiratory system

Hamburger (Chloride) shift

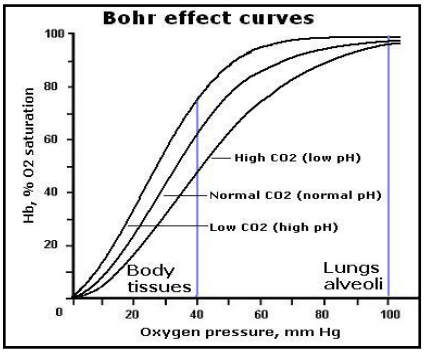
- CO2 diffuses into RBCs
- CO2 + H20 ---- carbonic anhydrase -→ HCO3- + H+
- H+ combines with Hb
- HCO3- diffuses out of cell, Cl- replaces it

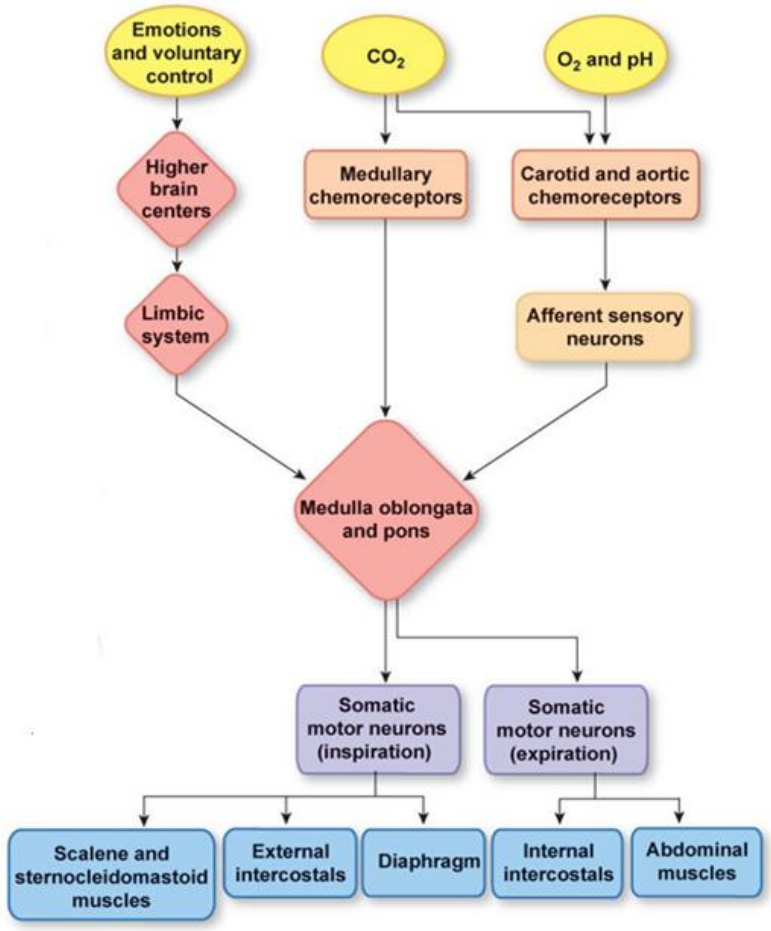
Bohr Effect

- increasing acidity (or pCO2) means O2 binds less well to Hb

Haldane effect

- increase pO2 means CO2 binds less well to Hb





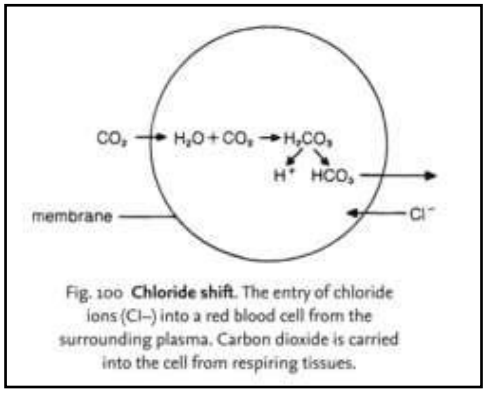
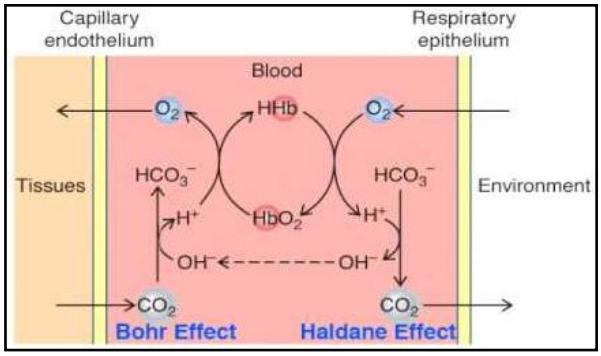


Fig. 100 **Chloride shift.** The entry of chloride ions (Cl–) into a red blood cell from the surrounding plasma. Carbon dioxide is carried into the cell from respiring tissues.



Control of respiration

Central regulatory centers; **C**entral and peripheral chemoreceptors; **P**ulmonary receptors

Central regulatory centers

Medullary respiratory center
Apneustic centre (lower pons)
Pneumotaxic center (upper pons)

Central and peripheral chemoreceptors

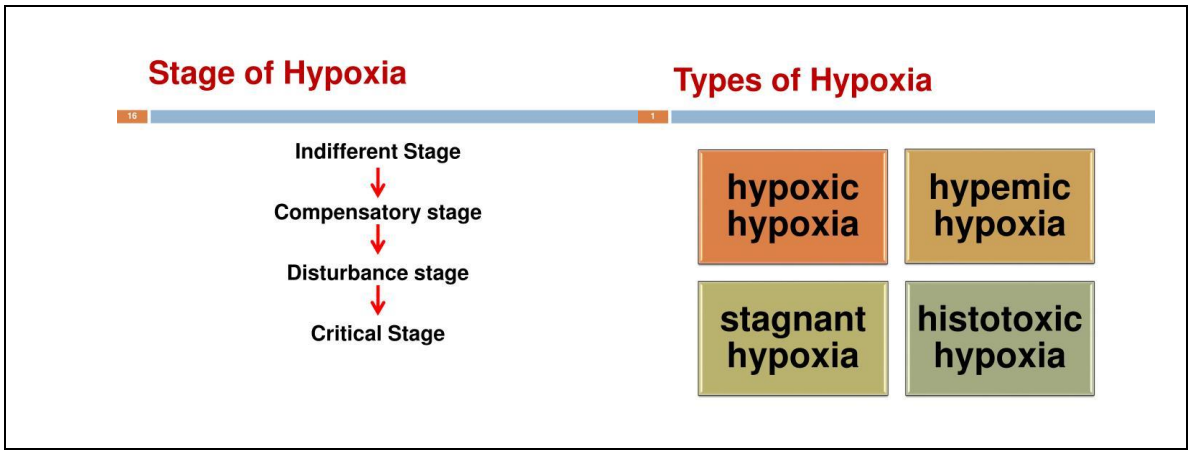
Central: raised [H+] in ECF stimulates respiration
Peripheral: carotid + aortic bodies, respond to raised pCO2 & [H+], lesser extent low pO2

Pulmonary receptors

Stretch receptors, lung distension causes slowing of respiratory rate (Hering-Bruer reflex)
Irritant receptor, leading to bronchoconstriction
Juxta-capillary receptors, stimulated by stretching of the microvasculature

Hypoxia

A **fall** in the partial pressure of oxygen **pO2**in the blood leads to **vasoconstriction of the pulmonary arteries**. →This allows blood to be diverted to better aerated areas of the lung and improves the efficiency of **gaseous exchange**



Pulmonary surfactant

- ✓ Surfactant is a mixture of phospholipids, carbohydrates and proteins released by **type 2 pneumocytes**.
- ✓ The main functioning component is dipalmitoyl phosphatidylcholine (DPPC) which **reduces alveolar surface tension**.
- ✓ first detectable around 28 weeks
- ✓ as alveoli decrease in size, surfactant concentration is increased, helping prevent the alveoli from collapsing
- ✓ reduces the muscular force needed to expand the lungs (i.e. decreases the work of breathing)

Pulmonary capillary wedge pressure (PCWP)

- ✓ Pulmonary capillary wedge pressure is measured using a balloon tipped **Swan-Ganz catheter** which is inserted into the **pulmonary artery**.
- ✓ The pressure measured is similar to that of the **left atrium** (normally **6-12 mmHg**).
- ✓ One of the main uses of measuring the PCWP is determining whether pulmonary edema is caused by either **heart failure** or **ARDS**.
- ✓ In many modern ITU departments PCWP measurement has been replaced by non-invasive techniques.

Lung compliance:

Defined as change in lung volume per unit change in airway pressure

Causes of increased compliance

- ✓ age
- ✓ emphysema - this is due to loss alveolar walls and associated elastic tissue

Causes of decreased compliance

- ✓ Pulmonary edema
- ✓ Pulmonary fibrosis
- ✓ Pneumonectomy
- ✓ Kyphosis

Oxygen dissociation curve

- ✓ The oxygen dissociation curve describes the relationship between the percentage of **saturated hemoglobin** and partial pressure of oxygen in the blood **pO_2.**
- ✓ It is not affected by hemoglobin concentration

Basics:

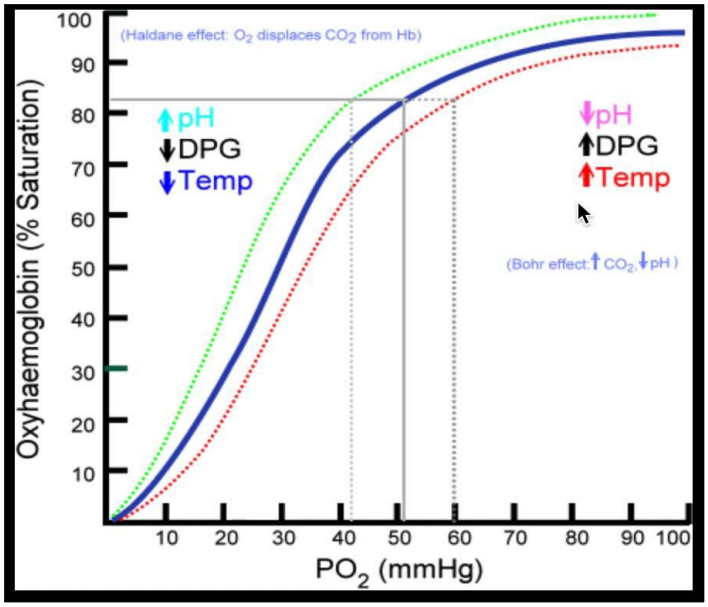
- ✓ **shifts to left** = for given oxygen tension there is increased saturation of Hb with oxygen i.e. decreased oxygen delivery to tissues
- ✓ **shifts to right** = for given oxygen tension there is reduced saturation of Hb with oxygen i.e. enhanced oxygen delivery to tissues

Shifts to Left = Lower oxygen delivery	**Shifts to Right = Raised oxygen delivery**
✓ HbF, methaemoglobin, carboxyhaemoglobin ✓ Low [H+] (alkali) ✓ Low pCO2 ✓ Low 2,3-DPG ✓ Low temperature	✓ Raised [H+] (acidic) ✓ Raised pCO2 ✓ Raised 2,3-DPG* ✓ Raised temperature

The L rule:
Shifts to L → Lower oxygen delivery, caused by

- Low [H+] (alkali)
- Low pCO2
- Low 2,3-DPG
- Low temperature

Mnemonic is:
'CADET, face Right!'
for CO2, Acid, 2,3-DPG*, Exercise and Temperature
*2,3-diphosphoglycerate



Carbon monoxide poisoning

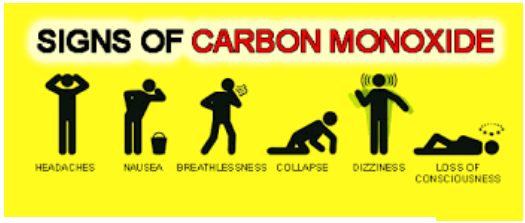
- ✓ Carbon monoxide binds with hemoglobin with a greater affinity than oxygen displacing it from the blood causing tissue hypoxia.
- ✓ In addition, carbon monoxide shifts the oxygen dissociation curve to the left reducing tissue delivery even more.

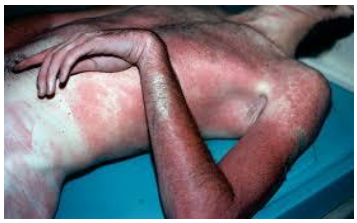
Symptoms of mild poisoning (carboxy hemoglobin levels = **10-30%)**

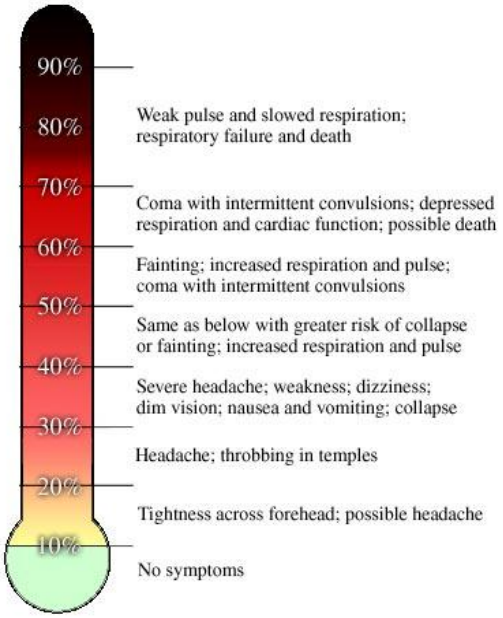
- ✓ Headache, tiredness, nausea, dizziness and poor concentration.
- ✓ With increasing levels vomiting and weakness then impaired consciousness may occur with hypertension, tachycardia and flushing.

Severe poisoning (carboxy hemoglobin levels more than **50%)**

- ✓ Convulsions, coma, respiratory depression and death can occur.







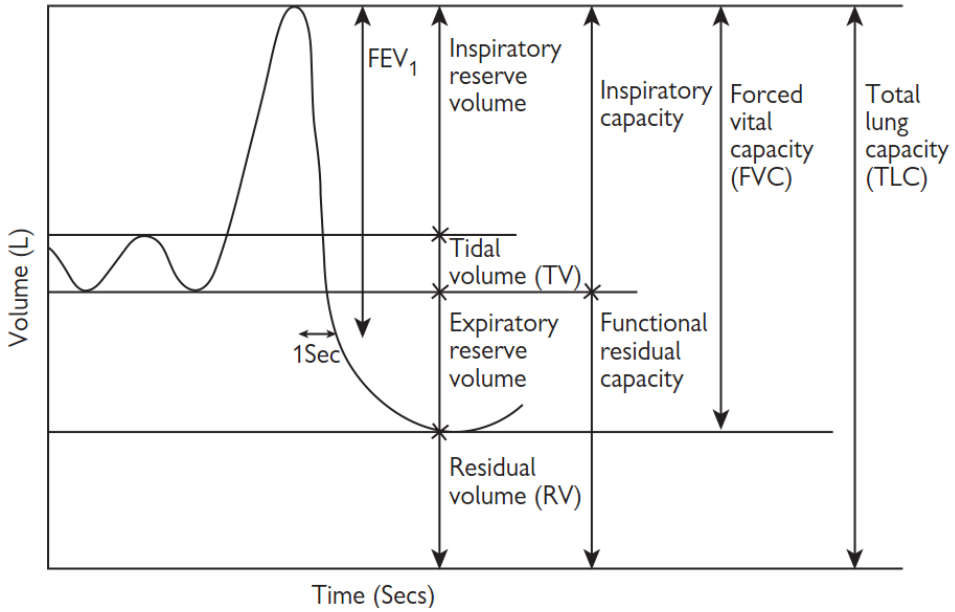
Treatment CO Poisoning

- Increasing the concentration of inhaled oxygen can help minimize the binding of CO to hemoglobin
- Some CO may be displaced from hemoglobin when the patient increases their inhaled oxygen concentrations
- Treatment begins with high index of suspicion and removal to a safer environment
- Immediately begin 100% O_2 delivery

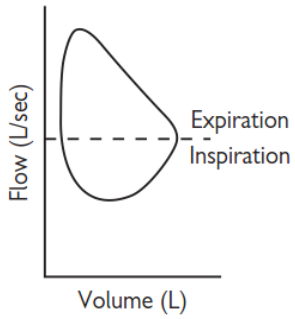
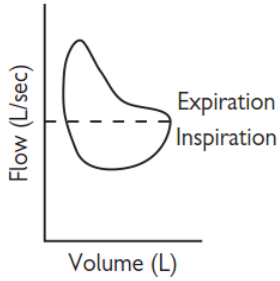
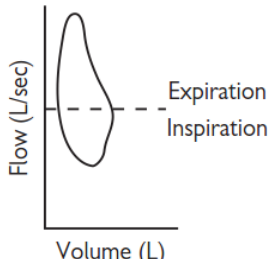
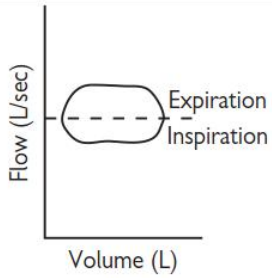
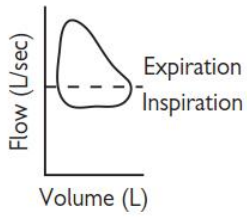
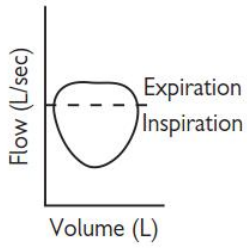
Interpreting pulmonary function tests

Spirometry [Lung volumes]

- ✓ Tidal volume (TV)
  - volume inspired or expired with each breath at rest
  - 500ml in males, 350ml in females
- ✓ Inspiratory reserve volume (IRV) = 2-3 L
  - maximum volume of air that can be inspired at the end of a normal tidal inspiration
  - inspiratory capacity = TV + IRV
- ✓ Expiratory reserve volume (ERV) = 750ml
  - maximum volume of air that can be expired at the end of a normal tidal expiration
- ✓ Residual volume (RV) = 1.2L
  - volume of air remaining after maximal expiration
  - increases with age
  - RV = FRC - ERV
- ✓ Vital capacity (VC) = 5L
  - maximum volume of air that can be expired after a maximal inspiration
  - 4,500ml in males, 3,500 mls in females
  - decreases with age
  - VC = inspiratory capacity + ERV
- ✓ Total lung capacity (TLC) is the sum of the vital capacity + residual volume
  - Measured using helium dilution or body plethysmography.
  - TLC increases in obstructive lung disease with air trapping and hyperinflation.
  - TLC decreases in restrictive lung disease
- ✓ Physiological dead space (V_D)
  - V_D = tidal volume * ($PaCO_2$ - $PeCO_2$) / $PaCO_2$
  - where $PeCO_2$ = expired air CO_2
- ✓ FEV 1 /FVC ratio:
  - Obstructive lung disease indicated if FEV 1 /FVC <0.70.
  - Restrictive lung disease (or normality) indicated if FEV 1 /FVC > 0.70.
- ✓ Diffusion capacity:
  - Calculated by inhaling a mixture of carbon monoxide (CO) (which crosses the alveolar membrane) and helium (which does not)? The amount of CO transferred across the alveolar capillary membrane into the blood is calculated and adjusted for hemoglobin levels (TLCO, total lung CO).
  - The total lung volume is calculated using helium concentration which would be diluted by gas already present in the lung on inspiration.



✓ Flow volume loops

Normal flow volume loop Flow rates are higher at the beginning of expiration and as lung volume reduces flow is limited by airway compression. During inspiration flow rates start small and increase as airways open up then reduce as the inspiratory muscles reach their maximum capacity	
Obstructive lung disease In expiration flow rates decrease more markedly at lower lung volumes due to narrowing of the airways, producing a 'scalloped' appearance. A sensitive test of airflow obstruction involves looking at the flow rates at 25–75% of the maximum expired lung volume.	
Restrictive lung disease The maximum flow rate and total lung volume is reduced with high flow in the latter part of expiration due to increased recoil	
Fixed upper airway narrowing Gives a square appearance as flow rate is limited in inspiration and expiration by the obstruction and is no longer dependent on lung volume	
Extrathoracic upper airway obstruction Obstruction occurs during inspiration giving a square appearance but airway is blown open during expiration	
Variable intrathoracic upper airway obstruction eg tumour at carina Obstruction occurs during expiration when chest is compressed and is reduced during inspiration	

- A normal flow volume loop is often described as a 'triangle on top of a semi-circle'
- Flow volume loops are the most suitable way of assessing **compression of the upper airway**

Obstructive lung disease	Restrictive lung disease
✓ FVC: reduced or normal ✓ FEV1: significantly reduced ✓ FEV1% (FEV1/FVC): reduced	✓ FVC: significantly reduced ✓ FEV1: reduced ✓ FEV1% (FEV1/FVC) - normal or increased
✓ Asthma ✓ COPD ✓ Bronchiectasis ✓ Bronchiolitis obliterans	✓ Pulmonary fibrosis ✓ Sarcoidosis (upper fibrosis) ✓ Asbestosis (lower fibrosis) ✓ Acute respiratory distress syndrome ✓ Infant respiratory distress syndrome ✓ Kyphoscoliosis ✓ Neuromuscular disorders

✓ **Transfer factor**

- The transfer factor describes the rate at which a gas will diffuse from alveoli into blood.
- Carbon monoxide is used to test the rate of diffusion.

Causes of a raised TLCO	Causes of a lower TLCO
✓ asthma ✓ pulmonary haemorrhage (Wegener's, Goodpasture's) ✓ left-to-right cardiac shunts ✓ polycythaemia ✓ hyperkinetic states ✓ male gender, exercise	✓ COPD (much trapped air) ✓ emphysema ✓ pneumonia ✓ pulmonary oedema ✓ pulmonary fibrosis ✓ pulmonary emboli ✓ anaemia ✓ low cardiac output

- KCO also tends to increase with age.

Conditions may cause an increased KCO with a normal or reduced TLCO

✓ Pneumonectomy/lobectomy
✓ Diffuse pleural thickening (usually asbestos)
✓ Scoliosis/kyphosis
✓ Neuromuscular weakness
✓ Ankylosis of costovertebral joints e.g. ankylosing spondylitis

Transfer factor

- **Raised**: asthma, hemorrhage, left-to-right shunts, polycythaemia
- **Low**: everything else

Where alveolar hemorrhage occurs the TLCO tends to increase due to the enhanced uptake of carbon monoxide by intra-alveolar hemoglobin

Chest x-ray finding in Respiratory system pathology

Cavitating lung lesion:

- ✓ abscess (Staph aureus, Klebsiella and Pseudomonas)
- ✓ tuberculosis
- ✓ aspergillosis, histoplasmosis, coccidioidomycosis
- ✓ squamous cell lung cancer
- ✓ pulmonary embolism
- ✓ Wegener's granulomatosis
- ✓ rheumatoid arthritis

Coin lesions:

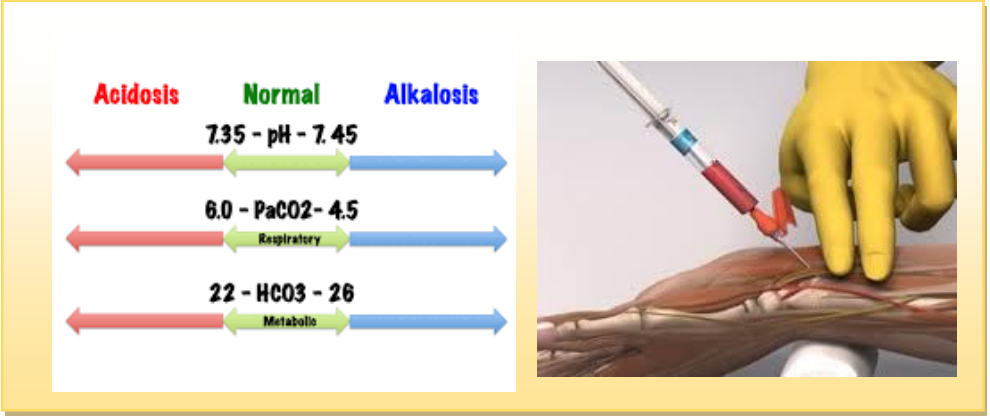
- ✓ malignant tumor: lung cancer or metastases
- ✓ benign tumor: hamartoma
- ✓ infection: pneumonia, abscess, TB, hydatid cyst
- ✓ AV malformation

Blood gases

An arterial blood gas (ABG) test measures the acidity (pH) and the levels of oxygen and carbon dioxide in the blood from an artery. This test is used to check how well your lungs are able to move oxygen into the blood and remove carbon dioxide from the blood.

As blood passes through your lungs, oxygen moves into the blood while carbon dioxide moves out of the blood into the lungs. An ABG test uses blood drawn from an artery, where the oxygen and carbon dioxide levels can be measured before they enter body tissues. An ABG measures:

- Partial pressure of oxygen (PaO_2). These measures the pressure of oxygen dissolved in the blood and how well oxygen is able to move from the airspace of the lungs into the blood.
- Partial pressure of carbon dioxide ($PaCO_2$). These measures the pressure of carbon dioxide dissolved in the blood and how well carbon dioxide is able to move out of the body.
- PH. The pH measures hydrogen ions (H+) in blood. The pH of blood is usually between 7.35 and 7.45. A pH of less than 7.0 is called acid and a pH greater than 7.0 is called basic (alkaline). So blood is slightly basic.
- Bicarbonate (HCO_3). Bicarbonate is a chemical (buffer) that keeps the pH of blood from becoming too acidic or too basic.
- Oxygen content (O_2CT) and oxygen saturation (O_2sat) values. O_2 content measures the amount of oxygen in the blood. Oxygen saturation measures how much of the hemoglobin in the red blood cells is carrying oxygen (O_2).
- Blood for an ABG test is taken from an artery. Most other blood tests are done on a sample of blood taken from a vein, after the blood has already passed through the body's tissues where the oxygen is used up and carbon dioxide is produced.



Asthma

Diagnosis:

- ✓ Whilst the diagnosis of asthma remains largely clinical, the British Thoracic Society (BTS) guidelines do offer some guidance on how we should approach this problem, for both adults and children.
- ✓ They recommend we classify patients as having either a high, intermediate or low probability of asthma based on the presence or absence of certain symptoms
- ✓ For adults, it is recommending that they have a clinical assessment including **spirometry** (or **Peak Expiratory Flow** measurement if spirometry is not available)
- ✓ When assessing patients, we should therefore look for symptoms which may support a diagnosis of asthma and those which may point to an alternative diagnosis.
- ✓ The BTS produced **a list of features** which are helpful when deciding this:

Features which make a diagnosis of asthma more likely	**Features which make a diagnosis of asthma less likely**
✓ More than one of the following symptoms: wheeze, breathlessness, chest tightness and cough particularly if: • symptoms worse atnight and in the early morning • symptoms in response to exercise, allergen exposure and cold air • symptoms after taking aspirin or beta blockers ✓ History of atopic disorder ✓ Family history of asthma and/or atopic disorder ✓ Widespread wheeze heard on auscultation of the chest ✓ Otherwise unexplained low FEV1 or PEF (historical or serial readings) ✓ Otherwise unexplained peripheral blood eosinophilia	✓ Prominent dizziness, light-headedness, peripheral tingling ✓ Chronic productive cough in the absence of wheeze or breathlessness ✓ Voice disturbance ✓ Symptoms with colds only ✓ Significant smoking history (i.e.> 20 pack-years) ✓ Cardiac disease ✓ Repeatedly normal physical examination of chest when symptomatic ✓ Normal PEF or spirometry when symptomatic

High probability:

- ✓ If a patient has many symptoms which make a diagnosis of asthma more likely
- ✓ Then the BTS recommend that we start a trial of treatment.
  - A good response is considered a positive 'test of reversibility'.
  - If poor response to treatment then further investigations should be considered.

Low probability:

- ✓ For patients with a low probability of asthma then an alternative diagnosis should be sought.
- ✓ Further investigations and referral to a respiratory specialist considered.

Intermediate probability:

- ✓ If a patient has an intermediate probability of asthma the BTS recommend carrying out further investigations, including an explicit trial of treatments for a specified period, before confirming a diagnosis and establishing maintenance treatment.
- ✓ The accompanying algorithm suggests this decision should be partly guided by the FEV_1/FVC ratio -a ratio of < 0.7 is suggestive of asthma. (normal 0.75-0.8)

- ✓ It should of course be noted that spirometry may be normal in asymptomatic patients so it may be necessary to repeat spirometry or peak flow readings on a number of occasions in patients where the diagnosis is not clear.
- ✓ Recent studies have shown the limited value of other 'objective' tests.
- ✓ It is now recognized that in patients with normal or near-normal pre-treatment lung function there is little room for measurable improvement in FEV1 or peak flow.
- ✓ **> 400 ml** improvement in **FEV1** is considered **significant**
  - before and after **400 mcg inhaled salbutamol** in patients with diagnostic uncertainty and airflow obstruction present at the time of assessment
  - if there is an incomplete response to inhaled salbutamol, after either inhaled corticosteroids (**200 mcg** twice daily **beclometasone** equivalent for **6-8 weeks**) or oral **prednisolone** (**30 mgOD** for **14 days**)

It is now advised to interpret peak flow variability with caution due to the poor sensitivity of the test

- diurnal variation % = [(Highest - Lowest PEFR) / Highest PEFR] x 100
- assessment should be made over 2 weeks
- > 20% diurnal variation is considered significant

The most appropriate initial treatment

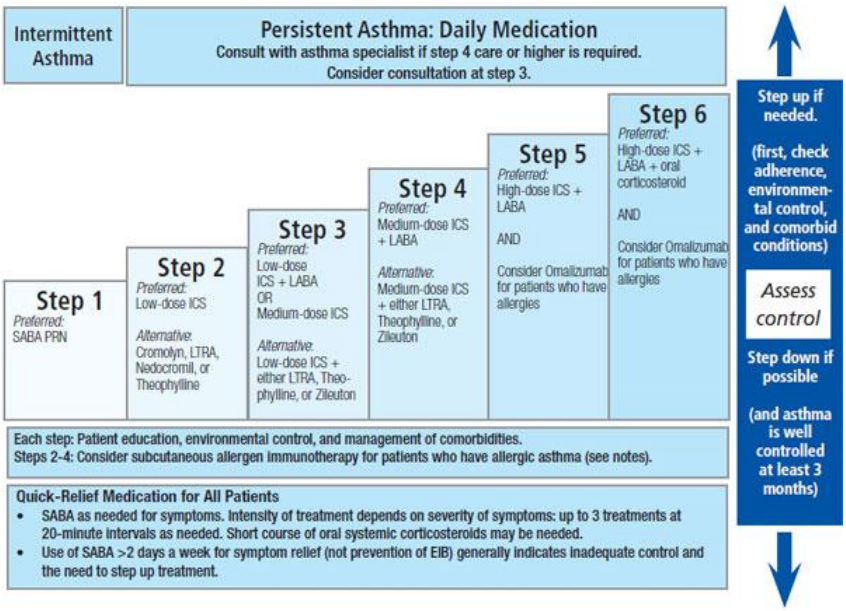
The BTS state the following:

- ✓ Patients should start treatment at the step most appropriate to the initial severity of their asthma
  - This means that for some patients prescribing a corticosteroid inhaler in addition to a salbutamol inhaler is appropriate.
- ✓ The BTS suggest the following patients would benefit from a corticosteroid inhaler:

(Inhaled steroids should be considered for patients with any of the following asthma-related features):

- ✓ exacerbations of asthma in the last two years
- ✓ using inhaled β2 agonists three times a week or more
- ✓ symptomatic three times a week or more
- ✓ waking one night a week

The last two points are probably the most relevant to newly diagnosed patients.



Asthma: stepwise management in adults

The management of stable asthma is now well established with a step-wise approach:

Step	Management
Step 1	**Inhaled short-acting B2 agonist** as required
Step 2	Add **inhaled steroid** at 200-800 mcg/day* 400 mcg is an appropriate starting dose for many patients. Start at dose of inhaled steroid appropriate to severity of disease
Step 3	✓ Add **inhaled long-acting B2 agonist** (LABA) ✓ Assess control of asthma: • good response to LABA →continue LABA • benefit from LABA but control **still inadequate:** ◦ continue LABA and ◦ increase inhaled steroid dose to 800 mcg/day* (if not already on this dose) • no response to LABA: ◦ stop LABA and ◦ Increase inhaled steroid to 800 mcg/ day.* ◦ If control still inadequate, institute trial of other therapies, → **leukotriene receptor antagonist** or **SR theophylline**
Step 4	**Consider trials of:** ✓ Increasing **inhaled steroid up to 2000 mcg/day*** ✓ Addition of a **fourth drug** e.g. • **Leukotriene receptor antagonist,** • **SR theophylline,** • **B2 agonist tablet** The BTS say there is little evidence to separate the different options available. They do however suggest that leukotriene receptor antagonists are least likely to cause side-effects. Monitoring of serum concentrations may also be required for theophyllines
Step 5	✓ Use **daily steroid tablet** in lowest dose providing adequate control. Consider other treatments to minimize the use of steroid tablets ✓ Maintain **high dose inhaled steroid at 2000 mcg/day*** ✓ Refer patient for **specialist care**

*beclometasone dipropionate or equivalent

Additional treatment

A) Leukotriene receptor antagonists:
e.g. Montelukast, zafirlukast
- ✓ have both anti-inflammatory and bronchodilatory properties
- ✓ should be used when patients are poorly controlled on high-dose inhaled corticosteroids and a long-acting b2-agonist
- ✓ particularly useful inaspirin-induced asthma
- ✓ associated with the development of Churg-Strauss syndrome

B) Fluticasone is more lipophilic and has a longer duration of action than beclometasone

C) Hydrofluoroalkane is now replacing chlorofluorocarbon as the propellant of choice. Only half the usually dose is needed with hydrofluoroalkane due to the smaller size of the particles

D) Long acting B2-agonists
- Acts as bronchodilators but also inhibit mediator release from mast cells.
- Recent meta-analysis showed adding salmeterol improved symptoms compared to doubling the inhaled steroid dose

Acute severe Asthma

Patients with acute severe asthma are stratified into moderate, severe or life-threatening

Moderate	Severe	Life-threatening
✓ PEFR **50-75%** best or predicted ✓ Speech normal ✓ RR < 25 / min ✓ Pulse < 110 bpm	✓ PEFR **33 - 50%** best or predicted ✓ Can't complete sentences ✓ RR > 25/min ✓ Pulse > 110 bpm	✓ **PEFR < 33%**best or predicted ✓ **O_2sats < 92%, PO_2 <60, normal PCO_2** ✓ **Silent chest**, **cyanosis** or **feeble** respiratory effort ✓ **Bradycardia, dysrhythmia** or **hypotension** ✓ Exhaustion, **confusion or coma**

Note that a patient having **any one of the life-threatening features** should be treated as having a life-threatening attack.

British Thoracic Society guidelines:
- ✓ **magnesium sulphate** recommended as next step for patients who are not responding (e.g. 1.2 - 2g IV over 20 mins)
- ✓ little evidence to support use of **IV aminophylline** (although still mentioned in management plans)
- ✓ if no response considers **IV salbutamol**

Occupational Asthma (10% of adult asthma)

- ✓ The symptoms do not usually develop immediately on first exposure but begin days, months or even years later.
- ✓ Patients may either present with concerns that chemicals at work are worsening their asthma or you may notice in the history that symptoms seem better at weekends / when away from work.
- ✓ Exposure to the following chemicals is associated with occupational asthma:
  - Isocyanates:
    - The most common cause.
    - Example occupations include spray painting and foam moulding using adhesives
  - platinum salts
  - soldering flux resin
  - glutaraldehyde
  - epoxy resins
  - proteolytic enzymes
  - Bakers (flour mainly but also enzymes such as amylase used in the baking process)
  - Chemical processors (acids, detergents, bleaches)
  - Plastics workers (polyethylene, polyvinyl chloride)
  - Soldiers (colophony), and laboratory technicians (rats, mice, rabbits, locusts).

- Serial measurements of **peak expiratory flow** are recommended at work and away from work.
- Referral should be made to a respiratory specialist for patients with suspected occupational asthma.
- Removal from exposure to the sensitizing agent at an early stage can lead to remission of asthma although sensitization to the agent is usually permanent.

Assessing a patient's **performance status** is important when evaluating the most appropriate treatment options. It is commonly used by **cancer** MDTs, but has a role in assessing patients with **chronic illnesses** including **COPD**.

WHO Scale	Description
0	Asymptomatic
1	Symptomatic but ambulatory (can carry out light work)
2	In bed <50% of the day. Unable to work but can live at home with some assistance
3	In bed >50% of the day but unable to care for self
4	Bedridden

Medical Research Council dyspnea scale:

Grade	Degree of breathlessness related to activities
1	Not troubled by breathlessness except on strenuous exercise
2	Short of breath when hurrying or walking up a slight hill
3	Walks slower than contemporaries on level ground because of breathlessness, or has to stop for breath when walking at own pace
4	Stops for breath after walking about 100 m or after a few minutes on level ground
5	Too breathless to leave the house, or breathless when dressing or undressing

Theophylline

- Theophylline, like caffeine, is one of the naturally occurring **methylxanthines**.
- The main use of theophyllines in clinical medicine is as a bronchodilator in the management of **asthma and COPD**
- The exact mechanism of action has yet to be discovered.
- One theory suggests theophyllines may be a non-specific inhibitor of phosphodiesterase resulting in an increase in **cAMP**.
- Other proposed mechanisms include antagonism of **adenosine** and **prostaglandin inhibition**

Theophylline poisoning

Features:

- Acidosis, hypokalaemia, hypoPh, hypoMg, hypoNa
- hyperglycemia & hyperCa
- vomiting
- tachycardia, arrhythmias, tremors

- seizures

Management:

- activated charcoal
- charcoal hemoperfusion is preferable to hemodialysis

Factors decreasing theophylline clearance

✓ Disease:

- Hepatic cirrhosis
- COPD
- Acute febrile illnesses, Pneumonia
- Congestive cardiac failure
- Acute pulmonary edema

✓ Drugs: Cimetidine, Oral contraceptive pill, Erythromycin, Ciprofloxacin

✓ Diet: Obesity, High carbohydrate intake, High methylxanthine intake (tea, coffee)

Factors increasing theophylline clearance

✓ Diet: Low carbohydrate, High protein intake

✓ Social: Cigarette smoking

✓ Drugs: Rifampicin, Carbamazepine.

Chronic Obstructive Pulmonary Disease (COPD)

Causes:

✓ Smoking!

✓ Alpha-1 antitrypsin deficiency

Other causes: 4 C+ grain

- cadmium (used in smelting)
- coal
- cotton
- cement
- grain

Diagnosis:

✓ NICE recommend considering a diagnosis of COPD in patients over 35 years of age who are smokers or ex-smokers and have symptoms such as exertion breathlessness, chronic cough or regular sputum production.

A. The following investigations are recommended in patients with suspected COPD: post-bronchodilator spirometry to demonstrate airflow obstruction:
   ⇒ FEV1/FVC ratio less than 70% (normal 75- 80%)
B. Chest x-ray:
   ⇒ Hyperinflation, bullae, flat hemidiaphragm.
   ⇒ Also, important to exclude lung cancer
C. full blood count: exclude secondary polycythaemia
D. body mass index (BMI) calculation

The severity of COPD is categorized using the FEV1*

Post-bronchodilator FEV1/FVC	FEV1 (of predicted)	Severity
< 0.7	> 80%	Stage 1 - Mild**
< 0.7	50-79%	Stage 2 - Moderate
< 0.7	30-49%	Stage 3 - Severe
< 0.7	< 30%	Stage 4 - Very severe

Measuring **peak expiratory flow** is of limited value in COPD, as it may underestimate the degree of airflow obstruction.

*note that the grading system has changed following the 2010 NICE guidelines. If the FEV1 is greater than 80% predicted but the post-bronchodilator FEV1/FVC is < 0.7 then this is classified as Stage 1 - mild
**symptoms should be present to diagnose COPD in these patients

Management of stable COPD

NICE updated its guidelines on the management of chronic obstructive pulmonary disease (COPD) in 2010.
General management:

- ✓ smoking cessation advice
- ✓ annual influenza vaccination
- ✓ one-off pneumococcal vaccination

Bronchodilator therapy:
A short-acting beta2-agonist (SABA) or short-acting muscarinic antagonist (SAMA) is first-line treatment for patients who remain breathless or have exacerbations despite using short-acting bronchodilators the next step is determined by the FEV1:

- ✓ FEV1 > 50% (Mild & Moderate COPD)
  - o long-acting beta2-agonist (LABA), for example salmeterol
  - o or long-acting muscarinic antagonist (LAMA), for example tiotropium
- ✓ FEV1 <50 % (Severe& Very severe COPD)
  - o LABA + inhaled corticosteroid (ICS) in a combination inhaler
  - o Or LAMA
- ✓ For patients with persistent exacerbations or breathlessness
  - o if taking a LABA then switch to a LABA + ICS combination inhaler
  - o Otherwise give a LAMA and a LABA + ICS combination inhaler

Reason for using inhaled corticosteroids –To reduce exacerbations

Oral theophylline:

- ✓ NICE only recommends theophylline after trials of short and long-acting bronchodilators or to people who cannot used inhaled therapy.
- ✓ the dose should be reduced if macrolide or fluoroquinolone antibiotics are co-prescribed.

Mucolytics:

- ✓ Should be considered in patients with a chronic productive cough and continued if symptoms improve

Factors which may improve survival in patients with stable COPD:

- ✓ smoking cessation - the single most important intervention in patients who are still smoking
- ✓ long term oxygen therapy (LTOT) in patients who fit criteria
- ✓ lung volume reduction surgery in selected patients

Management of acute COPD exacerbations:

A) The most common bacterial organisms that cause infective exacerbations of COPD are:
   - Hemophilus influenza (most common cause)
   - Streptococcus pneumoniae
   - Moraxella catarrhalis

B) Respiratory viruses account for around 30% of exacerbations, with the human rhinovirus being the most important pathogen.

NICE guidelines from 2010 recommend the following:

- ✓ increase frequency of bronchodilator use and consider giving via a nebulizer
- ✓ give prednisolone 30 mg daily for 7-14 days
- ✓ It is common practice for all patients with an exacerbation of COPD to receive antibiotics. NICE do not support this approach. They recommend giving oral antibiotics 'if sputum is purulent or there are clinical signs of pneumonia'

Long-term oxygen therapy in COPD:

- The 2010 NICE guidelines on COPD clearly define which patients should be assessed for and offered long-term oxygen therapy (**LTOT**).
- Patients who receive LTOT should breathe supplementary oxygen for **at least 15 hours/day**.
- Oxygen concentrators are used to provide a fixed supply for LTOT.
- Assess patients if **any** of the following:
  - **Very severe** airflow obstruction (**FEV1 < 30%** predicted). Assessment should be 'considered' for patients with **severe** airflow obstruction (FEV1 **30-49%** predicted)
  - cyanosis
  - oxygen **saturations** less than or equal to **92%** on room air
  - polycythaemia
  - peripheral edema
  - raised jugular venous pressure
- Assessment is done by measuring **ABG** on **2 occasions at least 3 weeks apart** in patients with **stable COPD** on **optimal management**.

Offer LTOT to patients with:

- ✓ **a pO2 < 7.3 (55)**or to
- ✓ those with a **pO2 of 7.3 - 8(55-60)**and **one** of the following:
  - secondary polycythaemia
  - nocturnal hypoxemia
  - peripheral edema
  - pulmonary hypertension

LTOT and smoking cessation are currently the only interventions in COPD that have been shown to prolong life.

Cor pulmonale

Features:

- peripheral edema,
- raised jugular venous pressure,
- systolic parasternal heave,
- loud P2

✓ use a loop diuretic for edema, consider long-term oxygen therapy

✓ ~~ACE-inhibitors~~, ~~calcium-channel blockers~~ and ~~alpha blockers~~ are not recommended by NICE

Alpha-1 antitrypsin deficiency

- A common inherited condition caused by **a lack of a protease inhibitor** (**Pi**) normally produced by the liver.
- The role of A1AT is to protect cells from enzymes such as **neutrophil elastase**.

Genetics

- ✓ located on chromosome 14
- ✓ inherited in an autosomal recessive / co-dominant fashion*
- ✓ alleles classified by their electrophoretic mobility: M for normal, S for slow, and Z for very slow:
  - ⇒ normal = PiMM
  - ⇒ homozygous PiSS (50% normal A1AT levels)
  - ⇒ homozygous PiZZ (10% normal A1AT levels)

Features:

- ✓ patients who manifest disease usually have PiZZ genotype
- ✓ lungs: panacinar emphysema, most marked in lower lobes
- ✓ liver:
  - cirrhosis and hepatocellular carcinoma in adults,
  - cholestasis in children

Investigations: A1AT concentrations

Management:

- ✓ no smoking
- ✓ supportive: bronchodilators, physiotherapy
- ✓ intravenous alpha1-antitrypsin protein concentrates
- ✓ surgery:

- volume reduction surgery, lung transplantation*trusted sources are split on which is a more accurate description

Obstructive sleep apnoea/hypopnoea syndrome

Predisposing factors:

- ✓ obesity
- ✓ macroglossia: acromegaly, hypothyroidism, amyloidosis
- ✓ large tonsils
- ✓ Marfan's syndrome

Consequence:

- daytime somnolence
- hypertension

SIGN guidelines for the diagnosis and management of patients with OSAHS were published in 2003

Assessment of sleepiness:

- ✓ Epworth Sleepiness Scale - questionnaire completed by patient +/- partner
- ✓ Multiple Sleep Latency Test (MSLT) - measures the time to fall asleep in a dark room (using EEG criteria)

Diagnostic tests:

Sleep studies:

Ranging from:

- ⇒ monitoring of pulse oximetry at night to
- ⇒ full polysomnography where a wide variety of physiological factors are measured including EEG, respiratory airflow, thoraco-abdominal movement, snoring and pulse oximetry

Management:

- ✓ weight loss, avoid alcohol excess & sedatives
- ✓ CPAP is first line for moderate or severe OSAHS
- ✓ intra-oral devices (e.g. mandibular advancement) may be used if CPAP is not tolerated or for patients with mild OSAHS where there is no daytime sleepiness
- ✓ limited evidence to support use of pharmacological agents

The **severity** of obstructive sleep apnoea/hypopnoea syndrome is dependent on the patient's **symptoms** but generally an **apnoea/ hypopnoea index** (**AHI**):

⇒ **4-14mild**

⇒ **15-30 moderate**

⇒ **>30 severe**.

DVLA must be involved.

Apnea---> cessation of airflow for > 10 seconds) and

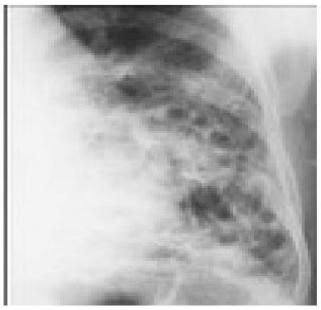
hypopnoea (50% reduction in airflow >10 seconds)

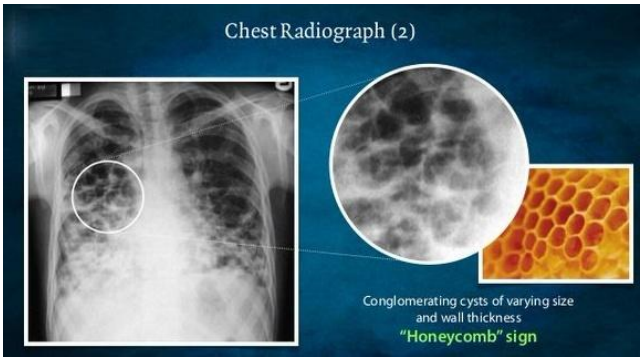
Bronchiectasis

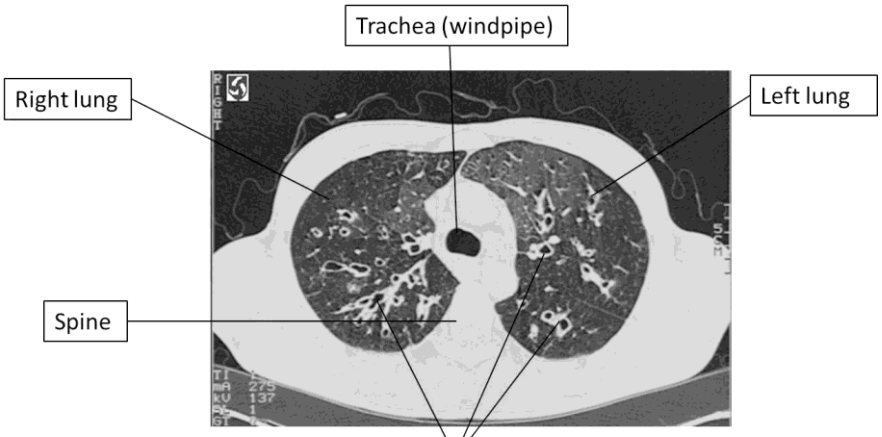
- Bronchiectasis describes a permanent dilatation of the airways secondary to chronic infection or inflammation.
- There are a wide variety of causes are listed below:
  Causes:
  - ✓ post-infective: tuberculosis, measles, pertussis, pneumonia
  - ✓ allergic bronchopulmonary aspergillosis (ABPA)
  - ✓ immune deficiency: selective IgA, hypogammaglobulinaemia
  - ✓ bronchial obstruction e.g. lung cancer/foreign body
  - ✓ cystic fibrosis
  - ✓ ciliary dyskinetic syndromes: Kartagener's syndrome, Young's syndrome
  - ✓ yellow nail syndrome (lymphoedema, pleural effusion & yellow nail)

Radiological features of Bronchiectasis

Bronchi, bronchioles are dilated the bronchial walls are thickened and shown as ring shadows on plain x ray.







Management:
After assessing for treatable causes (e.g. immune deficiency) management is as follows:
physical training (e.g. inspiratory muscle training) - has a good evidence base for patients with non-cystic fibrosis bronchiectasis

- ✓ postural drainage
- ✓ antibiotics for exacerbations + long-term rotating antibiotics in severe cases
- ✓ bronchodilators in selected cases
- ✓ immunizations
- ✓ surgery in selected cases (e.g. Localized disease)

Most common organisms isolated from patients with bronchiectasis:

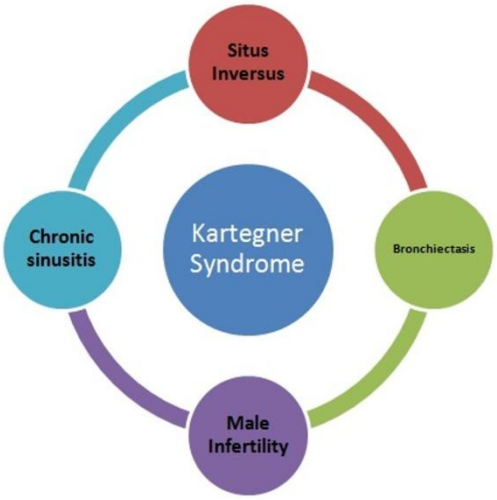
- ✓ Hemophilus influenza (most common)
- ✓ Pseudomonas aeruginosa
- ✓ Klebsiella spp.
- ✓ Streptococcus pneumoniae

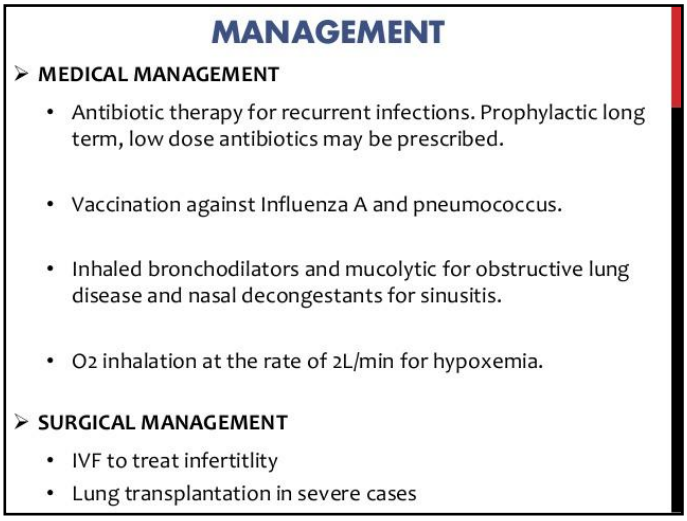
Kartagener's syndrome

- ✓ Also, known as primary ciliary dyskinesia
- ✓ First described in 1933
- ✓ Most frequently occurs in examinations due to its association with dextrocardia (e.g. 'quiet Heart sounds', 'small volume complexes in lateral leads')

Features:

- ✓ dextrocardia or complete situs inversus
- ✓ bronchiectasis
- ✓ recurrent sinusitis
- ✓ subfertility (secondary to diminished sperm motility and defective ciliary action in the fallopian tubes)



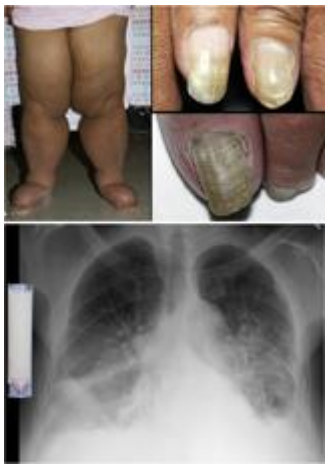


Yellow nail syndrome

Caused by hypoplastic lymphatics and is characterized by the triad of:

- ✓ Lymphoedema
- ✓ Pleural effusions, and
- ✓ Yellow discoloration of the nails.

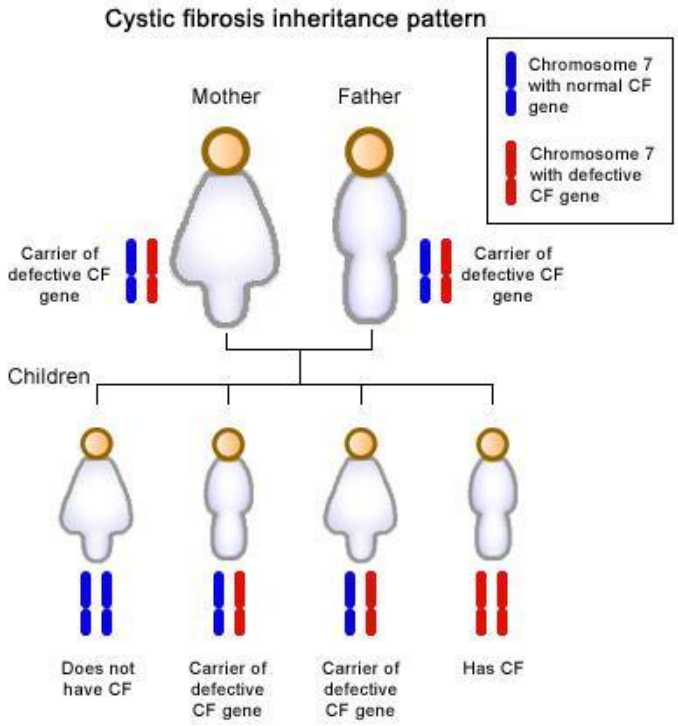
Approximately 40% of patients also have bronchiectasis.

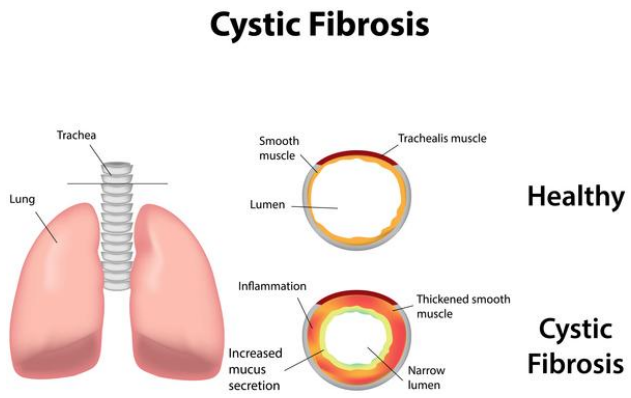


Cystic fibrosis

Autosomal recessive (mucoviscidosis)
Presenting features:

- ✓ Neonatal period (around 20%):
  - meconium ileus,
  - less commonly prolonged jaundice
- ✓ Recurrent chest infections (40%) (pseudomonas)
- ✓ malabsorption (30%): steatorrhoea, failure to thrive
- ✓ other features (10%): liver disease





Other features of cystic fibrosis:

- ✓ short stature
- ✓ delayed puberty
- ✓ diabetes mellitus
- ✓ rectal prolapse (due to bulky stools)
- ✓ nasal polyps
- ✓ Male infertility (as the vas deferens fails to develop).
- ✓ female subfertility

Diagnosis:

- ✓ Sweat test:
  Patients with CF have abnormally high sweat chloride& sodium
  - ⇒ normal value < 40 mEq/l,
  - ⇒ CF indicated by > 60 mEq/l
- ✓ The diagnosis is usually confirmed by determining the patient's genotype.

Causes of false positive sweat test:

- ✓ malnutrition
- ✓ glycogen storage diseases
- ✓ G6PD
- ✓ adrenal insufficiency
- ✓ nephrogenic diabetes insipidus
- ✓ hypothyroidism, hypoparathyroidism
- ✓ ectodermal dysplasia

Management:
Management of cystic fibrosis involves a multidisciplinary approach
Key points:

- ✓ Regular (at least twice daily) chest physiotherapy and postural drainage. Parents are usually taught to do this. Deep breathing exercises are also useful

- ✓ High calorie diet, including high fat intake*
- ✓ pancreatic enzyme supplements taken with meals& vitamin supplementation
- ✓ heart and lung transplant

*this is now the standard recommendation - previously high calorie, low-fat diets have been recommended to reduce the amount of steatorrhoea

Acute respiratory distress syndrome (ARDS)

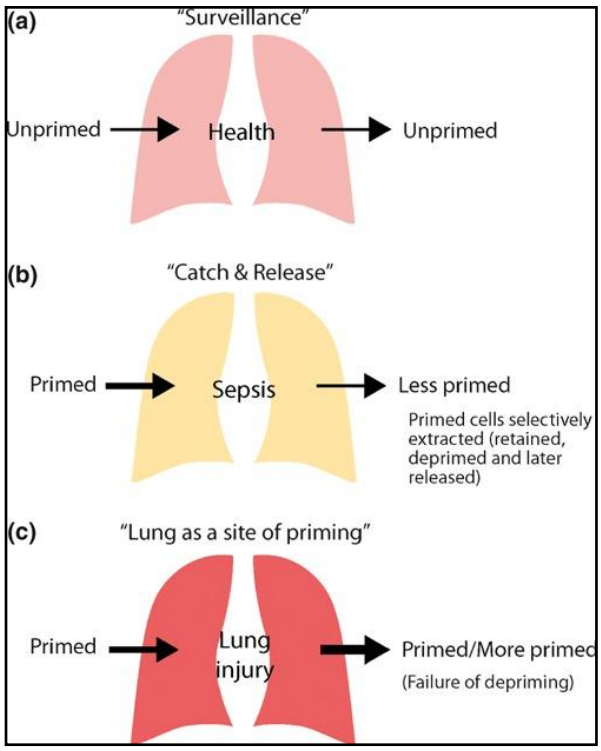
Caused by increased permeability of alveolar capillaries leading to fluid accumulation in alveoli i.e. non-cardiogenic pulmonary edema

Criteria (American-European Consensus Conference)

- ✓ acute onset
- ✓ bilateral infiltrates on CXR
- ✓ non-cardiogenic (pulmonary artery wedge pressure needed if doubt)
- ✓ pO2/FiO2 < 200 mmHg

Causes:

- ✓ infection: sepsis, pneumonia
- ✓ massive blood transfusion
- ✓ trauma
- ✓ smoke inhalation
- ✓ pancreatitis
- ✓ cardio-pulmonary bypass



ARDS in sepsis

- ARDS is a common complication of severe sepsis.
- The ARDS net guidelines feature prominently in the Surviving Sepsis guidelines, with a special emphasis on factors that are important in severe sepsis.
- The target tidal volume is based on ideal, rather than actual body weight. Fat has no alveoli! A target tidal volume of 6 ml/kg ideal body weight should be set maintaining plateau pressures of less than 30 cmH_2O.
- A high-PEEP strategy is recommended to reduce atelecto-trauma.
- Turning the patient prone and recruitment maneuvers are recommended for worsening hypoxaemia.
- Pulmonary artery catheters should not be used routinely and a conservative fluid strategy should be used where possible.
- Non-invasive ventilation (NIV) should not be routinely used and only in carefully considered in a minority of cases.

Community-acquired Pneumonia

CAP may be caused by the following infectious agents:

- ✓ Streptococcus pneumoniae (accounts for around 80% of cases)
- ✓ Haemophilus influenza
- ✓ Staphylococcus aureus: commonly after the 'flu
- ✓ Atypical pneumonias (e.g. Due to Mycoplasma pneumoniae)

- ✓ Viruses
- ✓ Klebsiella pneumoniae is classically in alcoholics

Streptococcus pneumoniae (pneumococcus)

- The most common cause of community-acquired pneumonia
- Characteristic features of pneumococcal pneumonia:
  - ✓ rapid onset
  - ✓ high fever
  - ✓ pleuritic chest pain
  - ✓ herpes labialis

Pneumonia prognostic factors:

CURB-65 criteria of severe pneumonia

- ✓ **Confusion** (abbreviated mental test score <= 8/10)
- ✓ **Urea**> 7 mmol/L
- ✓ **Respiratory rate**>= 30 / min
- ✓ **BP:** systolic <= **90** or diastolic <= **60** mmHg
- ✓ **age**>= 65 years
- **Low** severity: ----------CURB-65 **0-1**, --------mortality **<3%**
- **Moderate** severity: ----CURB-65 **2,** ----------mortality **9%**
- **High severity**: ----------CURB-65 **3-5**, --------mortality **15-40%**

Other factors associated with a poor prognosis include:

- ✓ presence of coexisting disease
- ✓ hypoxemia (pO_2 < 8 = 60) independent of FiO_2
- ✓ Temperature less than 35°C or more than 40°C.
- ✓ WBC less than 4 ×109/L or greater than 20 ×109/L
- ✓ Multi-lobar involvement on CXR

Management:

The British Thoracic Society published guidelines in 2009:

A) **Low Severity CAP:**→Oral amoxicillin alone whether treated at home or in hospital.

B) **Moderate CAP:** →amoxicillin **+** clarithromycin

- If the oral route is not possible, benzylpenicillin and clarithromycin should be used.
- Doxycycline may be used as an alternative antibiotic regime, but is not the preferred treatment

C) **high severity CAP:**

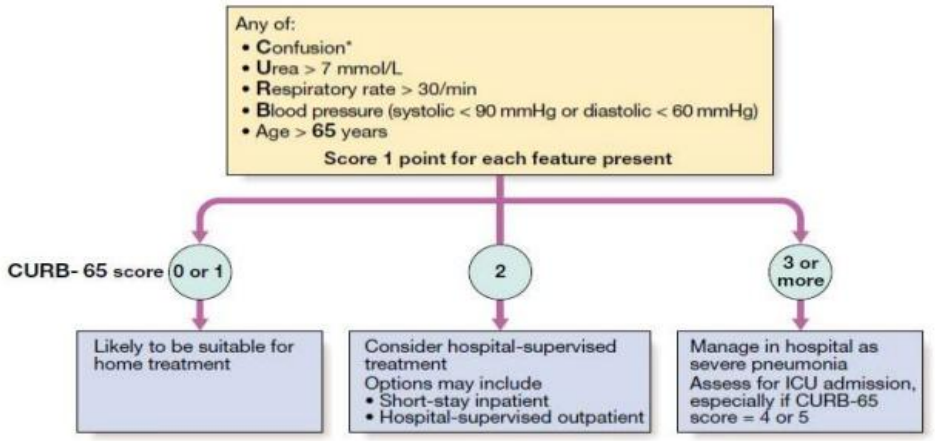
1) Intravenous co-amoxiclav + clarithromycin **OR**
2) Cefuroxime (Zinacef) + clarithromycin **OR**
3) Cefotaxime (Claforan) + clarithromycin

The current BNF has slightly different recommendations

A) For high severity CAP:
   1) Intravenous **Benzylpenicillin** + clarithromycin OR
   2) Intravenous **Benzylpenicillin** + doxycycline.

B) For 'life-threatening' infections the same as BTS guidelines for high-severity CAP

Treatment plain according to -CURB-65



Hospital CURB-65. *Defined as a Mental Test Score of 8 or less, or new disorientation in person, place or time. (A urea of 7 mmol/L ≅ 20 mg/dL.)

Mycoplasma pneumoniae

- Mycoplasma pneumoniae is a cause of **atypical pneumonia**
- Often affects **younger patients** (15-30 yrs).
- It is associated with a number of characteristic complications such as:
    1) **erythema multiforme** and
    2) **Cold autoimmune haemolytic anaemia.**
- **Epidemics** of Mycoplasma pneumoniae classically occur every **4 years**.
- It is important to recognize atypical pneumonias as **they may not respond** to penicillins or cephalosporins due to it **lacking** a **peptidoglycan cell wall.**

Features:

- ✓ the disease typically has a prolonged and gradual onset
- ✓ flu-like symptoms classically precede a dry cough
- ✓ bilateral consolidation on x-ray
- ✓ complications (10%) may occur as below

Complications:

- ✓ cold agglutins (IgM) may cause an **hemolytic anemia**, **thrombocytopenia**
- ✓ **erythema multiforme**, erythema nodosum
- ✓ meningoencephalitis, meningism,**Guillain-Barre syndrome**
- ✓ **bullous myringitis:** painful vesicles on the tympanic membrane
- ✓ pericarditis/**myocarditis**
- ✓ gastrointestinal: hepatitis, pancreatitis
- ✓ renal: **acute glomerulonephritis**

Investigations:

1) diagnosis is generally by **Mycoplasma serology**
   Diagnosis is based on demonstration of anti-mycoplasma antibodies in paired sera.
2) positive **cold agglutination test**

Management:

1) **First choice treatment is with macrolide antibiotic (erythromycin/clarithromycin)**
2) Tetracyclines such as **doxycycline** are an alternative

Legionella

- Legionnaire's disease is caused by the **intracellular bacterium Legionella pneumophilia**.
- Gram negative rod
- It is typically colonizes water tanks and hence questions may hint at air-conditioning systems or foreign holidays.
- Person-to-person transmission is not seen

Features:

- ✓ flu-like symptoms including fever (present in > 95% of patients)
- ✓ dry cough
- ✓ relative bradycardia (also in Typhoid)
- ✓ confusion
- ✓ lymphopaenia
- ✓ hyponatraemia (SIADH), renal failure
- ✓ deranged liver function tests
- ✓ proteinuria
- ✓ pleural effusion: seen in around 30% of patients

Progresses to bilateral involvement in 50% of cases

Diagnosis:

- Urinary antigen

Management:

1) Monotherapy:
   - ⇒ The newer quinolones (especially levofloxacin) and the newer macrolides (especially azithromycin) are effective for treating legionellosis.
   - ⇒ In comparison with erythromycin, they are more potent, have better tissue penetration and significantly less gastrointestinal toxicity.
2) Rifampin combined with erythromycin, combination therapy is now only recommended in patients who are failing standard therapy.

Psittacosis (parrot fever)

- A zoonotic disease caused by **Chlamydia psittaci**
- Contracted from parrots cockatiels and budgerigars, and pigeons,sparrows, ducks, hens, gulls and many other species of bird.
- IP of **5–19 days**,
- The symptoms of the disease range from in apparent illness to systemic illness with severe atypical pneumonia.
- High fevers, joint pains, diarrhea, conjunctivitis, **nose bleeds**.
- **Spleen enlargement** is common.

Complications:

- ✓ endocarditis, myocarditis
- ✓ hepatitis, , arthritis,
- ✓ keratoconjunctivitis, and encephalitis may occasionally occur.

Diagnosis:

- ✓ Can be suspected if respiratory infection with splenomegaly and/or epistaxis.
- ✓ X-rays show patchy infiltrates or a diffuse whiteout of lung fields.
- ✓ Leukopenia, thrombocytopenia and moderately elevated liver enzymes.
- ✓ Culture from respiratory secretions or increase in antibody titers against C. psittaci .
- ✓ Typical inclusions within macrophages in BAL.

Treatment:

Tetracyclines & chloramphenicol are the drugs of choice at least 10–14 days after fever abates

Pneumocystis jiroveci pneumonia

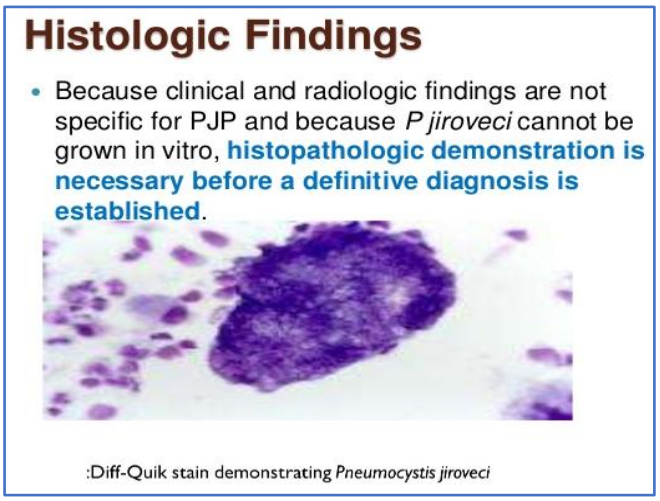
- ✓ The term Pneumocystis carinii pneumonia (PCP) is still in common use
- ✓ Pneumocystis jiroveci is an unicellular eukaryote, generally classified as a fungus but some authorities consider it a protozoa
- ✓ PCP is the most common opportunistic infection in AIDS
- ✓ All patients with a CD4 count < 200/mm should receive PCP prophylaxis & pre CLL chemotherapy with fludarabin (as any of purine analogues).

Features:

- ✓ Dyspnoea, exercise induced desaturation.
- ✓ dry cough
- ✓ fever
- ✓ very few chest signs
- ✓ Pneumothorax is a common complication of PCP.

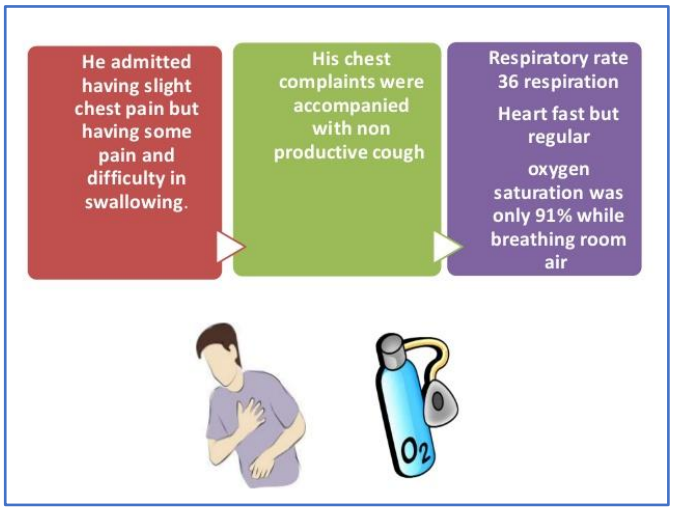
Extrapulmonary manifestations are rare (1-2% of cases), may cause

- ✓ hepatosplenomegaly
- ✓ lymphadenopathy
- ✓ choroid lesions



Investigation:

- ✓ CXR:
  - Typically shows bilateral interstitial pulmonary infiltrates but
  - Can present with other x-ray findings e.g. lobar consolidation.
  - May be normal
- ✓ sputum often fails to show PCP,
- ✓ Definitive diagnosis is by bronchial alveolar lavage (BAL) with silver staining
  (Silver stain shows characteristic cysts)



Management:

- ✓ co-trimoxazole
- ✓ If allergic to co-trimoxazole alternative therapy would be IV pentamidine or clindamycin with primaquine.
- ✓ IV pentamidine in severe cases
- ✓ steroids if hypoxic (if pO_2 < 9.3 (70)
  - steroids reduce risk of respiratory failure by 50% and death by a third
  - It is important that steroids be started right away if indicated, because their purpose is to keep people stable during those first few days of treatment.
  - long term steroid is immunosuppressive but 21 day tapering course has been shown to be safe and effective

Patients often deteriorate after starting therapy for PCP as the pneumonitis worsens due to the inflammation associated with dying pneumocysts.
Oral prednisolone is added to reduce the inflammatory effect.

Treatment

Mild to Moderate	Moderate to Sever
Preferred Therapy: • TMP-SMX: (TMP 15–20 mg/kg/day and SMX 75–100 mg/kg/day), given PO in 3 divided doses *or* • TMP-SMX DS - 2 tablets TID . *Alternative Therapy:* • Dapsone 100 mg PO daily + TMP 15 mg/kg/day PO (3 divided doses) *or* • Primaquine 30 mg (base) PO daily + Clindamycin PO (300 mg q6h or 450 mg q8h) *or* • Atovaquone 750 mg PO BID with food	*Preferred Therapy:* **TMP-SMX** : (TMP 15–20 mg and SMX 75–100 mg)/kg/day IV given q6h or q8h , may switch to PO after clinical improvement . *Alternative Therapy:* • **Pentamidine** 4 mg/kg IV once daily infused over at least 60 minutes ; may reduce the dose to 3 mg/kg IV once daily because of toxicities *or* • **Primaquine** 30 mg (base) PO once daily + (Clindamycin [IV 600 q6h or 900 mg q8h] or [PO 300 mg q6h or 450 mg q8h]). • Adjunctive corticosteroid may be indicated in some moderate to severe cases

For Moderate to Severe PCP Based on the Following Criteria :

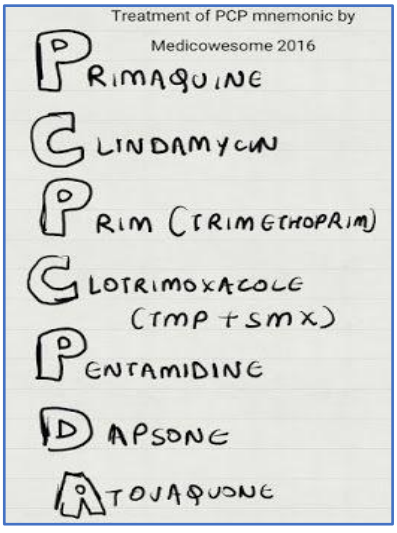
- PaO_2 <70 mmHg at room air *or*
- Alveolar-arterial O_2 gradient ≥35 mmHg

Prednisone doses (beginning as early as possible and within 72 hours of PCP therapy)

Schedule	Dosage
Days 1 to 5	40 mg of prednisone **twice** daily
Days 6 to 10	40 mg of prednisone once daily
Days 11 to 21	20 mg of prednisone once daily*

* No further tapering is necessary.

- ➢ **IV methylprednisolone can be given as 75% of prednisone dose**
- ➢ **The risk of reactivating tuberculosis or acquiring another infection appears to be minimal.**



-IV pentamidine-------->Trypanosuma (sleeping sickness) or Suramineor Meralsoprol (late)

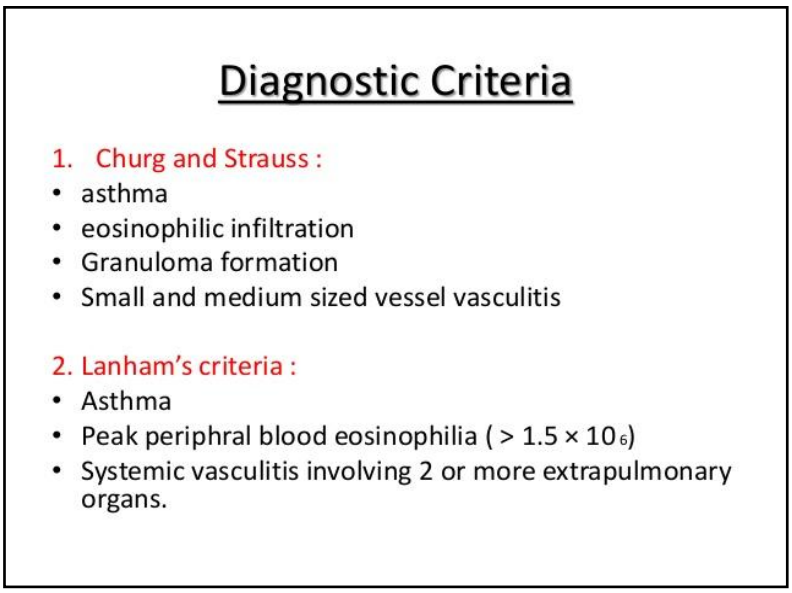
Churg-Strauss syndrome

ANCA associated small-medium vessel vasculitis.

Features:

- ✓ asthma
- ✓ paranasal sinusitis
- ✓ Blood eosinophilia (e.g. > 10%)
- ✓ mononeuritis multiplex
- ✓ pANCA positive in 60%

Leukotriene receptor antagonists may precipitate the disease



Goodpasture's syndrome

- ✓ Goodpasture's syndrome is rare condition
- ✓ Associated with:
  - **Pulmonary hemorrhage** and
  - **Rapidlyprogressive glomerulonephritis**.
- ✓ It is caused by **anti-glomerular basement membrane** (anti-GBM) antibodies against type **IV collagen**.
- ✓ Goodpasture's syndrome is more common in **men** (sex ratio 2:1)
- ✓ It has a **bimodal age** distribution (peaks in **20-30** and **60-70** age bracket).
- ✓ It is associated with **HLA DR2**.

Features:

- ✓ pulmonary hemorrhage
- ✓ followed by **RPGN** rapidly progressive glomerulonephritis

Factors which increase likelihood of pulmonary hemorrhage:

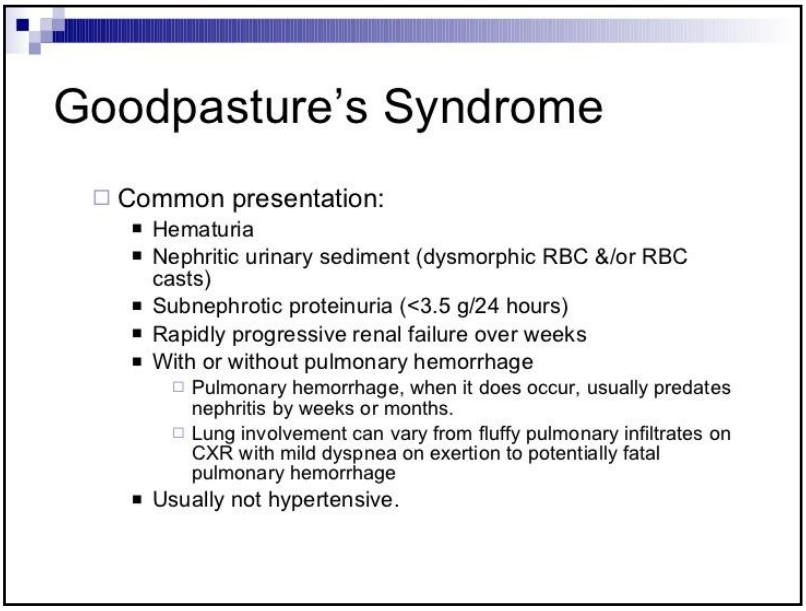
- ✓ young males
- ✓ smoking
- ✓ inhalation of hydrocarbons
- ✓ lower respiratory tract infection
- ✓ pulmonary edema

Investigations:

- ✓ renal biopsy: linear IgG deposits along BM
- ✓ raised transfer factor secondary to pulmonary hemorrhages

Management:

- ✓ plasma exchange
- ✓ steroids
- ✓ cyclophosphamide

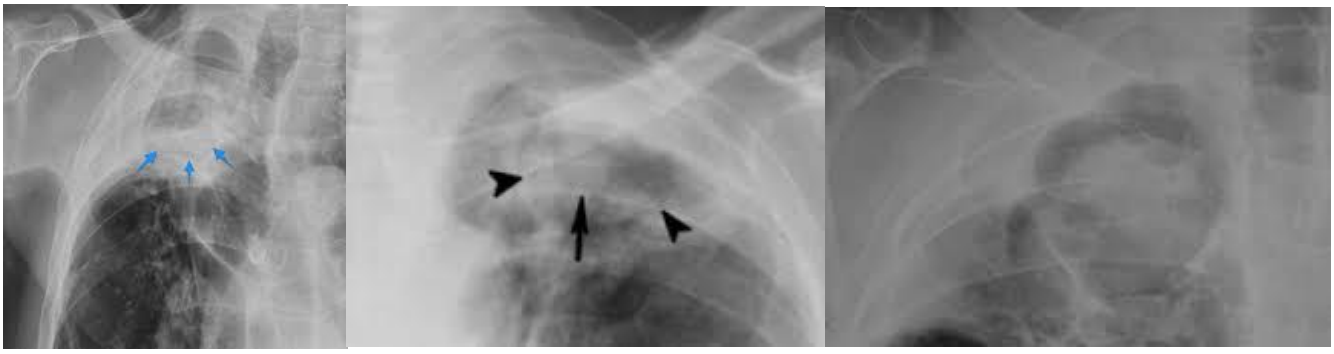


Aspergilloma

- ✓ An aspergilloma is a **mycetoma** (mass-like **fungus ball**) which often **colonizes an existing lung cavity** (e.g. **secondary** to tuberculosis, lung cancer or cystic fibrosis)
- ✓ Usually **asymptomatic** but features may include:
  - **cough**
  - **hemoptysis** (may be severe)

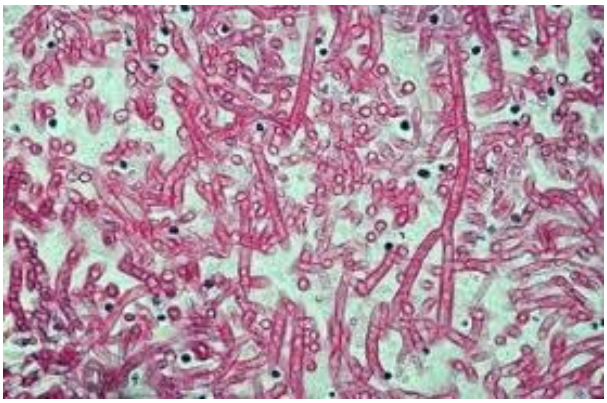
Investigations:

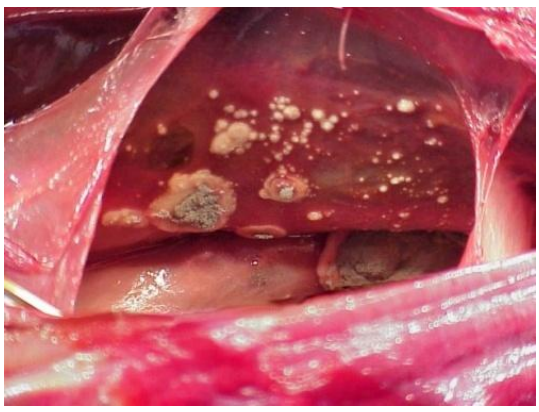
- ✓ **chest x-ray: a solidopacity within a cavity often associated with a rim of air.**
- ✓ **high titers IgG Aspergillus precipitins (95%)**



Aspergillosis

- ✓ a fungal infection, and develops mainly in immunocompromised
- ✓ It is a leading cause of death in **acute leukaemia** and **hemopoietic stem cell transplantation**.
- ✓ Signs and symptoms include **cough, hemoptysis, chest wall pain, fever and shock**.
- ✓ It is often seen on chest x rays and CT scan, and demonstrates an **air crescent** sign.
- ✓ Other investigations include microscopy and the **galactomannan test**.





Treatment

Intravenous amphotericin B (Note)

Treatment of aspergillosis: clinical practice guidelines of IDSA

Condition	Primary Rx	Alternative Rx
Invasive pulmonary aspergillosis Invasive sinus aspergillosis Tracheobronchial aspergillosis Chronic necrotizing pulmonary aspergillosis (subacute invasive pulmonary aspergillosis) Aspergillosis of the CNS	Voriconazole (6 mg/kg IV every 12 h for 1 day, followed by 4 mg/kg IV every 12 h; oral dosage is 200 mg every 12 h)	L-AMB (3–5 mg/kg/day IV), ABLC (5 mg/kg/day IV), Caspofungin (70 mg day 1 IV and 50 mg/day IV thereafter), Micafungin (IV 100–150 mg/day; dose not established), Posaconazole (200 mg QID initially, then 400 mg BID PO after stabilization of diseased), Itraconazole (dosage depends upon formulation)

Surgical debridement may be indicated

Walsh TJ, Anaissie EJ, Denning DW et al. Treatment of aspergillosis: clinical practice guidelines of the Infectious Diseases Society of America. Clin Infect Dis 2008; 46: 327–60.

Allergic bronchopulmonary aspergillosis

- ✓ Results from an **allergy** to **Aspergillus spores**.
- ✓ In the exam questions often give a history of **bronchiectasis** and **eosinophilia.**

Predisposing conditions
Bronchial asthma, cystic fibrosis
Obligatory criteria
Elevated serum total IgE levels (>1000 IU/ml)
Elevated serum IgG and/or IgE against *A. fumigatus*
Other criteria (at least three of five)
Immediate type I reaction to *A. fumigatus* antigen
Presence of serum *A. fumigatus* precipitins
Transient and/or permanent chest radiographic opacities
Eosinophil count >1000 cells/μl in peripheral blood
Central bronchiectasis on HRCT chest

Features:

- ✓ bronchoconstriction: wheeze, cough, dyspnoea
- ✓ bronchiectasis (proximal)

Investigations:

- ✓ eosinophilia
- ✓ raised IgE
- ✓ positive radioallergosorbent (RAST) test to Aspergillus
- ✓ positive IgG Aspergillus precipitins (not as positive as in aspergilloma)
- ✓ flitting CXR changes
- ✓ CT: Bronchocoeles are a common feature of allergic bronchopulmonary aspergillosis (ABPA)

Diagnostic criteria	Findings in the present case
Major criteria	
Bronchial asthma	-
Type I cutaneous hypersensitivity	+
Eosinophilia	-
Serum total IgE	+
Serum precipitins against *Aspergillus*	-
Specific IgE/IgG against *Aspergillus*	+
Fleeting pulmonary opacities	-*
Central bronchiectasis	+
Minor criteria	
Expectoration of sputum plugs	-
Isolation of fungus from sputum	-
Type III cutaneous hypersensitivity	+

*Previous radiographs of the patient were not available, ABPA - Allergic bronchopulmonary aspergillosis

Management:

- ✓ steroids
- ✓ itraconazole is sometimes introduced as a second line agent

Extrinsic allergic alveolitis (EAA)

Hypersensitivity pneumonitis

- ✓ A condition caused by **hypersensitivity induced lung damage** due to a variety of inhaled organic particles.
- ✓ It is thought to be largely caused by **immune-complex mediated tissue damage** (**type III hypersensitivity**) although **delayed hypersensitivity (type IV)**is also thought to play a role in EAA, especially in the chronic phase.

Examples:

- ✓ **Bird fanciers lung:**
  avian proteins. (It occurs most commonly among pigeon fanciersand budgerigar owners
- ✓ **farmers lung:**
  spores of **Saccharopolyspora rectivirgula** (formerly Micropolyspora faeni)
- ✓ **mushroom workers lung:**
  thermophilic actinomycetes*
- ✓ **Malt workers lung:**
  Aspergillus clavatus

*here the terminology is slightly confusing as thermophilic actinomycetes is an umbrella term covering strains such as Micropolyspora faeni

Malt workers

Presentation

- ✓ **acute:** SOB, dry cough, fever
  Symptoms may develop 4- 6 hrs after exposure to allergen and last 12 hours up to several days following removal of the antigen.
- ✓ **chronic**

Patients present with a progressive dyspnoea on exertion. Inspiratory crackles are heard on auscultation of the lungs. CXR shows fine linear opacities in the upper lobes which may progress to honeycombing. The diagnosis of HP is based on typical clinical, radiological and lung function changes in the presence of an identified source of antigen with positive precipitating antibodies in the patient's serum to the causal antigen.
Improvement of clinical abnormalities following avoidance of the causal antigen helps confirm the diagnosis.

Investigation

- ✓ chest x-ray: **upper/mid-zone fibrosis**
- ✓ bronchoalveolar lavage**BAL**: **lymphocytosis**
- ✓ blood: **NO eosinophilia**

Pulmonary eosinophilia

Causes of pulmonary eosinophilia

- ✓ Churg-Strauss syndrome
- ✓ allergic bronchopulmonary aspergillosis (ABPA)
- ✓ Loffler's syndrome
- ✓ eosinophilic pneumonia
- ✓ hypereosinophilic syndrome
- ✓ tropical pulmonary eosinophilia
- ✓ drugs: nitrofurantoin, sulphonamides
- ✓ less common: Wegener's granulomatosis

Loffler's syndrome

- ✓ thought to be due to parasites such as Ascaris lumbricoides causing an alveolar reaction
- ✓ Presents with a fever, cough and night sweats which often last for less than 2 weeks.
- ✓ generally a self-limiting disease
- ✓ transient CXR shadowing and blood eosinophilia

Tropical pulmonary eosinophilia

- ✓ associated with Wuchereria bancrofti infection

Lung fibrosis

- ✓ It is important in the exam to be able to differentiate between conditions causing predominately upper or lower zone fibrosis.
- ✓ It should be noted that the more common causes (cryptogenic fibrosing alveolitis, drugs) tend to affect the lower zones

Fibrosis predominately affecting the upper zones:

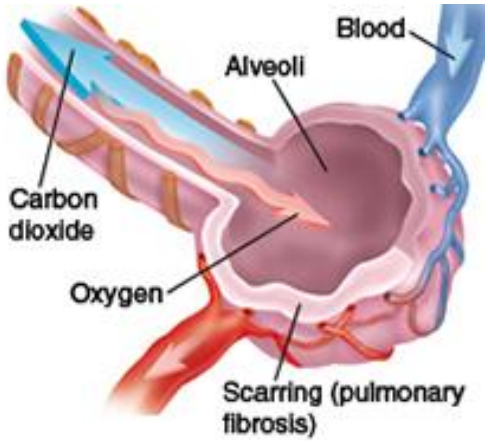
- ✓ extrinsic allergic alveolitis
- ✓ coal worker's pneumoconiosis (progressive massive fibrosis)
- ✓ silicosis
- ✓ sarcoidosis, tuberculosis
- ✓ histiocytosis
- ✓ ankylosing spondylitis (rare)

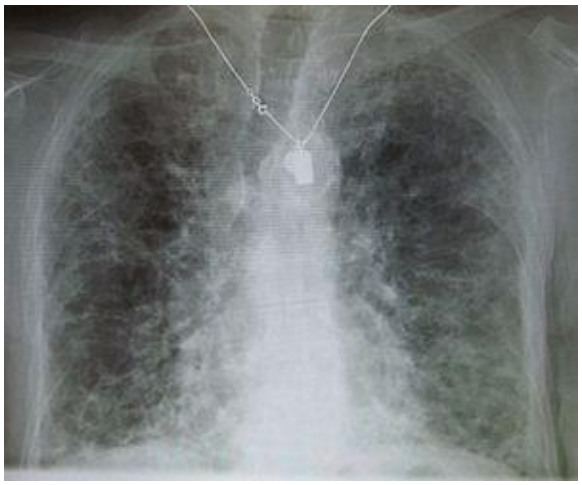
Fibrosis predominately affecting the lower zones:

- ✓ cryptogenic fibrosing Alveolitis (**IPF)**
- ✓ most connective tissue disorders (except ankylosing spondylitis)
- ✓ drug-induced: amiodarone, bleomycin, methotrexate
- ✓ asbestosis

Note: All pathological lesions in the lung ends with [osis] present in the upper lobe except asbestosis in the lower lobe

Silicosis usually results in small nodular opacities in the mid and upper zones. There may be associated hilar gland enlargement which may calcify producing characteristic 'eggshell' calcification. These radiological shadows are not usually associated with symptoms or loss of lung function.





Drugs causing lung fibrosis

- ✓ Amiodarone
- ✓ Cytotoxic agents: busulphan, bleomycin
- ✓ Anti-rheumatoid drugs: methotrexate, sulfasalazine, gold
- ✓ Nitrofurantoin
- ✓ Ergot-derived dopamine receptor agonists (bromocriptine, cabergoline, pergolide)

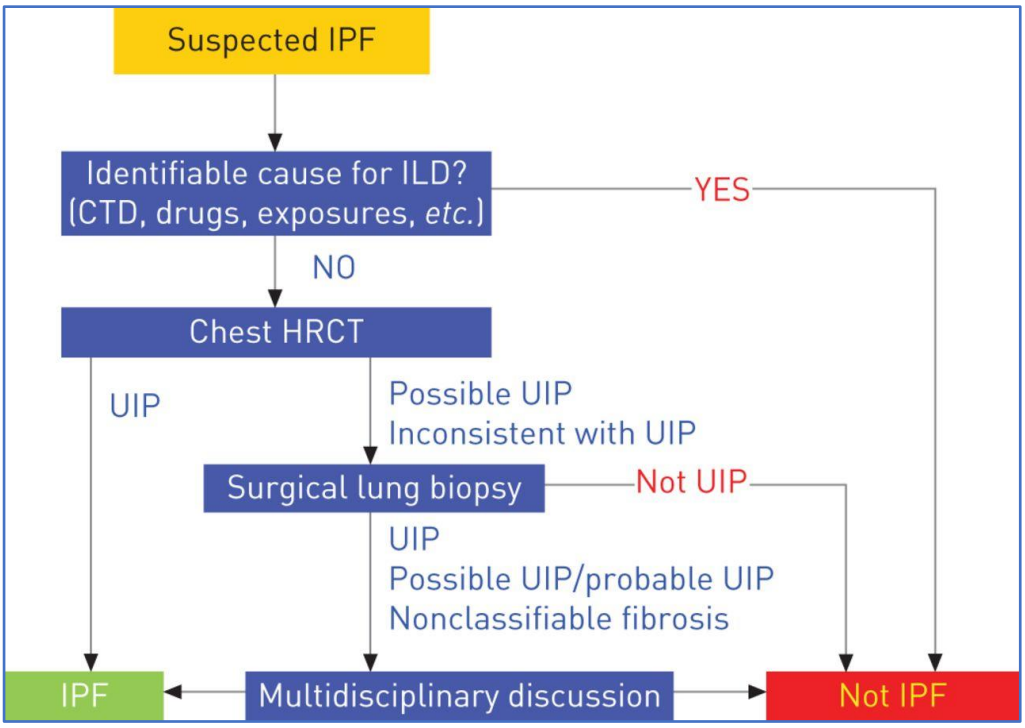
Idiopathic pulmonary fibrosis (IPF)

- ✓ previously termed cryptogenic fibrosing alveolitis
- ✓ A chronic lung condition characterized by progressive fibrosis of the interstitium of the lungs.
- ✓ Whilst there are many causes of lung fibrosis (e.g. medications, connective tissue disease, asbestos) the term IPF is reserved when **no underlying cause** exists.

- ✓ IPF is typically seen in patients aged **50-70** years and is **twice** as common in **men**.

Features:

- ✓ progressive exertion dyspnoea
- ✓ dry cough
- ✓ bibasal crackles on auscultation
- ✓ clubbing



Diagnosis:

- ✓ **spirometry:**
  - Classically a **restrictive** picture

  (FEV1 normal/decreased, FVC decreased, FEV1/FVC increased)
- ✓ **impaired gas exchange:**reduced transfer factor (TLCO)
- ✓ **imaging:**
  - CXR
  - **bilateral interstitial** shadowing

  (Typically small, irregular, peripheral opacities '**ground-glass**'
  Later progressing to '**honeycombing**')
  - **high-resolution CT scanning HRCT** is the **investigation of choice**and required to make a diagnosis of IPF
- ✓ **ANA positive in 30%,rheumatoid factor positive in 10%**but this does not necessarily mean that the fibrosis is secondary to a connective tissue disease. **Titers** are usually **low**

Management:

- ✓ **Pulmonary rehabilitation**
- ✓ Very few medications have been shown to give any benefit in IPF.
  - There is some evidence that **pirfenidone** (an **antifibrotic** agent) may be useful in selected patients
- ✓ many patients will require **supplementary oxygen** and eventually a **lung transplant**

Prognosis:

- poor, average life expectancy is around **3-4 years**

Sarcoidosis

- ✓ Multisystem disorder of unknown etiology characterized by **non-caseating granulomas**.
- ✓ It is more common in **young adults** and in people of **African** descent.
- ✓ Sarcoidosis **remits without treatment** in approximately **two-thirds** of people

Features:

- ✓ **Acute: Lofgren's syndrome:**
  - erythema nodosum,
  - bilateral hilar lymphadenopathy,
  - swinging fever,
  - polyarthralgia (sarcoidosis typically targets the **ankle joint**.)
- ✓ **Insidious**: dyspnoea, non-productive cough, malaise, weight loss
- ✓ **Lupus pernio**erythematous nodular lesions on the cheek, nose, forehead and chin.
- ✓ **Hypercalcaemia**: macrophages inside granulomas cause an increased conversion of **vitamin D** to its active form (1,25-dihydroxycholecalciferol), may lead to **nephropathy**
- ✓ **Cranial nerve palsies**, **Lymphadenopathy and hepatosplenomegaly**

Investigation:

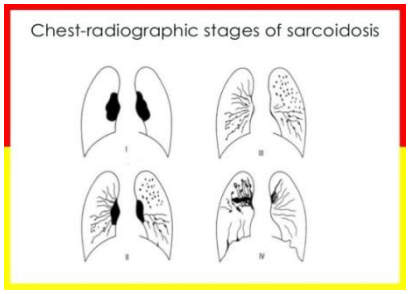
There is no one diagnostic test for sarcoidosis and hence diagnosis is still largely clinical.

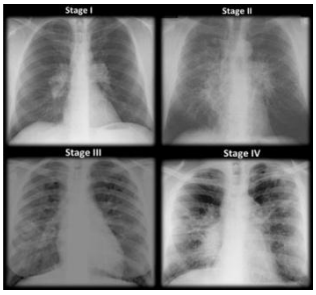
- ✓ **ACE** levels:
  - It has a **sensitivity 60%** and
  - **specificity 70%** and is therefore not reliable in the diagnosis of sarcoidosis although
  - They may have a role in **monitoring disease activity**.
- ✓ Routine bloods may show **hypercalcaemia** (seen in **10%** if patients) and a **raised ESR**
- ✓ **A chest x-ray** may show the following changes:
  - stage 0 = normal
  - stage 1 = bilateral hilar lymphadenopathy (BHL)
  - stage 2 = BHL + interstitial infiltrates
  - stage 3 = diffuse interstitial infiltrates only
  - stage 4 = diffuse fibrosis

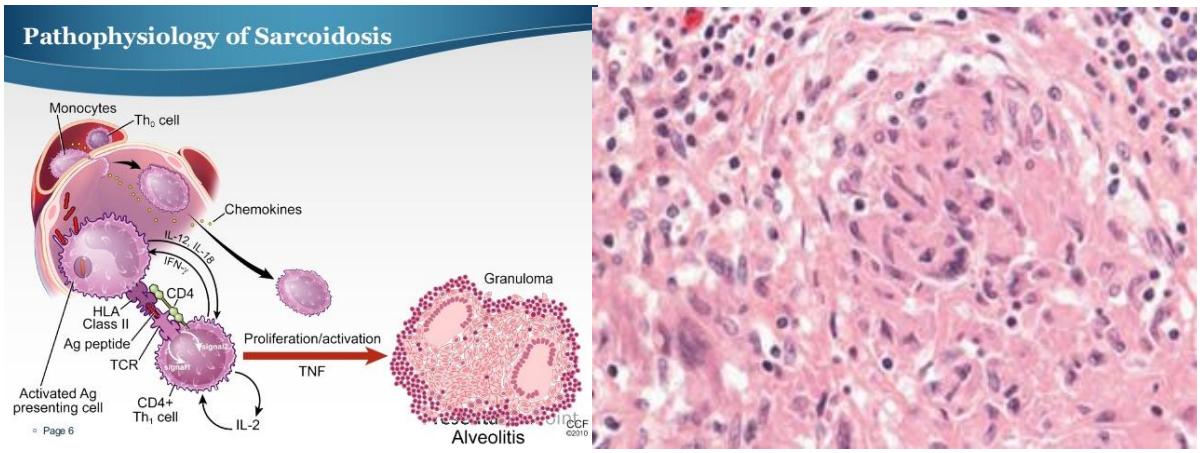
Other investigations*

- ✓ **spirometry:** may show a **restrictive** defect
- ✓ **tissue biopsy: non-caseating granulomas**
- ✓ **gallium-67 scan:** not used routinely

*the **Kveim test** (where part of the spleen from a patient with known sarcoidosis is injected under the skin) is no longer performed due to concerns about cross-infection







Sarcoid granulomas in the heart

Sarcoidosis management:

Sarcoidosis remits without treatment in approximately two-thirds of people

Indications for steroids

- ✓ Patients with chest x-ray **stage 2 or 3 disease** that have moderate to severe or progressive symptoms.
  - Patients with **asymptomatic** and **stable stage 2 or 3 disease** who have only mildly abnormal lung function **do not require treatment**
- ✓ **hypercalcemia**
- ✓ **eye, heart or neuro** involvement

Sarcoidosis prognostic features:

Factors associated with poor prognosis:

- ✓ black people
- ✓ insidious onset, symptoms > 6 months
- ✓ CXR: stage III-IV features
- ✓ extrapulmonary manifestations: e.g. lupus pernio, splenomegaly
- ✓ absence of erythema nodosum

Syndromes associated with sarcoidosis:

Lofgren's syndrome:

- ✓ An acute form sarcoidosis characterised by:
- bilateral hilar lymphadenopathy (BHL),
- erythema nodosum,
- Fever and polyarthralgia.
- ✓ It typically occurs in young females and carries an excellent prognosis.

Mikulicz syndrome:

- ✓ there is enlargement of the **parotid** and **lacrimal glands** due to sarcoidosis, tuberculosis or lymphoma
- ✓ this term is now considered outdated and unhelpful by many as there is a confusing overlap with Sjogren's syndrome

Heerfordt's syndrome (**uveo-parotid fever**)

- ✓ there is **parotid enlargement**,
- ✓ fever and uveitis secondary to sarcoidosis

Bilateral hilar lymphadenopathy

The most common causes of bilateral hilar lymphadenopathy are:

- ✓ sarcoidosis and
- ✓ tuberculosis

Other causes include:

- ✓ lymphoma/other malignancy
- ✓ pneumoconiosis e.g. berylliosis (non-caseating granuloma occupational disease)
- ✓ fungi e.g. histoplasmosis, coccidioidomycosis

Lung cancer

Types:

- ✓ squamous: ---------- 35%
- ✓ adenocarcinoma: -- 30%
- ✓ small (oat) cell: -----15%
- ✓ large cell: ----------10%
- ✓ other c. 5%

Other tumors:

✓ Bronchoalveolar cell carcinoma:

- ✓ not related to smoking,
- ✓ ++sputum,
- ✓ Bronchoalveolar cell carcinoma classically presents with progressive breathlessness and the production of large amounts of sputum (**bronchorrhoea**)
- ✓ Almost a half of patients are diagnosed on routine CXR, usually demonstrating a **peripheral lesion**.
- ✓ Its name arises from its pattern of growth along the alveolar walls without actually destroying them.
- ✓ It is an **adenocarcinoma**.
- ✓ In those whose tumor is not resectable, prognosis is poor.

✓ bronchial adenoma:

- mostly carcinoid

Lung cancer risk factors:

- ✓ Smoking: increases risk of lung ca by a **factor of 10**

Other factors:

- ✓ asbestos - increases risk of lung ca by a **factor of 5**
- ✓ arsenic
- ✓ radon
- ✓ nickel
- ✓ chromate
- ✓ aromatic hydrocarbon
- ✓ cryptogenic fibrosing alveolitis (IPF)

Factors that are NOT related:
Coal dust

Smoking and **asbestos** are **synergistic**, i.e. a smoker with asbestos exposure has a 10 * 5 = **50 times** increased risk

Non-small cell Lung cancer

There are three main subtypes of non-small cell lung cancer:

A) Squamous cell cancer (35%)
- typically central (cavitating lung lesion)
- associated with parathyroid hormone-related protein (PTHrP) secretion → hypercalcaemia
- hyperthyroidism due to ectopic TSH
- strongly associated with finger clubbing
- hypertrophic pulmonary osteoarthropathy (HPOA)

B) Adenocarcinoma (30%)
- most common type of lung cancer in non-smokers, although the majority of patients who develop lung adenocarcinoma are smokers
- typicallylocated on the lung periphery
- gynecomastia

C) Large cell lung carcinoma(10%)

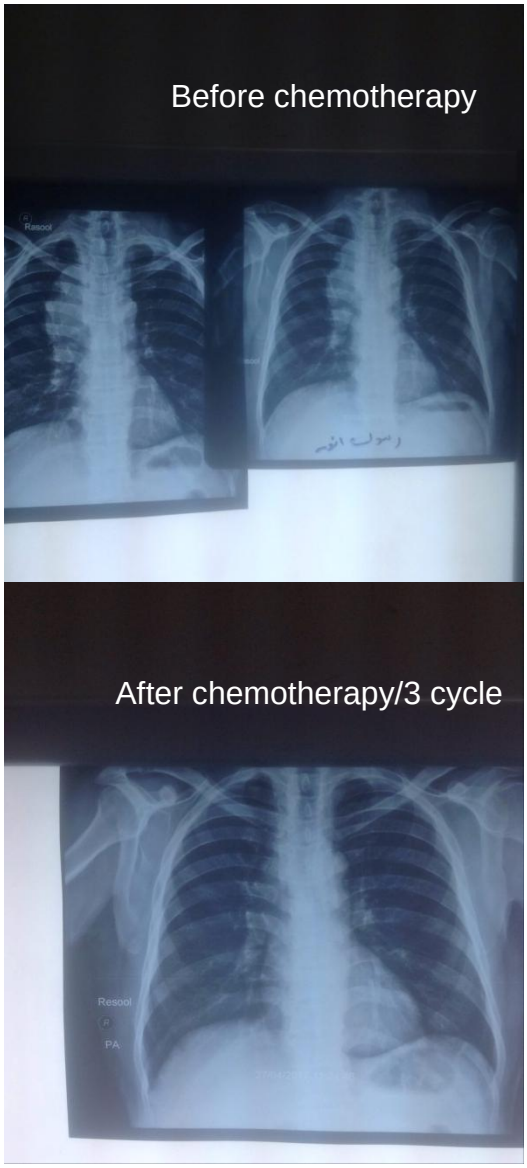
Management of Non-small cell Lung cancer:

- ✓ only 20% suitable for surgery
- ✓ Mediastinoscopy performed prior to surgery as CT does not always show mediastinal lymph node involvement
- ✓ curative or palliative radiotherapy
- ✓ poor response to chemotherapy

Surgery contraindications:

1) assess general health
2) stage IIIb or IV (i.e. metastases present)
3) FEV1 < 1.5 liters is considered a general cut-off point*
4) malignant pleural effusion
5) tumor near hilum
6) vocal cord paralysis
7) SVC obstruction

* However, if FEV1 < 1.5 for lobectomy or < 2.0 for pneumonectomy then some authorities advocate further lung function tests as operations may still go ahead based on the results



Small Cell Lung Cancer (15%)

Features:

- ✓ usually **central**
- ✓ arise from **APUDcells** (Amine Precursor Uptake Decarboxylase)
- ✓ associated with ectopic **ADH**, **ACTH** secretion
  - ADH → hyponatraemia
  - ACTH → Cushing's syndrome
  - ACTH secretion can cause:
    - bilateral adrenal hyperplasia,
    - the high levels of cortisol can lead to **hypokalemic alkalosis**
- ✓ **Lambert-Eaton syndrome:** antibodies to voltage gated calcium channels causing myasthenic like syndrome

*an acronym for:

- Amine - high amine content
- Precursor Uptake - high uptake of amine precursors
- Decarboxylase - high content of the enzyme decarboxylase

Management:

- ✓ usually metastatic disease by time of diagnosis
- ✓ Patients with very early stage disease (**T1-2a, N0, M0**) are now considered for **surgery**. NICE support this approach in their 2011 guidelines
- ✓ however, most patients with limited disease receive a **combination** of chemotherapy and radiotherapy
- ✓ patients with more extensive disease are offered **palliative chemotherapy**

Paraneoplastic features of lung cancer

- squamous cell: PTHrp, TSH, clubbing, HPOA
- small cell: ADH, ACTH, Lambert-Eaton syndrome

Lung carcinoid (Bronchial adenomas) (1%)

- ✓ The vast **majority** of **bronchial adenomas** are carcinoid tumors, arising from the amine precursor uptake and decarboxylation (APUD) system, like small cell tumors.
- ✓ Lung carcinoid accounts **1%** of **lung tumors** and for **10% of carcinoid tumors**.
- ✓ The term bronchial adenoma is being phased out.

Features:

- ✓ typical age = **40-50 years**
- ✓ smoking **not risk factor**
- ✓ slow growing: e.g. long history of cough, recurrent hemoptysis
- ✓ often **centrally** located and not seen on CXR
- ✓ 'cherry red ball' often seen on bronchoscopy
- ✓ carcinoidsyndrome itself is **rare** (associated with liver metastases)

Management:

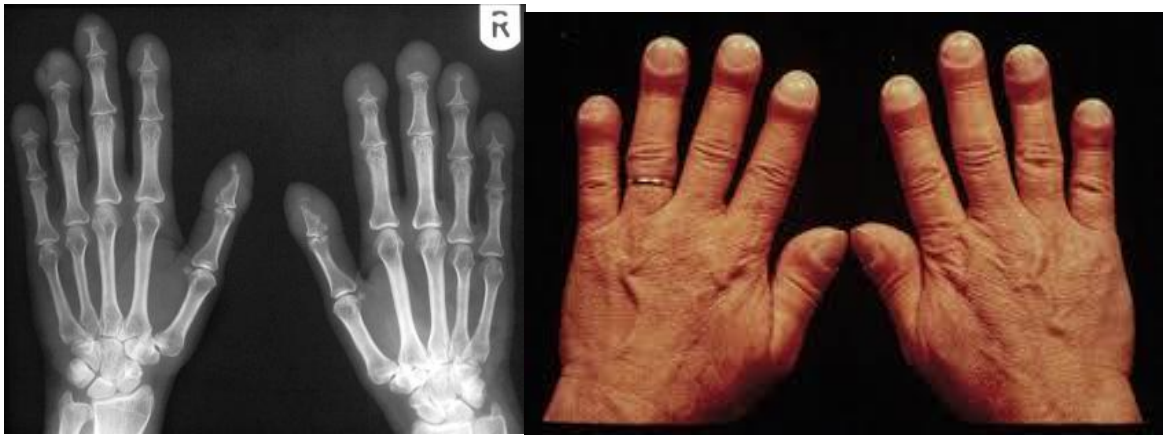
- ✓ surgical resection
- ✓ if no metastases then 90% survival at 5 years

Paraneoplastic features in Lung cancer

A) **Squamous cell:**
- parathyroid hormone-related protein (PTH-rp) secretion causing hypercalcaemia
- hyperthyroidism due to ectopic TSH
- hypertrophic pulmonary osteoarthropathy (HPOA)
- clubbing

B) **Adenocarcinoma:**
- Gynaecomastia

C) **Small cell:**
- ADH
- ACTH (Increased cortisol)- not typical, hypertension, hyperglycaemia, hypokalaemia, alkalosis and muscle weakness are more common than buffalo hump etc
- Lambert-Eaton syndrome



The TNM Classification of Malignant Tumors(TNM)

A cancer staging system that describes the extent of cancer in a patient's body
- T describes the size of the tumor and whether it has invaded nearby tissue,
- M describes distant metastasis (spread of cancer from one body part to another),
- N describes regional lymph nodes that are involved.

Primary Tumor (T)	
TX	• Primary tumor cannot be assessed, or • tumor proven by the presence of malignant cells in sputum or bronchial washings but not visualized by imaging or bronchoscopy
T0	No evidence of primary tumor
Tis	Carcinoma in situ
T1	• Tumor **3 cm** or less in greatest dimension, • surrounded by lung or visceral pleura, **without** bronchoscopic evidence of **invasion** more proximal than the lobar bronchus (for example, **not in the main bronchus**)
T1a	Tumor **2 cm** or less in greatest dimension
T1b	Tumor more than **2 cm but 3 cm** or less in greatest dimension

T2	Tumor more than **3 cm but 7** cm or less or tumor with any of the following features (T2 tumors with these features are classified T2a if 5 cm or less): • involves main bronchus, 2 cm or more distal to the carina; • invades visceral pleura (PL1 or PL2); • associated with atelectasis or obstructive pneumonitis that extends to the hilar region but does not involve the entire lung
T2a	Tumor more than **3 cm but 5 cm** or less in greatest dimension
T2b	Tumor more than **5 cm but 7 cm** or less in greatest dimension
T3	Tumor **more than 7 cm** or one that directly invades any of the following: • parietal pleural (PL3), • chest wall (including superior sulcus tumors), • diaphragm, phrenic nerve, • mediastinal pleura, parietal pericardium; or Tumor in the main bronchus less than 2 cm distal to the carina1 but without involvement of the carina; or associated atelectasis or obstructive pneumonitis of the entire lung or separate tumor nodule(s) in the same lobe
T4	Tumor of **any size** that **invades any of the following**: • mediastinum, heart, great vessels, • trachea, recurrent laryngeal nerve, esophagus, • vertebral body, carina, • separate tumor nodule(s) in a different ipsilateral lobe

Distant Metastasis (M)	
M0	No distant metastasis
M1	Distant metastasis
M1a	• Separate tumor nodule(s) in a contralateral lobe, • tumor with pleural nodules or malignant pleural (or pericardial) effusion
M1b	Distant metastasis (in extrathoracic organs)

Regional Lymph Nodes (N)	
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastases
N1	Metastasis in ipsilateral peribronchial and/or ipsilateral hilar lymph nodes and intrapulmonary nodes, including involvement by direct extension
N2	Metastasis in ipsilateral mediastinal and/or subcarinal lymph node(s)
N3	• Metastasis in contralateral mediastinal, contralateral hilar, • ipsilateral or contralateral scalene, or • supraclavicular lymph node(s)

Occupational lung disease

Asbestos and the lung

Asbestos can cause a variety of lung disease from benign pleural plaques to mesothelioma.

✓ **Pleural plaques**

- Pleural plaques are **benign** and **do not undergo malignant change**.
- They are the **most common** form of asbestos related lung disease
- Generally, occur after a latent period of **20-40 years**

✓ **Pleural thickening**

- Asbestos exposure may cause **diffuse pleural thickening (bilateral)** in a similar pattern to that followingTB or Bacterial empyema or hemothorax (causes of unilateral pleural thickening).
- The underlying pathophysiology is not fully understood.

✓ **Asbestosis**

- The severity of asbestosis is**related to the length of exposure**.
- The **latent period is typically 15-30 years**.
- Asbestosis typically causes **lower lobe fibrosis**.
- As with other forms of lung fibrosis the most common symptoms are shortness-of-breath and reduced exercise tolerance.

✓ **Mesothelioma**

- Mesothelioma is a malignant disease of the pleura.
- **Crocidolite (blue)** asbestos is the most dangerous form.
- Very limited exposure can cause disease.

Possible features:

- progressive shortness-of-breath
- chest pain
- pleural effusion

Management:

- Patients are usually offered **palliative chemotherapy**
- There is also a **limited role for surgery and radiotherapy**.
- Unfortunately, the **prognosis is very poor**
- Median survival from diagnosis of **8-14 months**

✓ **Lung cancer**

- Asbestos exposure increases risk of lung ca by a **factor of 5**
- Also has a synergistic effect with cigarette smoke.

Smoking and **asbestos** are **synergistic**, i.e. a smoker with asbestos exposure has a 10 * 5 = **50 times** increased risk

Silicosis

- Silicosis is a fibrotic lung disease associated with the inhalation of silicon dioxide (silica).
- It is usually found in quarry workers or minersand sandblasters, pottery workers and stonemasons (if the dust contains quartz).
- Diagnosis is made on industrial history and typical chest x ray changes.
- The pathognomonic radiological changes are hilar eggshell calcification.
- Silicosis is a risk factor for developing **TB** (silica is toxic to macrophages)

Features:

- **Fibrosing** lung disease (upper lung zone)
- **Egg-shell calcification** of the **hilar lymph nodes**

Disease	Agent	Effects
Aluminosis	Alum, and al. oxide	Fibrosis, bullae, pneumothorax
Asbestosis	Asbestos	Pleural plaques, lung cancer, mesothelioma
Byssinosis	Cotton, flax, hemp	Airway obstruction, loss of elasticity
Metal fume fever	Cadmium, cobalt, nickel, zinc and others	Chemical pneumonitis
Occupational asthma	Western Red Cedar and others	Reversible airway obstruction
Siderosis	Iron oxide	Dust deposits
Silicosis	Silica	Dust deposits and fibrosis
Talcosis	Talc, hydrated Mg. silicates	Perivascular fibrosis

Disorders of the chest wall and pleura

Mesothelioma

Basics:

- ✓ Malignancy of mesothelial cells of pleura
- ✓ Metastases to **contralateral lung** and **peritoneum**
- ✓ **Right lung** affected more often than left

Features:

- ✓ Dyspnea, weight loss, chest wall pain
- ✓ **Clubbing**
- ✓ **30%** present as painless pleural effusion
- ✓ Only **20%** have pre-existing **asbestosis**
- ✓ Very limited exposure can cause disease.
- ✓ History of asbestos exposure in **85-90%,** latent period of **30 years {Crocidolite (blue)}**

Diagnosis:

- ✓ suspicion is normally raised by a chest x-ray showing either a pleural effusion or pleural thickening
- ✓ the next step is normally a **pleural CT**

- ✓ if a pleural effusion is present fluid should be sent for MC&S, biochemistry and cytology (**but cytology is only helpful in 20-30% of cases**)
- ✓ **Local anesthetic thoracoscopybiopsy** (Video-assisted thoracoscopic surgery VATSbiopsy) is increasingly used to investigate **cytology negative exudative effusions** as it has a high **diagnostic** yield (around **95%**)
- ✓ if an area of **pleural nodularity** is seen on CT then an **image-guided pleural biopsy** may be used

Management:

- ✓ Symptomatic
- ✓ Industrial compensation
- ✓ Chemotherapy, Surgery if operable
- ✓ Prognosis poor, median survival 12 months

Pleural effusion

Exudate (> 30g/L protein)	**Transudate (< 30g/L protein)**
✓ **infection:** pneumonia, TB, subphrenic abscess ✓ **connective tissue disease:** RA, SLE ✓ **neoplasia:** lung cancer, mesothelioma, metastases ✓ pancreatitis ✓ pulmonary embolism ✓ Dressler's syndrome ✓ yellow nail syndrome	✓ heart failure ✓ hypoalbuminaemia (liver disease, nephrotic syndrome, malabsorption) ✓ hypothyroidism ✓ Meigs' syndrome

Pleural effusion investigation

The British Thoracic Society (BTS) produced guidelines in 2010 covering the investigation of patients with a pleural effusion:

- ✓ **Imaging:**
  1) Posterior anterior (PA) **chest x-rays** should be performed in all patients
  2) **Ultrasound** is recommended: it increases the likelihood of successful pleural aspiration and is sensitive for detecting pleural fluid septations
- ✓ **Pleural aspiration:**
  - as above, ultrasound is recommended to reduce the complication rate
  - a 21G needle and 50ml syringe should be used
  - fluid should be sent for pH, protein, lactate dehydrogenase (LDH), cytology and microbiology
    - **exudates** have a protein level **>35 g/L**,
    - **transudates** have a **protein level <35 g/L**

Light's criteria were developed in 1972 to help distinguish between a transudate and exudates. The BTS recommend using the criteria for borderline cases:

- If the protein level is between 25-35 g/L, Light's criteria should be applied.
- **An exudate is likely if at least one of the following criteria are met:**
  - pleural fluid protein divided by serum protein >0.5
  - pleural fluid LDH divided by serum LDH >0.6
  - pleural fluid LDH more than two-thirds the upper limits of normal serum LDH

Pleural infection:

- ✓ **all patients** with a pleural effusion in association with sepsis or a pneumonic illness require **diagnostic pleural fluid** sampling
- ✓ if the fluid is **purulent** or **turbid/cloudy** a **chest tube** should be placed to allow drainage
- ✓ if the fluid is clear but the **pH is less than 7.2** in patients with suspected pleural infection a **chest tube** should be placed

Other characteristic pleural fluid findings:

- ✓ **low glucose:** rheumatoid arthritis, tuberculosis
- ✓ **raised amylase:** pancreatitis, oesophageal perforation
- ✓ **heavy blood staining:** mesothelioma, pulmonary embolism, tuberculosis

Pneumothorax

- ✓ The British Thoracic Society (BTS) published updated guidelines for the management of spontaneous pneumothorax in 2010.
- ✓ A pneumothorax is termed primary if there is no underlying lung disease and secondary if there is.

Primary Pneumothorax

Recommendations include:

- ✓ If the rim of air is **< 2cm** and the patient is **not short of breath** then **discharge** should be considered & **CXR after 2 weeks**.
- ✓ **Otherwise aspiration** should be attempted
- ✓ **if this fails** (defined as > 2 cm or still short of breath) then a **chest drain** should be inserted
- ✓ patients should be advised to **avoid smoking** to reduce the risk of further episodes
- ✓ the lifetime risk of developing a pneumothorax
  - in **healthy smoking** men is around **10%**
  - **non-smoking men → 0.1%**

Secondary Pneumothorax

All patients should be admitted for at least 24 hours

- ✓ **if the pneumothorax is less the 1cm** then the BTS guidelines suggest:
  - **giving oxygen** (high flow O2) and
  - **admitting for 24 hours**
- ✓ If the patient is:
  - **50 years old and the rim of air is > 2cm** and/or
  - **The patient is short of breath** then
    - A **chest drain should be inserted**.

Otherwise **aspiration** should be attempted if the **rim of air is between 1-2cm**.
If aspiration fails (i.e. pneumothorax is still greater then 1cm) a **chest drain** should be inserted.

- For a **second unilateral pneumothorax** in a fit individual is referral for **bullectomy and pleurectomy**. This is conducted under video assisted guidance, video assisted thoracoscopic surgery (**VATS**), until then, **diving and flying** are **contraindicated.**

- Regarding scuba diving, the BTS guidelines state: **Diving** should be permanently avoided unless the patient has undergone **bilateral surgical pleurectomy** and has **normal lung function** and **chest CT** scan **postoperatively**.
- **Pleurodesis** could be considered in elderly or frail individuals.

Iatrogenic Pneumothorax

Recommendations include:

- ✓ less likelihood of recurrence than spontaneous pneumothorax
- ✓ majority will resolve with **observation**, if treatment is required then **aspiration** should be used
- ✓ **ventilated** patients need **chest drains**, as may some patients with **COPD**

Questions sometimes discuss the size of the pneumothorax in percentage terms rather than giving the interpleural distance. A variety of formulas have been proposed to convert between the two.
As a very general rule of thumb:

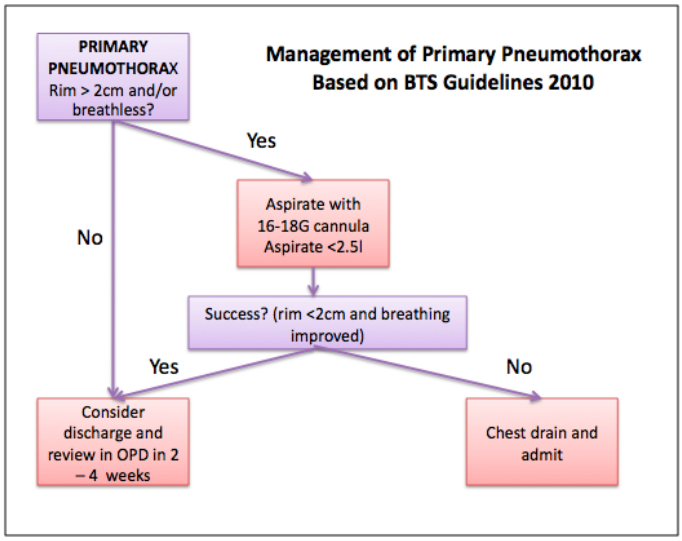
Average interpleural distance	Approximate size of pneumothorax
0.5 cm	10%
1 cm	15 %
2 cm	30%
3 cm	45%
4 cm	60%

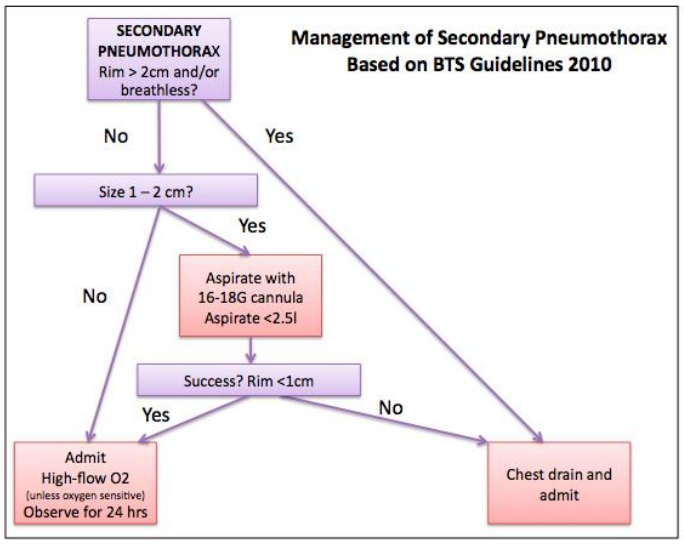
A pneumothorax of 20% if therefore within the 2-cm limit suggested by the British Thoracic Society for observation, if the patient is not short of breath

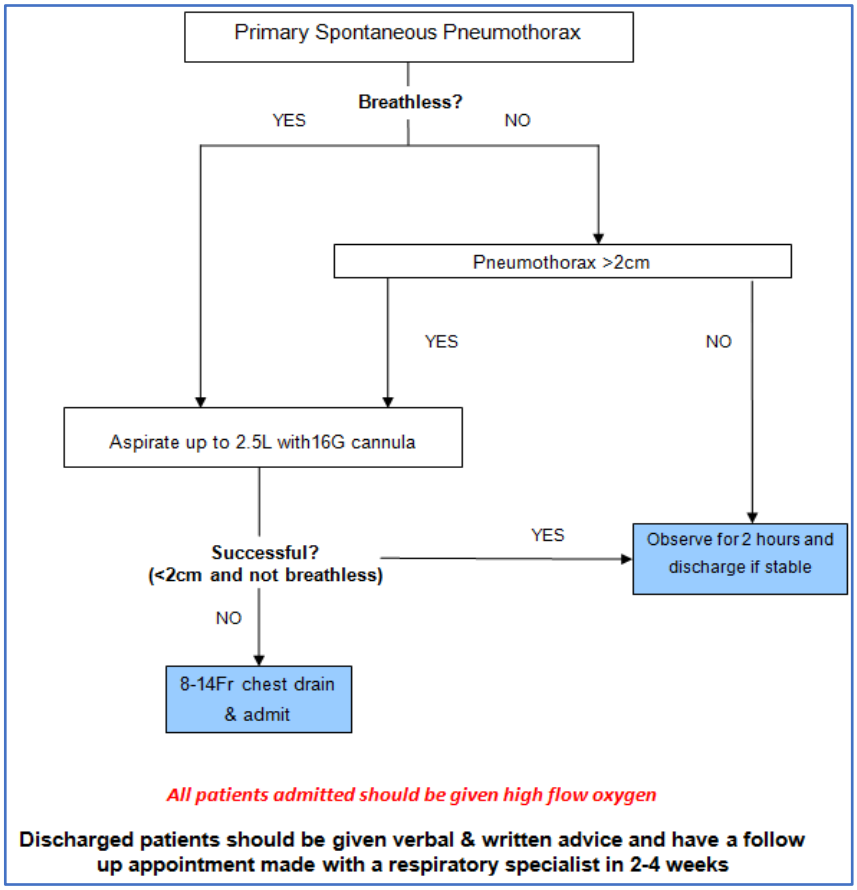
Small bore drains are just as effective as large bore drains and are less painful when in situ. The most appropriate point for chest drain insertion is in the 'safe triangle' in the **mid-axillary** line. This reduces injury to the internal mammary artery, muscle, liver and spleen.

Scarring from insertion is less obvious than in the second intercostal space and mid-clavicular line, particularly in women.

Loculated apical pneumothoraces (as demonstrated by a CT scan) may be drained using a **posteriorly sited** (**suprascapular)** apical tube.







Fitness to fly

The Civil Aviation Authority (CAA) has issued guidelines on air travel for people with medical conditions; please see the link provided.

Cardiovascular disease

- ✓ unstable angina, uncontrolled hypertension, uncontrolled cardiac arrhythmia, decompensated heart failure, severe symptomatic valvular disease: **should not fly**
- ✓ **uncomplicated MI**: may fly after 7-10 days
- ✓ **complicated MI**: after 4-6 weeks
- ✓ **CABG**: after 10-14 days
- ✓ **PCI**: after 5 days

Respiratory disease

- ✓ pneumonia: should be 'clinically improved with no residual infection'
- ✓ **Pneumothorax: absolute contraindication**, the CAA suggests patients may travel 2 weeks after successful drainage if there is no residual air.
- ✓ The British Thoracic Society used to recommend not travelling by air for a period of 1 week post check x-ray

Pregnancy

- ✓ most airlines do not allow travel **after 36 weeks** for a single pregnancy and after **32 weeks** for a multiple pregnancy
- ✓ most airlines require a **certificate after 28 weeks** confirming that the pregnancy is progressing normally

Surgery

- ✓ travel should be avoided for **10 days** following **abdominal surgery**
- ✓ **laparoscopic surgery**: after **24 hours**
- ✓ **colonoscopy**: after **24 hours**
- ✓ following the application of a **plaster cast**, the majority of airlines restrict flying for **24** hours on flights**< 2 hours** or **48 hours** for longer flights

Hematological disorders

- ✓ patients with a hemoglobin**> 8 g/dl may** travel without problems (assuming there is no coexisting condition such as cardiovascular or respiratory disease)

Pulmonary embolism

Investigation

- ✓ We know from experience that few patients (around **10%**) present with the medical student textbook triad of **pleuritic chest pain, dyspnea and haemoptysis.**
- ✓ Pulmonary embolism can be difficult to diagnose as it can present with virtually any cardiorespiratory symptom/sign depending on its location and size.

So, which features make pulmonary embolism more likely?

- ✓ The PIOPED study in 2007 looked at the frequency of different symptoms and signs in patients who were diagnosed with pulmonary embolism.
- ✓ The relative frequency of common clinical signs is shown below:
  - **Tachypnea** (RR>16/min) - **96%**
  - **Crackles - 58%**
  - **Tachycardia** (HR>100/min) - **44%**
  - **Fever** (>37.8C) - **43%**
- ✓ It is interesting to note that the Well's criteria for diagnosing a PE use **tachycardia rather than tachypnea**.

2012 NICE guidelines

- ✓ All patients with symptoms or signs suggestive of a PE should have a history taken, examination performed and a **chest x-ray** to exclude other pathology.
- ✓ If a PE is still suspected a two-level PE **Wells score** should be performed.

Clinical feature	Points
Clinical **S & S** of **DVT** (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE (PE is #1 diagnosis)	3
Heart rate > 100 beats per minute	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Hemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Traditional interpretation:
- High score: >6.0
- Moderate score: 2.0 to 6.0
- Low score: <2.0

Alternate interpretation score:
- >4 - PE likely. Consider diagnostic imaging
- ≤4 - PE unlikely. Consider D-dimer to rule out PE.

If a PE is 'likely' (> 4 points)
- Arrange an **immediate computed tomography pulmonary angiogram** (CTPA).
- If there is a delay in getting the CTPA then give **LMWH** until the scan is performed

If a PE is 'unlikely' (4 points or less)
- Arrange a **D-dimer** test.
- If this is positive arrange an **immediate** (**CTPA**).
- If there is a delay in getting the CTPA then give LMWH until the scan is performed.
- If the patient has an **allergy** to contrast media or **renal impairment** a **V/Q scan** should be used instead of a CTPA.

CTPA or V/Q scan?
The consensus view from the British Thoracic Society and NICE guidelines is as follows:
Computed tomographic pulmonary angiography (CTPA)
- ✓ It is now the recommended **initial lung-imaging modality** for non-massive PE.
- ✓ Advantages compared to V/Q scans include:
  - speed, easier to perform out-of-hours,
  - a reduced need for further imaging and the possibility of providing an alternative diagnosis if PE is excluded
  - if the CTPA is negative then patients do not need further investigations or treatment for PE

Ventilation-perfusion scanning may be used initially if appropriate facilities exist, the chest x-ray is normal, and there is no significant symptomatic concurrent cardiopulmonary disease

Some other points
D-dimers:

- **sensitivity** = **95-98%,** but **poor specificity (good -ve)**

ECG:

- ✓ **S1Q3T3:** the classic ECG changes seen in PE are a large S wave in lead I, a large Q wave in lead III and an inverted T wave in lead III - '**S1Q3T3**'. However this change is seen in no more than **20%** of patients
- ✓ **RBBB** and **RAD** are also associated with PE
- ✓ **sinus tachycardia** may also be seen

V/Q scan:

- ✓ **sensitivity = 98%;specificity = 40%** - high negative predictive value, i.e. if normal virtually excludes PE
- ✓ other causes of **mismatch in V/Q** include:
  - old pulmonary embolisms,
  - AV malformations,
  - vasculitis,
  - previous radiotherapy
  - **COPD** gives **matched defects**

CTPA:

- ✓ peripheral emboli affecting **subsegmental arteries** may be **missed**

Pulmonary angiography:

- ✓ the **gold standard**
- ✓ **significant complication** rate compared to other investigations

Pulmonary embolism management

- ✓ The NICE guidelines of 2012 provided some clarity on how long patients should be anticoagulated for after a pulmonary embolism (PE). Selected points are listed below.
- ✓ **LMWH** or **fondaparinux** should be given initially after a PE is diagnosed.
  An exception to this is for:
  - Patients with a massive PE where **thrombolysis** is being considered.
  - In such a situation **unfractionated heparin** should be used.

- ✓ A vitamin K antagonist (i.e. **warfarin**) should be given within 24 hours of the diagnosis
- ✓ the LMWH or fondaparinux should be continued for **at least 5 days** or **until INR is 2.0** or above for **at least 24 hours**, **whichever is longer**, i.e. LMWH or fondaparinux is given at the same time as warfarin until the INR is in the therapeutic range
- ✓ Warfarin should be continued for **at least 3 months**. At 3 months, NICE advice that clinicians should 'assess the risks and benefits of extending treatment'
- ✓ NICE advice extending warfarin beyond 3 months for patients with **unprovoked PE.** This essentially means that if there was no obvious cause or provoking factor (surgery, trauma, significant immobility) it may imply the patient has a tendency to thrombosis and should be given treatment longer than the norm of 3 months
- ✓ For patients with **active cancer** NICE recommend using **LMWH** for **6 months (lifelong)** or **till cure of cancer**.

Thrombolysis

- ✓ Thrombolysis is now recommended as the **first-line treatment** for **massive PE** where there is circulatory failure (e.g. hypotension, acidosis).
- ✓ Other invasive approaches should be considered where appropriate facilities exist

Pregnancy: DVT/PE investigation

Guidelines were updated in 2010 by the Royal College of Obstetricians. Key points include:

- ✓ **chest x-ray** should be performed in all patients
- ✓ compression **duplex Doppler:**
  - should be performed if the chest x-ray is normal
  - this may provide indirect evidence of a pulmonary embolism and negate the need for further radiation exposure
- ✓ the decision to perform a **V/Q or CTPA** should be taken at a local level after discussion with the patient and radiologist

Comparing CTPA to V/Q scanning in pregnancy

CTPA	V/Q scanning
• CTPA slightly increases the lifetime risk of **maternal breast cancer** (increased by up to 13.6%, background risk of 1/200 for study population). • Pregnancy makes breast tissue particularly sensitive to the effects of radiation	• V/Q scanning carries a slightly increased risk of **childhood cancer** compared with CTPA (1/280,000 versus less than 1/1,000,000)

D-dimer is of limited use in the investigation of thromboembolism as it **often rose in pregnancy.**

Rheumatoid arthritis: respiratory manifestations

- ✓ pulmonary fibrosis
- ✓ pulmonary nodules
- ✓ Caplan's syndrome - massive fibrotic nodules with occupational coal dust exposure
- ✓ bronchiolitis obliterans
- ✓ pleurisy
- ✓ pleural effusion (the commonest)
- ✓ bronchiectasis (especially non-smokers)
- ✓ complications of drug therapy e.g. methotrexate pneumonitis, sulfasalzine, gold
- ✓ infection (possibly atypical) secondary to immunosuppression

Respiratory acidosis

Respiratory acidosis may be caused by a number of conditions

- ✓ COPD
- ✓ decompensation in other respiratory conditions e.g. life-threatening asthma / pulmonary oedema
- ✓ sedative drugs: benzodiazepines, opiate overdose

Respiratory alkalosis

Common causes

- ✓ anxiety leading to hyperventilation
- ✓ pulmonary embolism
- ✓ salicylate poisoning*
- ✓ CNS disorders: stroke, subarachnoid haemorrhage, encephalitis
- ✓ altitude
- ✓ pregnancy

***Salicylate overdose** leads to a mixed respiratory alkalosis and metabolic acidosis. Early stimulation of the respiratory centre leads to a respiratory alkalosis whilst later the direct acid effects of salicylates (combined with acute renal failure) may lead to an acidosis

Oxygen therapy

- ✓ The British Thoracic Society published guidelines on emergency oxygen therapy in 2008.
- ✓ The following selected points are taken from the guidelines.
  - **In patients who are critically ill (anaphylaxis, shock etc) oxygen should initially be given via a reservoir mask at 15 l/min. Hypoxia kills.**
  - The BTS guidelines specifically exclude certain conditions where the patient is acutely unwell (e.g. myocardial infarction) but stable.

Oxygen saturation targets:

- ✓ acutely ill patients: 94-98%
- ✓ patients at risk of hypercapnia (e.g. COPD patients): 88-92% (see below)
- ✓ oxygen should be reduced in stable patients with satisfactory oxygen saturation

Management of COPD patients:

- ✓ prior to availability of blood gases, use a **28% Venturi mask at 4 l/min** and aim for an oxygen saturation of **88-92%** for patients with risk factors for hypercapnia but no prior history of respiratory acidosis
- ✓ adjust target range to **94-98%** if the **pCO_2 is normal**

Situations where oxygen therapy should not be used routinely if there is no evidence of hypoxia:

- ✓ myocardial infarction and acute coronary syndromes
- ✓ stroke
- ✓ obstetric emergencies
- ✓ anxiety-related hyperventilation

Non-invasive ventilation

- ✓ The British Thoracic Society (BTS) published guidelines in 2002 on the use of non-invasive ventilation in acute respiratory failure.
- ✓ Following these the Royal College of Physicians published guidelines in 2008.

Key indications

1) **COPD** with respiratory acidosis **pH 7.25-7.35**
2) **type II respiratory** failure secondary to chest wall deformity, neuromuscular disease or obstructive sleep apnoea
3) **cardiogenic pulmonary oedema** unresponsive to CPAP
4) **weaning** from tracheal intubation

Recommended initial settings for bi-level pressure support in COPD

- ✓ Expiratory Positive Airway Pressure (**EPAP**): **4-5 cm H2O**
- ✓ Inspiratory Positive Airway Pressure (**IPAP**): RCP advocate **10 cm H20** whilst BTS suggest **12-15 cm** H2O
- ✓ Back up rate: **15 breaths/min**
- ✓ Back up inspiration: expiration ratio:**1:3**

Acute exacerbation of COPD (AECOPD) & type II respiratory failure

- ✓ The choices are either non-invasive ventilation (**NIV**) through a full face or nasal mask, or endotracheal intubation (**ETI**) with ventilation on an ICU.
- ✓ In selected patients with type 2 respiratory failures due to AECOPD **NIV** has been shown to **decrease mortality** and **length of hospital stay** over ETI.

However, there are **contraindications to NIV:**

- ✓ Hemodynamically unstable
- ✓ Confusion / impaired consciousness
- ✓ Vomiting
- ✓ Inability to protect airway
- ✓ Fixed obstruction of the upper airway

- ✓ Facial burns/traums, and
- ✓ Undrained pneumothorax.

It should be noted however NIV may be used despite many of these contraindications if NIV is to be the ceiling of treatment.

The criteria for LTOT are PaO_2 less than 7.3 kPa (55 mmHg) with or without hypercapnia or PaO_2 less than 8.0 kPa (60 mmHg) if there is evidence of pulmonary hypertension/cor pulmonale/polycythaemia. See COPD for more details
LTOT and smoking cessation are currently the only interventions in COPD that have been shown to prolong life.

Acute epiglottitis:

- ✓ Sudden airway obstruction may occur and it is vital to obtain the assistance of an anesthetist urgently.
- ✓ **No attempt** should be made to visualize the epiglottis until an anesthetist is present as there is a high risk of causing acute airway obstruction by touching the inflamed tissue.
- ✓ The diagnosis may be confirmed on direct visualisation of a cherry-red epiglottis.
- ✓ **Early intubation is essential**, especially in cases where there is respiratory distress.
- ✓ Adult epiglottitis is much less common but has a higher mortality.
- ✓ The usual causative organism is ***Haemophilus influenzae* type b**.

Management:

A significant number of strains are resistant to ampicillin and a **third-generation cephalosporin** is **the treatment of choice.**

Assessing a patient's performance status is important when evaluating the most appropriate treatment options. It is commonly used by cancer MDTs, but has a role in assessing patients with chronic illnesses including COPD.

WHO Scale	Description
0	Asymptomatic
1	Symptomatic but ambulatory (can carry out light work)
2	In bed <50% of the day. Unable to work but can live at home with some assistance
3	In bed >50% of the day but unable to care for self
4	Bedridden

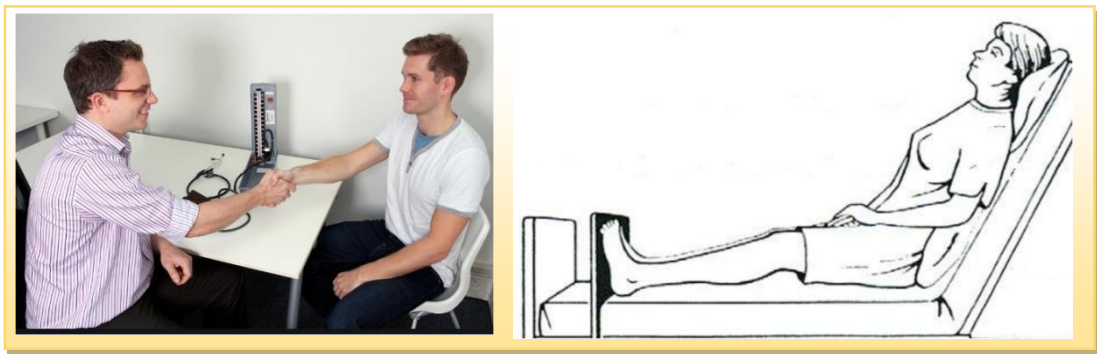
Clinical Approach

Chest

- Inspection:
  - shape: hyperinflated in severe asthma
  - movement of chest/silent chest (life-threatening)
  - chest deformity:
  - recession:
- Palpation:
  - chest expension may be reduce (hyperinflated)/ normal
  - apex beat: may be displaced
  - -vocal fremitus: decrease
- Percussion:
  - may be hyperresonance / normal
- Auscultation:
  - breath sound: vesicular
  - ronchi in expiratory phase, may be both in severe asthma
  - prolonged expiratory phase
  - -vocal resonance decrease / normal

General Physical Examination

⇒ Introduce yourself, and ensure that the patient is sitting comfortably at 45° and then stand back.



⇒ Placing your hands behind your back is a good way to show you remember the importance of inspection.
⇒ Look around the bedside for metered dose inhalers, nebulizers or sputum pots.
⇒ Note any cachexia.
⇒ Count the respiratory rate; (tachypnoea, defined as a respiratory rate > 20 breaths/minute (normal is 12–20 breaths/minute, albeit arbitrary), is often the first sign of respiratory or hemodynamic compromise) and note if the patient is breathless at rest.

Listen

⇒ Listen to the breathing with unaided ears, noting any of the sounds:
- Expiration longer than inspiration (obstructive airways disease).
- Expiratory wheeze (obstructive airways disease).
- Inspiratory strider (obstruction of upper airways, e.g. mediastinal mass, bronchial carcinoma)
- Clicks (bronchiectasis)
- Gurgling (airway secretions)

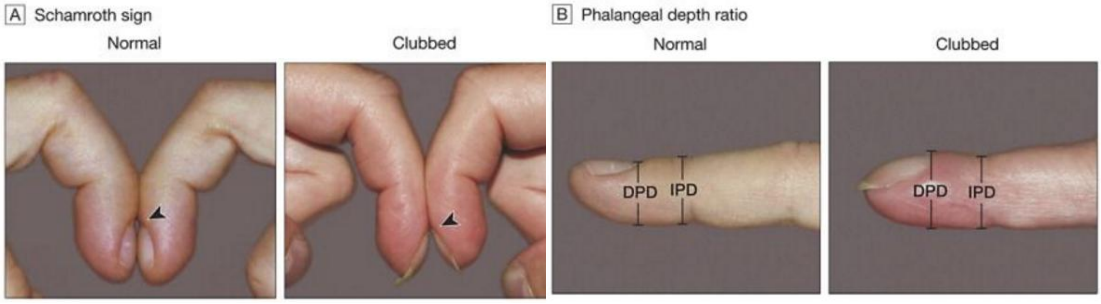
Hands

Look for

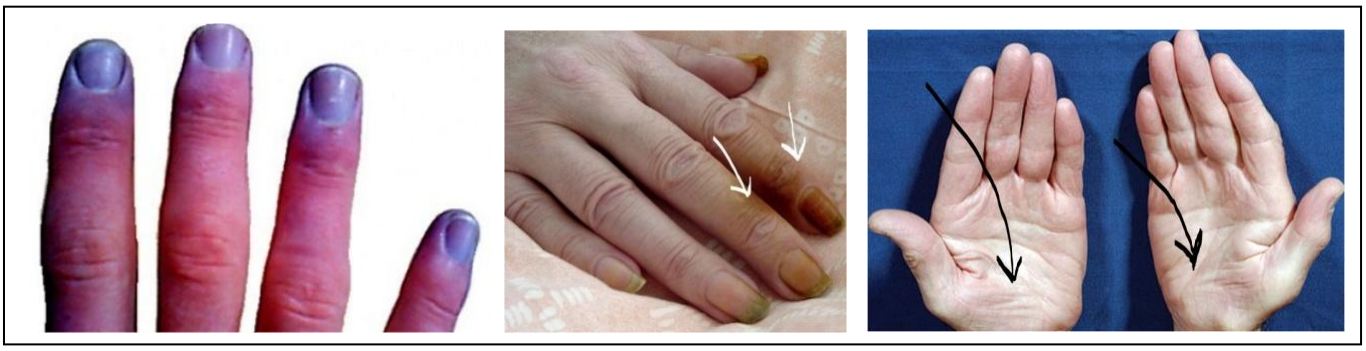
⇒ palmar erythema (CO_2 retention).
⇒ wasting of the small muscles of the hand (Pancoast tumour).
⇒ changes of rheumatological conditions associated with respiratory
⇒ disease e.g. rheumatoid arthritis, scleroderma.

Inspect the nails for:

⇒ clubbing with the patient's fingers directly in line with your vision so that the nail is observed at 90°.
Schamroth's sign (directly opposing distal phalanges of corresponding fingers and looking for the obliteration of the usual diamond-shaped window between the nailbeds) can be performed to assess clubbing.



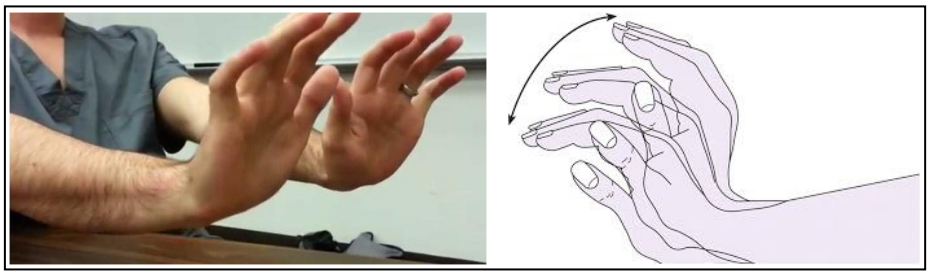
⇒ Peripheral cyanosis, tar staining, yellow nails (yellow nail syndrome) and muscle wasting.



Ask the patient to straighten out their arms and hold out their hands looking for:

⇒ A fine tremor associated with beta 2 agonist use.

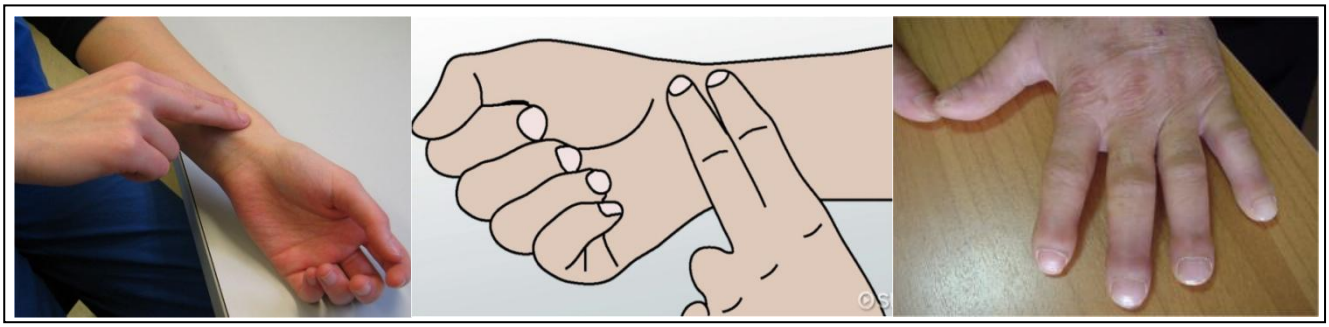
⇒ A course flapping tremor of CO_2 retention by asking the patient to cock their wrists back as if they were about to stop traffic. Asterixis is more likely to be present in an acutely unwell patient rather than a patient selected for the PACES exam.



⇒ Feel the patient's radial pulse. A bounding pulse is a characteristic sign of CO 2 retention.

⇒ Calculate their respiratory rate by counting the number of respirations over 15 seconds whilst feeling the pulse.

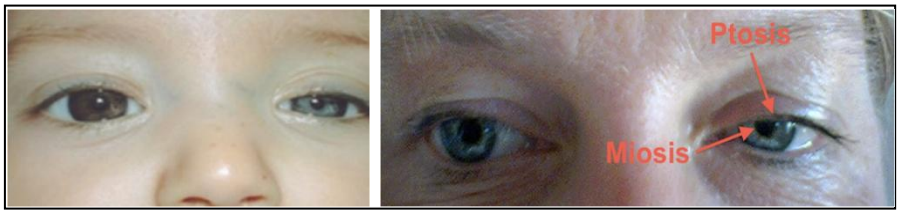
⇒ Pain and/or swelling of hands/wrists suggesting possible hypertrophic pulmonary osteoarthropathy.



Eyes

⇒ Suffused conjunctivae present in secondary polycythaemia.

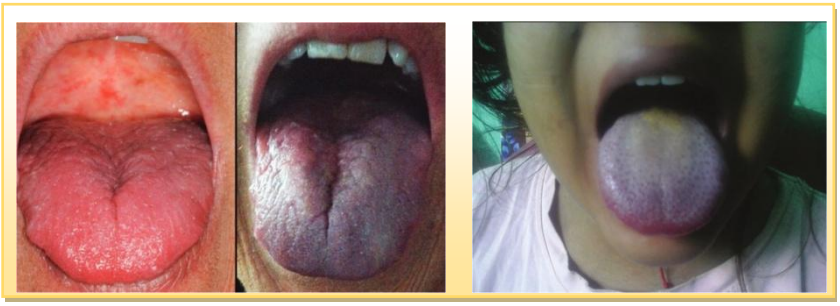
⇒ Horner's syndrome (meiosis, partial ptosis, enophthalmous, anhydrosis).



⇒ Ask the patient to look up and warn them that you are going to pull down gently on their lower eyelid.
Look for:
anaemia present in chronic disease e.g. cystic fi brosis.
jaundice which may indicate malignancy.

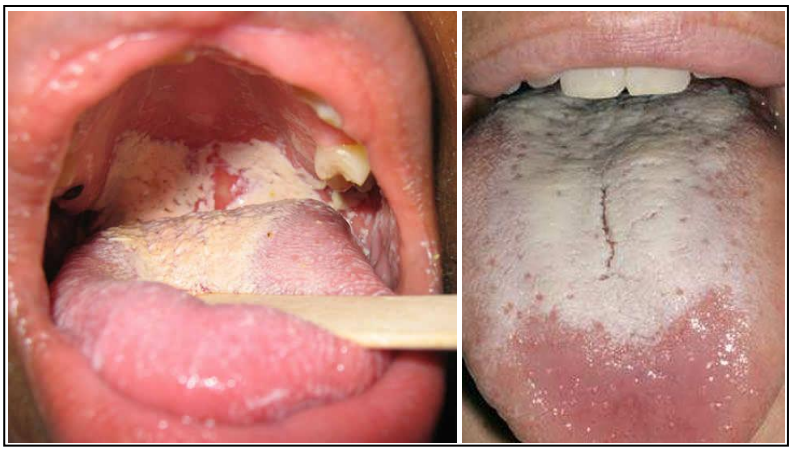
Mouth

⇒ Look for central cyanosis (best seen at the tongue).



⇒ Look for pursed lip breathing (PLB); that is a breathing technique that consists of exhaling through tightly pressed (pursed lips) and inhaling through the nose with the mouth closed.

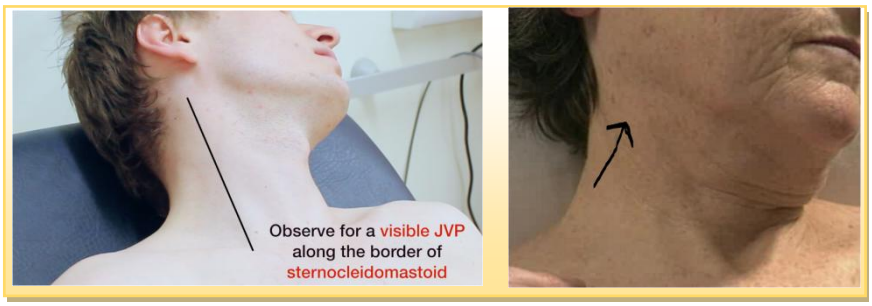
⇒ Evidence of oral candidiasis consistent with steroid use.



Neck

⇒ Look at the patient's neck size (larger collar size increases risk of obstructive sleep apnoea

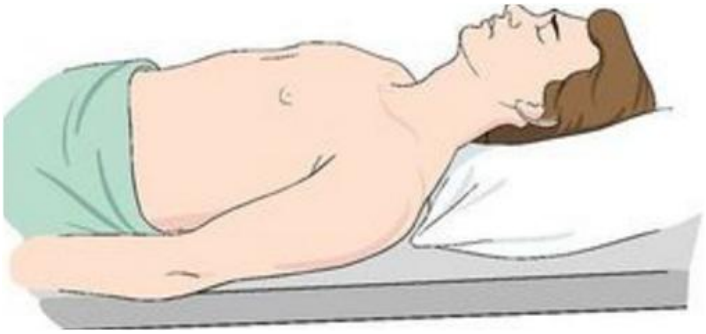
⇒ Assess the patient's jugular venous pressure (JVP) at 45 °.
The JVP is raised and pulsatile in cor pulmonale but fixed in superior vena cava obstruction, the latter characteristically causing marked venous distension in the neck and sometimes distension of veins in the hands, the underside of the tongue and upper chest wall (more detail in the history and physical examination section).



⇒ Observe any scars in the supraclavicular fossae.

Inspection

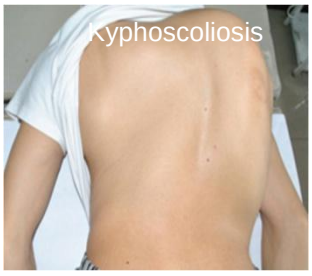
⇒ Exposed from the waist up.

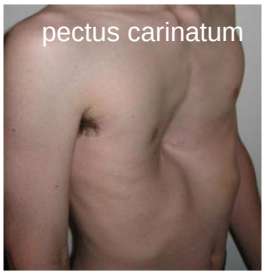


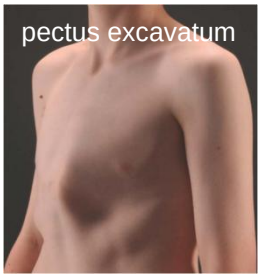
⇒ Take time to inspect the patient for important clues from the end of the bed. Stand directly opposite the patient in order to correctly assess any differences in each hemithorax.

⇒ Remember to cover the patient up after completing each element of the examination in order to maintain dignity.

⇒ Look at the size and shape of the chest, and note any deformities (e.g. kyphoscoliosis, pectus carinatum, pectus excavatum), thoracotomy scars, radiotherapy field markings, telangiectasia or muscle wasting.







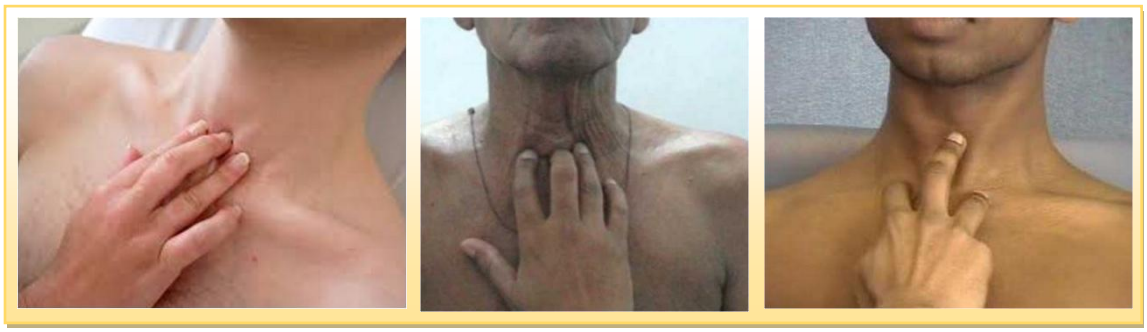
As a simple rule, a 'big chest' (with a large anteroposterior diameter, little lateral expansion, and lifting of the rib-cage on inspiration) alerts you to chronic obstructive pulmonary disease, whilst a 'small chest' alerts you to possible fibrotic lung disease.

⇒ Look at chest wall movement (upwards in emphysema; asymmetrical in fibrosis, collapse, pleural effusion or pneumothorax).
⇒ Note any use of accessory muscles, including abdominal or scalene muscles, or intercostal indrawing.

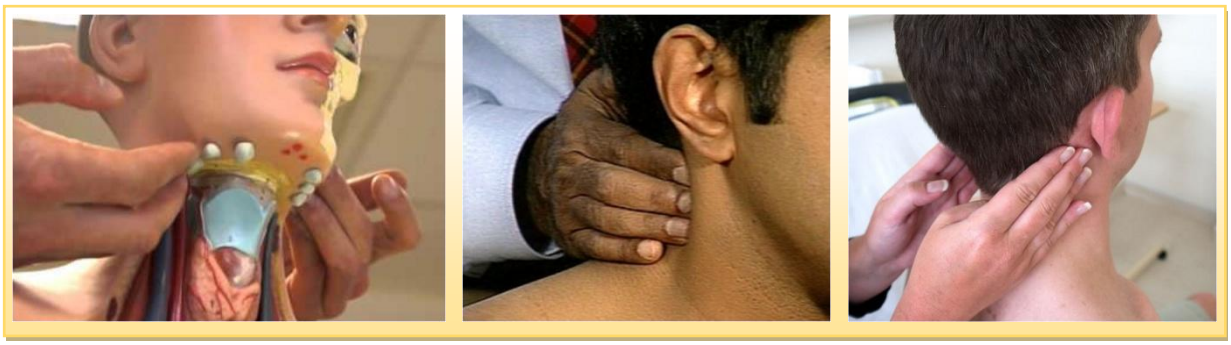
Palpation

Neck

⇒ Tracheal position:
- ✓ Ensure patient's neck musculature is relaxed – chin slightly downwards
- ✓ Dip index finger into the thorax beside the trachea
- ✓ Then gently apply side pressure to locate the trachea
- ✓ Compare this space to the other side of trachea using the same process
- ✓ A difference in the amount of space between the sides suggests deviation
- ✓ The trachea **deviates away** from pneumothorax and large pleural effusions
- ✓ The trachea **deviates towards** lobar collapse and pneumonectomy
- ✓ Palpation of the trachea can be uncomfortable, so warn the patient and apply a gentle technique
- ✓ Feel the trachea to determine any mediastinal shift (using the middle finger as the exploring finger and the index and ring fingers resting on the manubriosternum either side; and note the approximate cricoid–suprasternal notch distance, decreased in hyperinflation from the normal three finger-breadths.



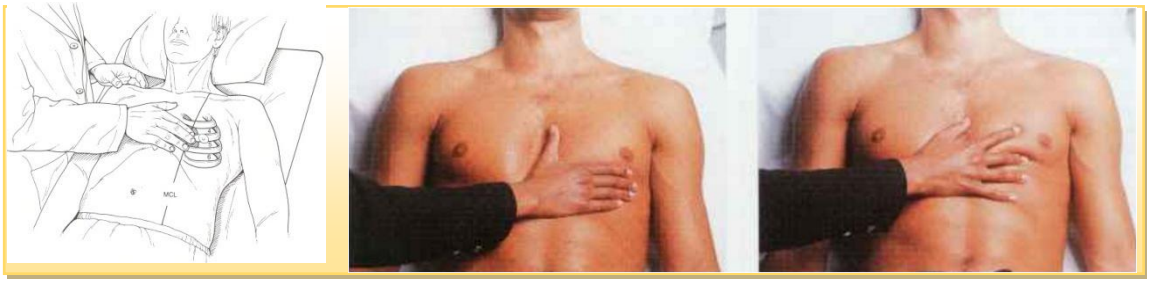
⇒ Feel for cervical, supraclavicular and axillary lymph nodes, always from behind using flat fingers and not poking with fingertips, some authors examine LN from front (more detail in the history and physical examination section).



Anterior chest

⇒ Apex beat:

- ✓ Normal position is 5th intercostal space – mid-clavicular line
- ✓ Right ventricular heave is noted in cor-pulmonale (right heart failure secondary to chronic hypoxic lung diseases such as COPD or ILD)



⇒ Chest expansion

Method #1

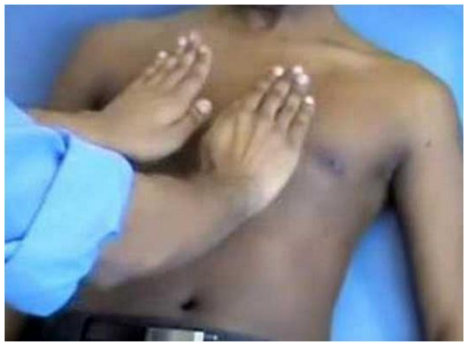
Either:

- ✓ **Inframammary** area use a 'bucket handle' approach with your fingers in the intercostal spaces either side of the chest and your thumbs floating in the midline. This allows the ribs to move outwards.
- ✓ **For the supramammary area**, where the ribs move predominantly upwards, place your hands on the chest wall with thumbs meeting.

Method #2

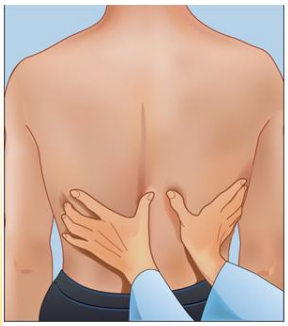
The physician places her/his left and right hand on the patient's left and right lateral chest walls.

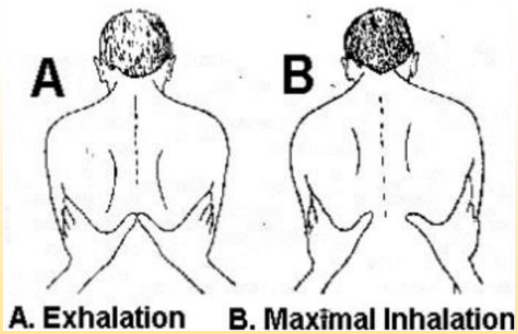
- ✓ The patient is instructed to take several deep breaths, while the physician observes both the motion of her hands and the force exerted on them by the patient's chest wall.
- ✓ Asymmetry in either the extent of hand movement, or the force exerted upon them, is an abnormal finding.

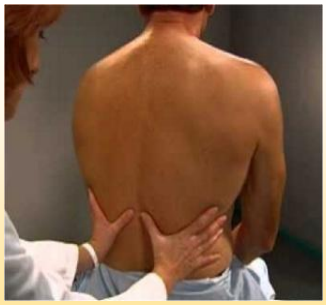


Posterior chest

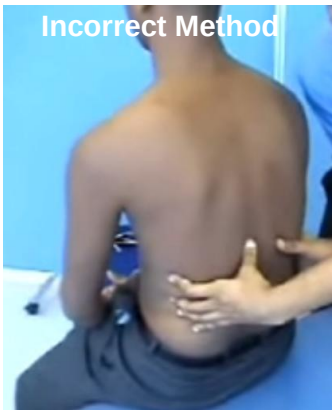
While standing behind the patient, the physician's hands are placed at the level of the patient's 10th ribs, with fingers pointed laterally and parallel to the ribs, and thumbs placed medially Then the physician's hands are slid medially to raise a small amount of loose skin between them, while keeping the thumbs 2-4 cm apart on the respective hemithoraces. The patient is instructed to take several deep breaths as the physician's thumbs are noted for symmetric or asymmetric movement.







Other method for back rarely used:



Interpretation

Asymmetric movement indicates a unilateral pathology on the side with less movement. Symmetric expansion has no diagnostic value, since it occurs in normal persons, patients with diffuse lung diseases, and many with unilateral pathologies. Thus this test is specific for unilateral pulmonary pathology, but it lacks sensitivity.

Percussion

Purpose

To determine if the area under the percussed finger is air filled **(sounding resonant like a drum)**, fluid filled (**a dull sound)** or solid **(a flat sound)**. To make this interpretation it is important not only to listen for the sound produced but also to feel the intensity and frequency of vibrations produced by this maneuver.

Percussion sites

- ✓ Supraclavicular areas
- ✓ Clavicles
- ✓ Chest on both sides

More than four or five levels of percussion are time consuming as a screen but percuss further to delineate any abnormality you find.

Percussion technique

Press the **distal phalanx** of the **middle finger** firmly on the area to be percussed and raise the second and fourth fingers off the chest surface; otherwise, both sound and tactile vibrations will be blunted.

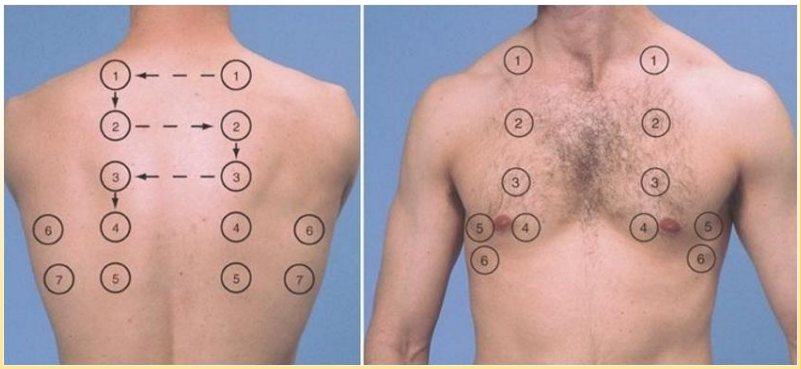
Use a quick, sharp wrist motion (like a catcher throwing a baseball to second base) to strike the finger in contact with the chest wall with **the tip of the third finger of the other hand**. The best percussion site is **between the proximal and distal interphalangeal joints**. The novice quickly learns to trim the fingernail to prevent personal discomfort of minor abrasions and lacerations.

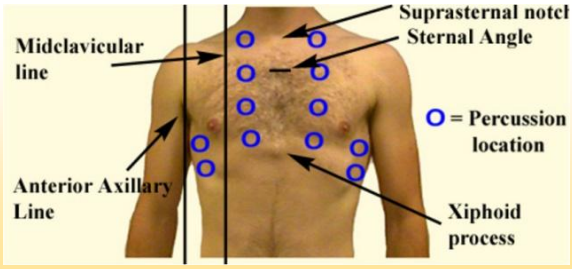
If the sound and the vibrations produced seem suboptimal, make sure that the finger placed directly on the thorax is making very firm direct contact with the chest wall. If not, few vibrations and little sound will be produced.

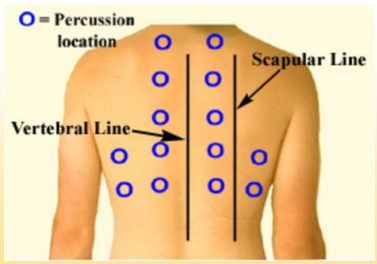
Percuss the posterior, lateral, and anterior chest wall in such a manner that the long axis of the percussed finger is roughly parallel to the ribs. **Compare one side to the other**.

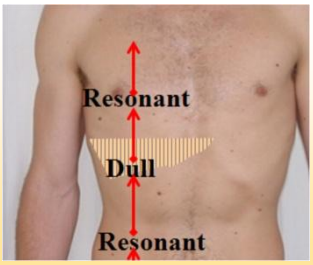
- ✓ Note the position of the diaphragm.
- ✓ Then ask the patient to inhale fully and "hold it";
- ✓ Continue to percuss inferiorly to determine the new level of the diaphragm, now during forced maximal inspiration.
- ✓ Then, don't forget to tell the patient to "breathe normally." The difference between the two levels is known as diaphragmatic excursion and should equal 2 to 3 cm.

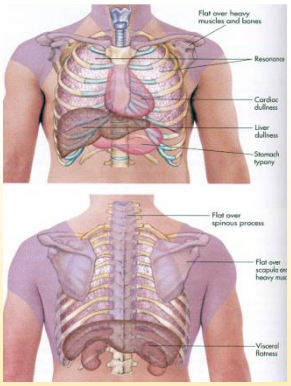
Remember that the upper level of liver dullness is around the sixth rib in the right mid-clavicular line. Resonance below this level is a sign of hyperinflation. Cardiac dullness may also be elicited.











Percussion

Hyperresonance

Resonance

Dullness

Flatness

Percussion sounds

Sound	Intensity	Pitch	Length	Quality	Example of origin
Resonance (heard over part air and part solid	Loud	Low	Long	Hollow	Normal lung
Hyper-resonance (heard over mostly air	Very loud	Low	Long	Booming	Lung with emphysema
Tympany (heard over air)	Loud	High	Moderate	Drum like	Puffed-out cheek, gastric bubble
Dullness (heard over more solid tissue	Medium	Medium	Moderate	Thud like	Diaphragm, pleural effusion
Flatness (heard over very dense tissue	Soft	High	short	Flat	Muscle, Bone, Thigh

Vocal resonance

⇒ Vocal resonance represents transmission of sound from the central airways to the chest wall. Sound transmission is enhanced through **solid tissue (consolidation)** provided the airways are patent and attenuated through **fluid (pleural effusion)** compared with transmission through **air (normal)**.

The principles are as for bronchial breathing in consolidation and reduced breath sounds in a pleural effusion. Enhanced sound transmission in consolidation is analogous to the vibration of an earthquake that can be felt through the ground before it can be heard through the air. Attenuated sound in a pleural effusion is analogous to diving underwater, the sound of people on land suddenly muffled.

⇒ '**Ninety-nine'** is the conventional sound used to assess vocal resonance, but the Intended nasal '**oi**' is better demonstrated by '**neun-und-neuzig**'.

⇒ **Whispering pectoriloquy**, when whispered sounds are heard clearly, confirms consolidation because a whispered voice is clearly audible through solid lung.

⇒ **Aegophony** is an unusual sign in which compressed lung above a pleural effusion creates a high-pitched bleat from conducted voice.

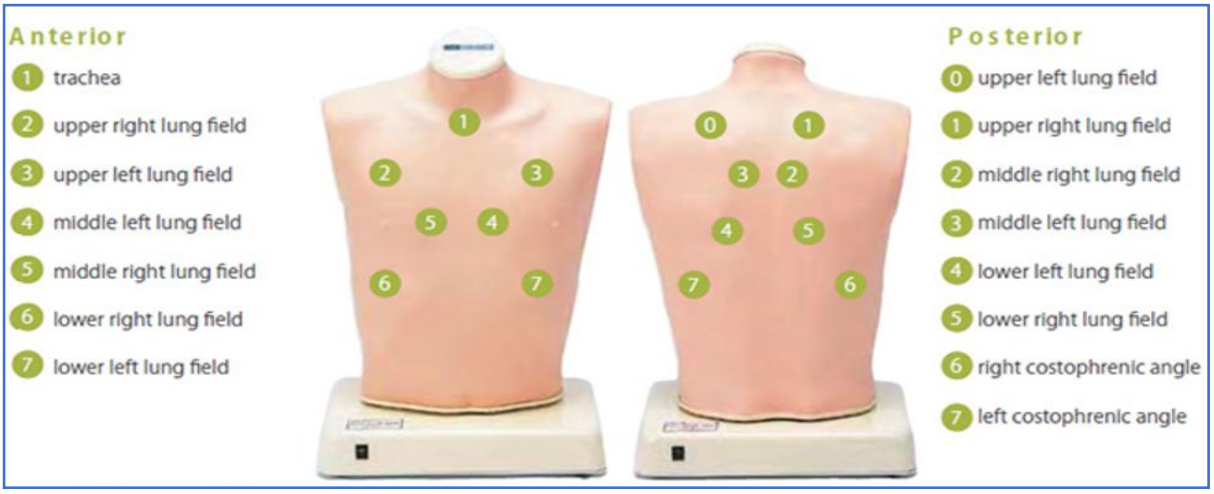
Percuss the following areas, comparing side to side:
- Supraclavicular (lung apices)
- Infraclavicular
- Chest wall (3-4 locations bilaterally)
- Axilla

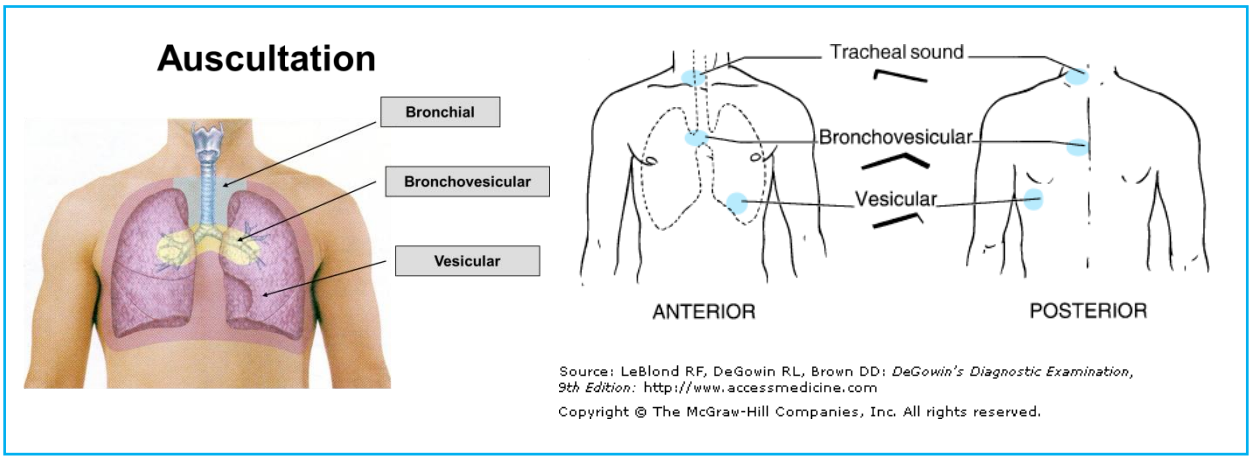
Auscultation

The stethoscope is an instrument that does **not significantly amplify sound**, but, more important, acts as a selective filter of sound. Briefly, the **bell** filters **high-frequency sounds** greater than 1500 cycles per second and therefore should be used to detect **low-frequency sounds**. On the other hand, the **diaphragm** selectively filters **low-frequency** sounds. Since sounds produced by breathing tend to be of relatively **high pitch**, the chest is ausculted with the diaphragm.

⇒ **Auscultation Steps**

- ✓ Auscultation is traditionally with the diaphragm, but the bell may not provoke as much extraneous sound from hairy chests and is able to get into the supraclavicular areas.
- ✓ Procedure of auscultation of the lungs and pleura
- ✓ Patient should be examined in a quiet room at normal or warm temperature to avoid muscle shivering
- ✓ Patient should be in sitting position. In case of serious patient
- ✓ examined in recumbent position by turning patient from side to side
- ✓ Ask the patient to take breath deeply and slightly forcefully than usual.
- ✓ Watch, whether the patient heaves and puff irregularly make noise from, the mouth.
- ✓ Start auscultation anterior and posterior lung from above downwards and compare the both right and left side simultaneously and sequentially
- ✓ Watch any change in character of breath sound and any added sound.





⇒ **Types of breath sounds:**

✓ **Vesicular Sounds**

- Vesicular sounds are generated by the turbulent flow of air through the airways of healthy lungs. These are typically soft and are characterized by inspiratory sounds that last **longer** than expiratory sounds.
- Normal lung tissues have a substantial amount of airspace to attenuate and soften the sound. These vesicular sounds vary considerably from patient to patient; thus, it is important to compare one hemidiaphragm to another by listening in a symmetrical pattern, as shown in the image below.

✓ **Bronchial Breath Sounds**

- Bronchial breath sounds often result from consolidation within lung parenchyma with a patent airway leading to the involved area.
- The resulting breath sounds are amplified through the consolidation, leading to a louder breath sound.
- Typically, there is a pause between inspiratory and expiratory sounds, as the involved parenchyma does not fill with air during this time in inspiration.
- The pitch is usually high, as the sounds arise from the bronchi, and the expiratory phase generally lasts longer and is as intense as, or more intense than, the inspiratory phase.

✓ **Absent/Attenuated Sounds**

- Absent/attenuated sounds occur when there is no airflow to the region being auscultated.
- This can occur in a pneumothorax, hemothorax, pleural effusion, or parenchymal consolidation, which includes the feeding airway.

✓ **Adventitious sounds**

⇒ **Discontinuous sounds**

- Crackles

Crackles are sounds that are intermittent, nonmusical, very brief, and more pronounced during inspiration. The sound of hair being rubbed between one's fingers is often used as an example to describe these types of sounds. Crackles can be classified as **fine or coarse**, depending on their sound quality.

Fine crackles are typically produced by the forced reopening of alveoli that had closed during the previous expiration. These crackles are softer, and higher in pitch.

Coarse crackles are louder and lower in pitch. Coarse crackles are typically a combination of alveolar reopening and bubbling of air through retained secretions in smaller airways.

Crackles can also be categorized as **early or late**, depending on when they are appreciated during the respiratory cycle.

Early inspiratory crackles occur immediately after initiation of inspiration and are more often associated with interstitial lung disease.
Late inspiratory crackles begin in the first half of inspiration and continue until the end of inspiration. This type of crackle is more often associated with pulmonary edema and asthma.

Differential Diagnoses of Crackles

Location in Respiratory Cycle	Fine	Coarse
Early inspiratory	Usual interstitial pneumonia Desquamative interstitial pneumonia Sarcoidosis Miliary tuberculosis Allergic alveolitis Asbestosis	Chronic bronchitis COPD
Late inspiratory	Atelectasis Asthma CHF Pulmonary edema Pneumonia/Consolidation Scleroderma	Fibrosing alveolitis
Mid-inspiratory and expiratory		Bronchiectasis, which can be secondary to the following: • Necrotizing pneumonia • Environmental exposures • Cystic fibrosis • Alpha-1 antitrypsin disorder

⇒ **Continuous sounds**

- Wheezes

Wheezes are continuous, high-pitched, musical, predominantly expiratory sounds that are produced by air flowing through narrowed bronchi, causing fluttering and resonance of the bronchial walls. Thus, they are caused by pathology leading to the narrowing of bronchi, most commonly **COPD, asthma, and bronchitis**.

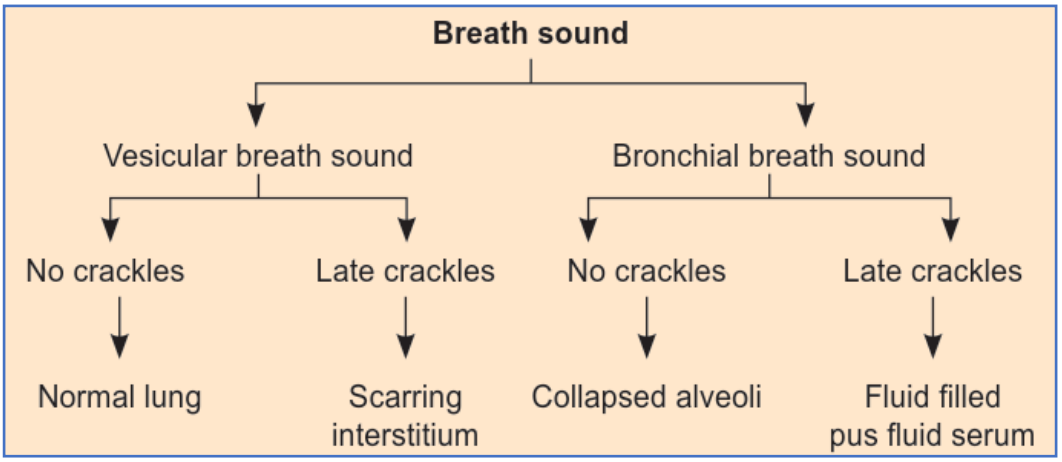
- Rhonchi

Rhonchi are low-pitched snore like sounds that may occur throughout the respiratory cycle. They are often characterized by secretions within the large airways and can be heard in a wide variety of pathologies, any of which cause increased secretions, such as in **cystic fibrosis, pneumonia, bronchitis, pulmonary edema, or emphysema**.

- Stridor

Stridor is a loud, rough, continuous, high-pitched sound that is pronounced during inspiration; it indicates proximal airway obstruction. The sound is created by turbulent air flowing through a narrowed trachea or larynx and is loudest over the trachea. This is commonly a medical emergency and should be recognized early. Diagnoses that may present with stridor include **epiglottitis, vocal cord dysfunction, croup, and airway edema** (which could be secondary to trauma or an allergic reaction).

⇒ **Vocal sounds**
- Bronchophony
- Whispering pectoriloquy
- Ego phony
- EA changes



Pleural Rub

- ✓ zIt is caused by friction of both inflamed pleural surface (both
- ✓ visceral and parietal) against each other
- ✓ It is grating in character
- ✓ It is heard during both in inspiratory and expiratory phases:

- ™™In inspiration, it is faster and louder
- ™™In expiration, it is longer.

- ✓ Sometimes, it may be in inspiratory phase
- ✓ It is not changed with coughing or change of posture
- ✓ It can be increased with pressure with diaphragm of stethoscope
- ✓ It is localized to small area only
- ✓ It is sometimes palpable.

Natural history of pleural rub

- ✓ Normally, pleura are lined by single layer of mesothelial cells. This layer is lubricated by thin film of fluid, so that during normal respiration the pleural surfaces glide on each other silently
- ✓ During pleural inflammation, pleural surface are lined by fibrin and inflammatory/neoplastic cells, as a result the roughened pleural surfaces rub against each other during respiration
- ✓ producing grating sound—pleural rub
- ✓ As the fluid accumulates in the pleural space, pleural surfaces are separated, pleural rub disappear. So pleural rub in pathgnomonic of pleural inflammation.
- ✓ Noninflammatory effusions such as nephritic syndrome, CCF, cirrhosis are not associated with pleural rub.

Most common causes of pleural rub

- ✓ Inflammation:
- ™™Infective—pneumococcal, staphylococcal, gram –ve bacteria
- ™™Noninfective—collagen vascular disease
- ✓ Neoplastic conditions:
- ™™By direct invasion
- ™™By producing inflammation
- ✓ Pulmonary embolism
- ✓ Parapneumonic pleuritis

Differences between pericardial and pleural rub:

By holding the breath if rub persist, it is pericardial. If rub disappears, it is pleural.

Differences between Crackles and Pleural Rub

Crackles	Pleural rub
• It is cracking in character	• It is grating in character
• It is usually inspiratory	• It is heard in both phases
• It can be changed with coughing and posture	• It cannot be charged with coughing or change of posture
• No relation with pressure with diaphragm of stethoscope	• It is increased with pressure of stethoscope
• It cannot be palpable	• It can be palpable

Differences between Wheeze and Pleural Rub

Wheeze	Pleural rub
• It is musical sound	• It is grating in character
• It is heard in expiration phase only	• It can be heard in both phases or in inspiratory
• It is a continuous sound	• It is interrupted sound

To complete the examination

- Suggest further assessments and investigations
- Check oxygen saturations
- Provide supplementary oxygen if indicated
- Perform peak flow assessment (if asthmatic)
- Request a chest x-ray – if abnormalities were noted on examination
- Take an arterial blood gas if indicated
- Perform a full cardiovascular examination if indicated

Communication Skills

Chronic obstructive pulmonary disease (COPD)

Candidate's information

Your role: you are a student in the general medical clinic. You have been asked by a GP to see this 50-year-old man with known COPD regarding his weight loss. He has also noticed a recent deterioration in his exercise tolerance.

Your task: to assess the patient's problems and address any questions or concerns raised by him.

Patient's information

You are: Trevor Bailey, a 50-year-old man. Your problem: you've had chronic chest problems for the past 10–15 years. Your breathing has got noticeably worse over the past 4 months and you have had to stop working as a cleaner because of your breathing. You used to be able to climb stairs and go shopping without having to stop due to breathlessness, but now you have to stop two or three times when climbing the stairs to go to bed. You've had a daily cough for years, producing brown/grey mucus. Your wheeze gets worse when you walk but improves with inhalers. You have been needing antibiotics frequently from your GP over this past 4 months or so, after you start coughing up green sputum. Your bowels are regular and you have no rectal bleeding, no heartburn and no tummy pain. Swallowing is fine. You've had a poor appetite and weight loss over the past 4 months (6 kg weight loss). The weight loss has been unintentional – you haven't been dieting. You have not been coughing up blood. Generally feeling weak and unwell.

Past medical history and drug history:
- COPD
- No history of cardiac disease, hypertension, diabetes mellitus
- Salbutamol inhaler as needed
- Tiotropium 18µg inh once per day
- Seretide 250 2 puffs twice per day

Social history:
- Worked as a cleaner for past 30 years. Never been on building sites and no known contact with asbestos. Never been a miner, potter or welder
- Born in the UK, last in Europe 3 years ago, never travelled to Asia or Africa
- Smoker: 30–40 cigarettes per day for past 35 years
- Occasional alcohol (1 or 2 glasses of wine per week for past 10 years, less since you have been losing weight)

Family history:
- No family history of TB or other lung conditions

Examiner questions for the candidate:
- Have I got cancer?
- Is it worth stopping smoking now? Isn't it just too late?

Candidate

History/key questions

- Ask about the key respiratory symptoms:
- ✓ Breathlessness – ask about triggers, current and previous exercise tolerance
- ✓ Cough, sputum, fever, chest pain, haemoptysis, stridor
- ✓ Any weight loss – quantify
- ✓ Change in sputum produced – colour, consistency, volume
- ✓ Ask about other causes of weight loss (consider nonchest causes too)
- ✓ Ask about appetite, nausea or vomiting
- ✓ Any change in bowel habit, rectal bleeding, dysphagia or new dyspepsia
- ✓ Any fever
- Drug history
  Current medications including inhalers, nebulizers
  Any use of oxygen (PRN cylinder or long-term oxygen therapy)
- Past medical history
- ✓ Any operations in the past
- ✓ What investigations has the patient had for their lungs – spirometry, pulmonary function tests, scans, bronchoscopies, pleural biopsies, chest drains? What has been diagnosed?
- Social history
- ✓ Smoking history: quantify using pack-years (20 cigarettes per day for 1 year = 1 pack-year) Rolling tobacco: quantify in ounces per week
- ✓ Ask about occupational history: asbestos exposure, coal mining, potteries, welding, and other sources of occupational dust; farming Pets
- ✓ Any family history of TB, any known TB contacts
- ✓ Foreign travel in the past and where to? Any travel to Africa, India/Pakistan, Asia
- Address patient's questions and concerns
- ✓ Have you had any thoughts about what is going on?
- ✓ Is there anything in particular you're concerned about?
- ✓ Do you have anything you would like to ask?

Key areas for examination

- ✓ A list of what to look for (in examination order; describe findings on examination)
- ✓ Overall inspection
  - Signs of weight loss; muscle wasting (generalized) with loose skin folds Respiratory rate and use of accessory muscles of breathing
  - Chest shape, scars on chest/drain sites/puncture wounds from biopsies
  - Hands: tar staining of fingers, finger clubbing, wasting of small muscles of the hand (unilateral with Pancoast tumour), CO_2 retention flap
  - Look for Horner's syndrome Mouth/tongue: cyanosis, angular cheilitis, pursed lips Lymphadenopathy: supraclavicular, cervical
  - Trachea: position/?central; hyperexpanded chest leading to tracheal tug JVP elevated in cor pulmonale or simply due to increased intrathoracic pressures
  - Expansion: hyperexpanded chest
- ✓ Percussion:
  - look for signs of effusion/consolidation
- ✓ Auscultation:
  - may be normal or prolonged expiration or wheeze.
  - Later, reduced breath sounds, crackles.
  - Listen for signs of effusion/consolidation including bronchial breathing
- ✓ Ask to see a recent chest X-ray, blood results, medication list, spirometry, sputum culture and sensitivity
- ✓ Examination in this patient – hyperexpanded chest, prolonged expiration

Differential diagnosis

- ✓ Chronic cough productive of sputum, consistent with chronic bronchitis; recurrent symptoms consistent with LRTI.
- ✓ Given multiple courses of antibiotics, need to consider malignancy and atypical infection including TB (patient with chronic lung disease, weight loss, extensive smoking history)

Discussion with patient

- ✓ Explain diagnosis
- ✓ Explain plan, including investigations and management
- ✓ Address patient's concerns/answer their questions
- ✓ You have had a chronic lung problem for some time but this has got worse over the past months with weight loss.
- ✓ This needs to be investigated, and so we'll start with some blood tests, a sputum sample to look for infection and a chest X-ray.
- ✓ We will also refer you for more detailed scans of your chest and arrange an appointment to see a chest specialist who may arrange further investigations.

Investigations

- ✓ Routine bloods for FBC, ESR, CRP, U&E, TFT, TTG (exclude other causes of weight loss) Arterial blood gas ECG: p pulmonale (RA hypertrophy), signs of RV hypertrophy
- ✓ Lung function tests
- ✓ Sputum for microscopy, culture and sensitivity; may include acid-fast bacilli (AFB) and TB culture CT chest (HRCT) ± staging CT chest for cancer
- ✓ ECHO if clinical suspicion of pulmonary hypertension, CCF or cor pulmonale

Management

- ✓ Treatment depends on cause; treat any infection, diagnose/treat any malignancy found
- ✓ Stop smoking and refer to smoking cessation services (combination of counseling and medical therapy)
- ✓ Use of pulmonary rehabilitation
- ✓ Supplements, e.g. nutritional supplements such as Fortisip/Fortijuice/Ensure, etc.
- ✓ Referral to dietitians
- ✓ Respiratory referral for consideration of further investigations based on results of the above tests, e.g. bronchoscopy to exclude obstructing lesion

Addressing concerns/answering questions

- ✓ Have I got cancer? There are several different possible causes for becoming more breathless and also losing weight.
- ✓ These vary from less serious problems such as infections through to more serious problems including the most serious.
- ✓ Cancer is one of the serious causes, but we need to carry out these tests to find out what is causing your problems.
- ✓ Is it worth stopping smoking now? Isn't it just too late? It is worth stopping smoking as it reduces the ongoing damage to your lungs (it slows the decline in lung function [FEV1]). We will arrange referral to the smoking cessation clinic to help you stop.

Discussion with examiners

- ✓ The examiners may ask you to clarify details of the above diagnosis and ask questions about your planned investigations and management.
- ✓ Other possible questions include:
- ✓ What is the MRC dyspnoea scale? According to NICE guidance, this scale grades the degree of breathlessness related to activities:
  1. Not troubled by breathlessness except on strenuous exercise
  2. Short of breath when hurrying or walking up a slight hill
  3. Walks slower than contemporaries on level ground because of breathlessness or have to stop for breath when walking at own pace

4. Stops for breath after walking about 100 meters or after a few minutes on level ground
5. Too breathless to leave the house, or breathless when dressing or undressing.

✓ Who should be assessed for supplemental oxygen (long term oxygen therapy) at home? According to NICE guidance, anyone with:

- Severe outflow obstruction (FEV1<30% predicted)
- Cyanosis
- Polycythaemia
- Peripheral oedema
- Raised JVP
- O_2 saturations of ≤92% breathing air

Examiners' information and requirements

The candidate should:

- Identify key features from the history: chronic SOB and daily sputum production consistent with chronic bronchitis; anorexia and marked weight loss; significant smoking history
- Identify key examination findings including evidence of chronic lung disease and signs of weight loss
- Provide a list of differential diagnoses, which includes COPD, neoplasia, TB
- Outline a sensible management plan including the investigations outlined above
- Give a clear explanation to the patient of your diagnosis and plan, address his concerns and answer his questions
- Further reading/resources

Haemoptysis

Candidate's information

Your role: you are a student in the medicine exame room and you've been referred Mr Gillingham, a 60-year-old man who reports several episodes of hemoptysis. He has been treated for a lower respiratory tract infection, but symptoms persist.

Your task: to assess the patient's problems and address any questions or concerns raised by him.

Patient's information

You are:
Gary Gillingham, a 60-year-old man. Your problem: you've coughed up blood once or twice a day for the past 3 weeks. Initially there were only little streaks in your spit every time, but this week you've been coughing up a little more; today was a teaspoon-full of blood, so you thought that it was time to come to hospital. Two weeks ago, you went to your GP who thought that it could all be due to a chest infection so prescribed a 5-day course of amoxicillin, but this didn't help. You saw a different GP today and she referred you up here. You have not had a chest X-ray yet. You usually cough up greeny-white sputum every morning; you call this your 'smoker's cough'. You've lost 5 kg in weight over the past 2 months and your appetite is slightly reduced. No chest pain, wheeze, fever or night sweats. Your exercise tolerance is unchanged – able to walk at least a mile on the flat.

Past medical history and drug history:
- No current medications
- No allergies known
- Never had TB, not known to have kidney problems Social history:
- Smoking: 20 cigarettes per day for the past 40 years
- You live alone with your puppy, Buster, who can't be left alone for long as he wrecks the house
- Born in the UK
- Last foreign travel 6 months ago to Spain, never been outside Europe

Family history:
- No-one in the family has been unwell
- No history of TB or cancer
- Both parents died in their sleep in their 70 s

Your questions for the candidate:
Is it cancer?
Do I have to stay in hospital?

Candidate

History/key questions

- Ask patient to describe the events.
- ✓ Need to be clear that it is haemoptysis that's being reported rather than haematemesis Onset, duration and frequency of haemoptysis
- ✓ Appearances of blood, volume (e.g. clots, large cupful versus streaks in sputum)
- ✓ Sputum: colour, change from normal
- ✓ Any evidence of infection: fever, sputum.
- ✓ Any chronic lung disease?
- ✓ Ask about respiratory symptoms including breathlessness, wheeze, chest pain, weight loss, cough and sputum
- ✓ Any known medical conditions (Ehlers–Danlos); any kidney problems in the past (Goodpasture's, Wegener's granulomatosis)?
- ✓ Ever had TB in the past?
- ✓ Any recent procedures (bronchoscopy, lung biopsy)
- ✓ Ask about foreign travel (PE)
- ✓ Any foreign body ingestion: choking episodes prior to the onset of haemoptysis?
- Drug history
- ✓ Anticoagulant, antiplatelet use
- ✓ Any cocaine use
- Social history
- ✓ Smoking history in pack-years
- ✓ Alcohol use
- Address patient's questions and concerns
- ✓ Have you had any thoughts about what is going on?
- ✓ Is there anything in particular you're concerned about?
- ✓ Do you have anything you would like to ask?

Key areas for examination

- ✓ A list of what to look for (in examination order; describe findings on examination)
- ✓ Overall inspection:
    - signs of weight loss
    - Respiratory rate and use of accessory muscles of breathing
    - Hands: tar staining of fingers, finger clubbing, wasting of small muscles of the hand (unilateral with Pancoast tumour), CO_2 retention flap
    - Look for Horner's syndrome
    - Mouth/tongue: cyanosis, angular cheilitis, pursed lips
    - Lymphadenopathy: supraclavicular, cervical, axillary
    - Trachea: position Chest examination: Chest shape, scars on chest/drain sites/puncture wounds from biopsies/radiation burns
    - Hyperexpanded chest/signs of chronic lung disease
- ✓ Expansion/percussion/auscultation:
    - Any evidence of collapse/consolidation/effusion
    - Look for oedema/DVT
- ✓ Ask to see observations chart including oxygen saturations, chest X-ray, routine blood tests, sputum culture if available

Differential diagnosis

Hemoptysis and weight loss in a 40 pack-year smoker without symptoms of respiratory infection: top differential diagnosis is bronchial neoplasia but important to exclude TB and pulmonary infection, vasculitis/Wegener's/Goodpasture's Haemoptysis can be caused by airways disease, pulmonary parenchymal or vascular diseases

Discussion with patient

- ✓ Explain diagnosis
- ✓ Explain plan, including investigations and management
- ✓ Address patient's concerns/answer their questions
    - Coughing up blood is concerning and we needed to look for causes for this in the breathing pipes.
    - I'll arrange a chest X-ray and blood tests here now and then we can discuss the results.
    - I think we'll need to arrange a scan of your chest as an outpatient, and refer you to the chest doctors for further investigations which may include a camera test looking into the airways.
    - It's important to stop smoking – I can refer you to the smoking cessation clinic.

Investigations

- ✓ Bloods including U&E, FBC, CRP, ESR, INR, calcium, anti-GBM antibody
- ✓ Urinalysis (Goodpasture's, Wegener's)
- ✓ CXR HRCT chest/staging CT as neoplasia suspected
- ✓ Sputum culture/MCS and AFB

Management

- ✓ Urgent respiratory referral (rapid access pathway/2- week wait clinic)
- ✓ Bronchoscopy Referral to smoking cessation service

Addressing concerns/answering questions

- ✓ Is it cancer? We need to look for all the different causes for you coughing up blood, ranging from infection to more serious problems, which include cancer. We are waiting for an X-ray today here and some blood tests but it's likely that we will need to arrange an urgent scan of your chest, and referring you to the chest doctors who will see you in clinic; they may well arrange a special camera test to look into the breathing tubes to find out the cause of the bleeding.
- ✓ Do I have to stay in hospital? We need to wait and see what the results show, but it's likely that we can arrange any necessary scans as an outpatient, and follow-up in the respiratory clinic.

Discussion with examiners

The examiners may ask you to clarify details of the above diagnosis and ask questions about your planned investigations and management.
A possible question includes:

- ✓ What are the causes of haemoptysis to consider?
  Pneumonia, neoplasia (lung cancer), tuberculosis, pulmonary embolism, bronchiectasis, vasculitis and pulmonary haemorrhage (Goodpasture's).

Examiners' information and requirements

The candidate should:

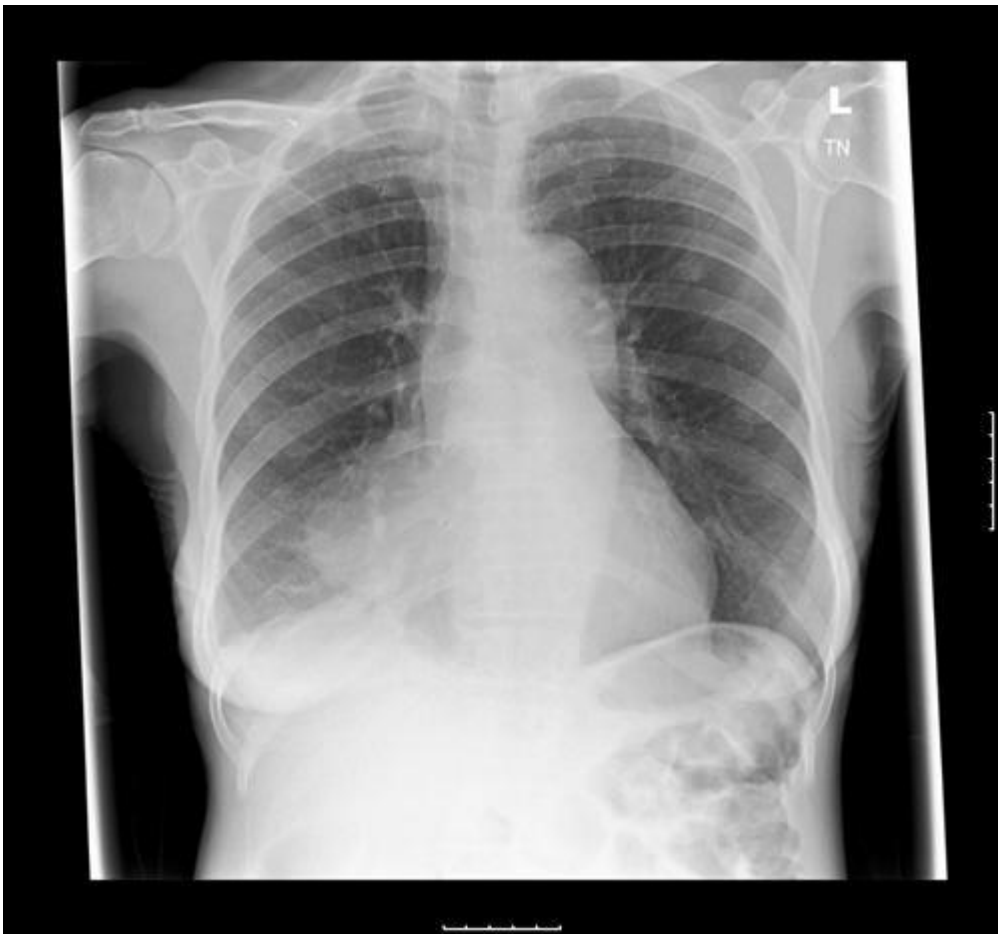
- Identify key features from the history. These include weight loss, smoking history, hemoptysis
- Identify key examination findings including signs of weight loss, tar staining, clubbing
- Provide a list of differential diagnoses including neoplasia and TB
- Outline a sensible management plan, including appropriate referrals for investigations and smoking cessation, and to respiratory clinic
- Give a clear explanation to the patient of your diagnosis and plan, address his concerns and answer his questions

Questions

1/A 65-year-old woman presents with a chronic productive cough and shortness of breath. She has a previous history of chronic obstructive airways disease.

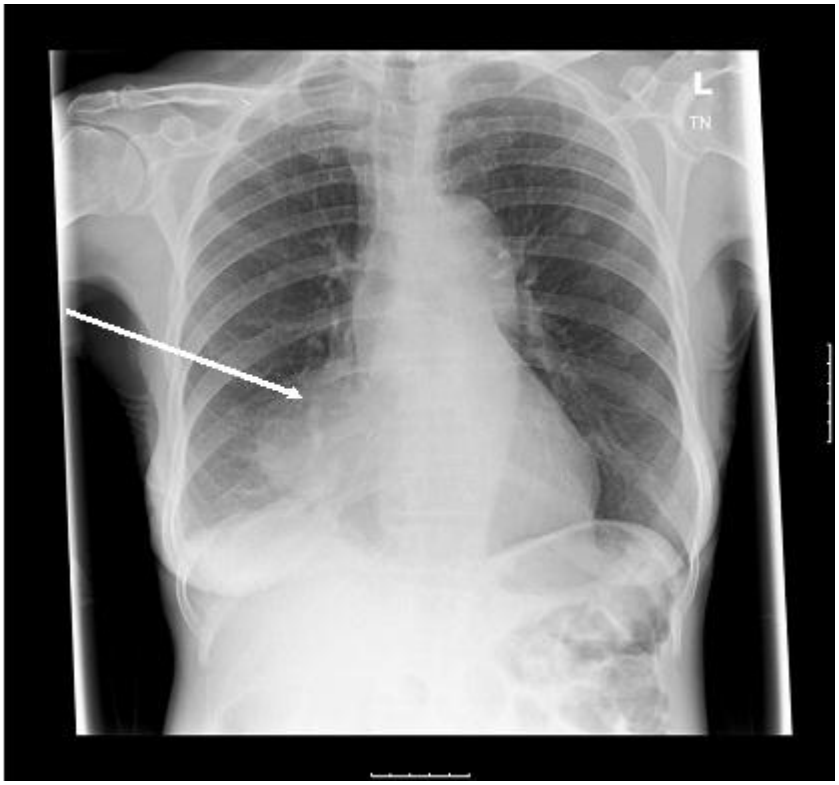
On examination she is cachectic and clubbed, there are coarse crackles in the right mid-zone. A chest x ray is performed and the image is shown below.

What is the most likely diagnosis?



(Please select 1 option)

○	Lung cancer
○	Pericardial effusion
○	Pleural effusion
○	Right middle lobe collapse
○	Right middle lobe pneumonia



The chest x ray demonstrates a large, spiculated density in the right mid-zone adjacent to the right heart border (A) in combination with the history the most likely diagnosis is a lung malignancy.

Whilst a loculated pleural effusion may have a similar appearance there is nothing in the history to suggest this as a cause and it would be unlikely to explain the history and examination findings without another simultaneous pathology.

Pericardial effusions tend to give the heart an enlarged globular appearance and are usually uniform.

There is no loss of volume to indicate collapse and the appearance is not indicative of right middle or lower lobe collapse (straight bordered rather than round bordered).

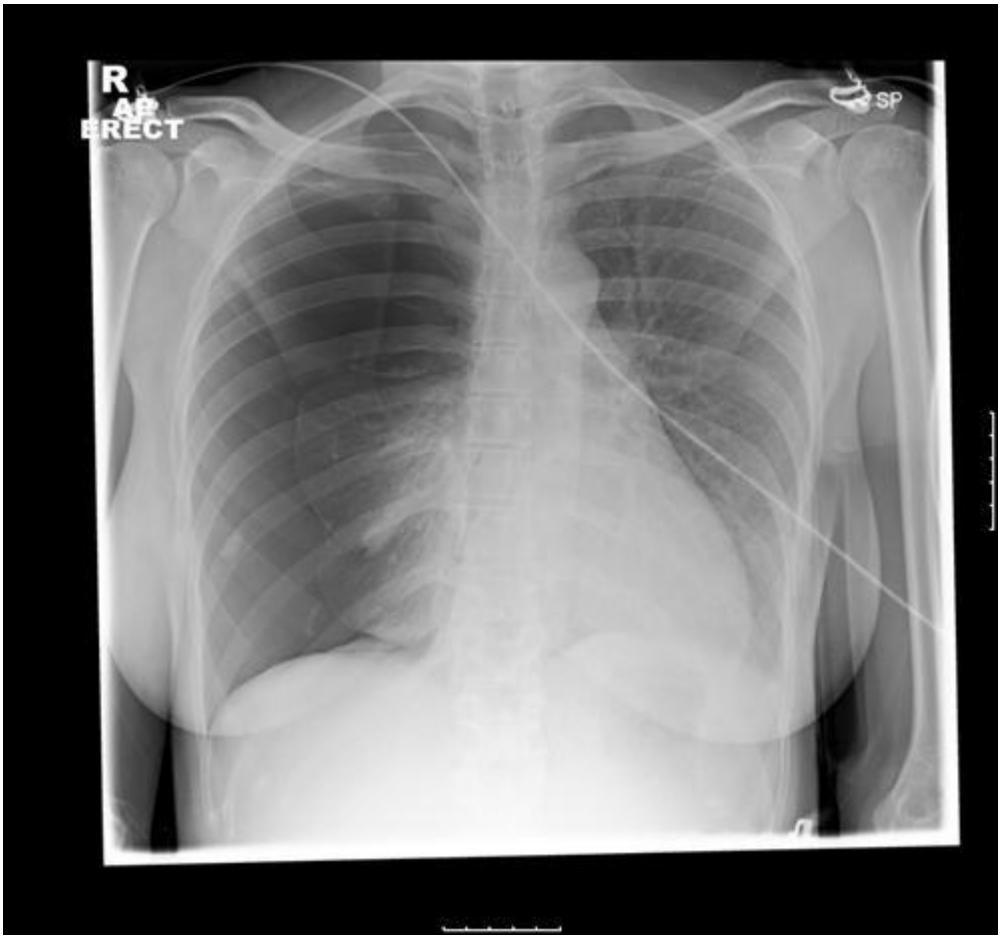
There are no air bronchograms to suggest consolidation

[A]

2/A 29-year-old woman is admitted to hospital with pleuritic chest pain and shortness of breath. She has a past medical history of asthma which is usually well controlled.

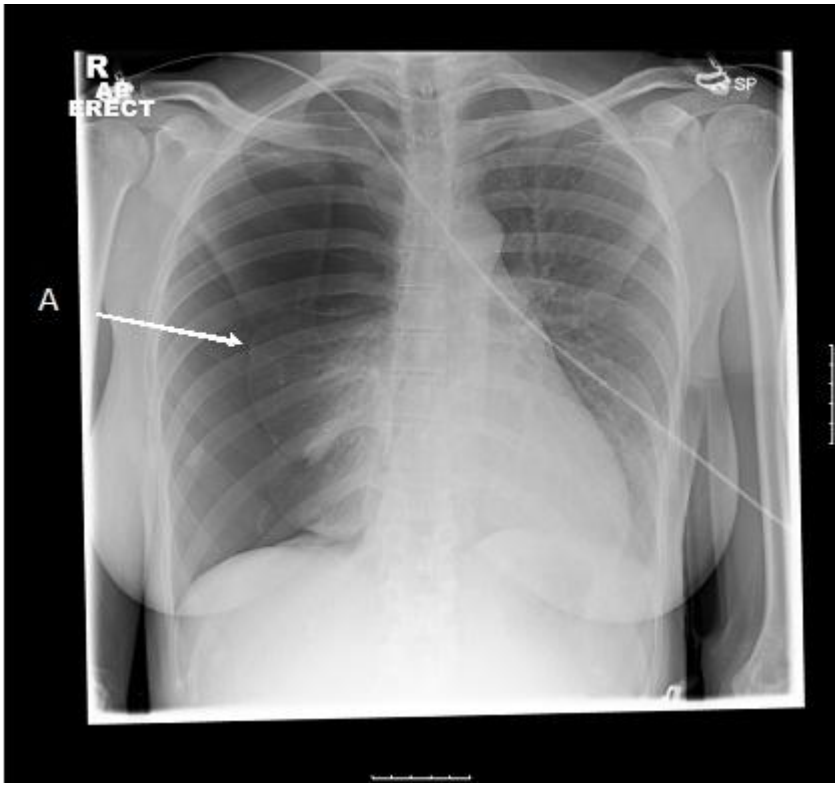
She drinks 24 units of alcohol per week and smokes 15 cigarettes a day. Physical examination demonstrates reduced breath sounds in the right hemithorax. A chest x ray is performed and the film is shown below.

What is the most appropriate treatment strategy?



(Please select 1 option)

○	Aspiration
○	Discharge
○	High flow oxygen
○	Intercostal drain insertion
○	Observation



The chest x ray demonstrates a large right-sided pneumothorax. This is classified as a secondary spontaneous pneumothorax due to the previous history of respiratory disease. The most appropriate management option in this setting would be insertion of an intercostal drain.

Aspiration may be considered for a large pneumothorax where there is no previous history of respiratory disease or a small pneumothorax where there is a previous history of respiratory disease. Small or large pneumothoraces are differentiated on the basis of the maximum interpleural distance, greater than 2 cm indicates a large pneumothorax.

High flow oxygen may assist the resolution of pneumothoraces and may be used, where not contraindicated, whether active or supportive management is chosen.

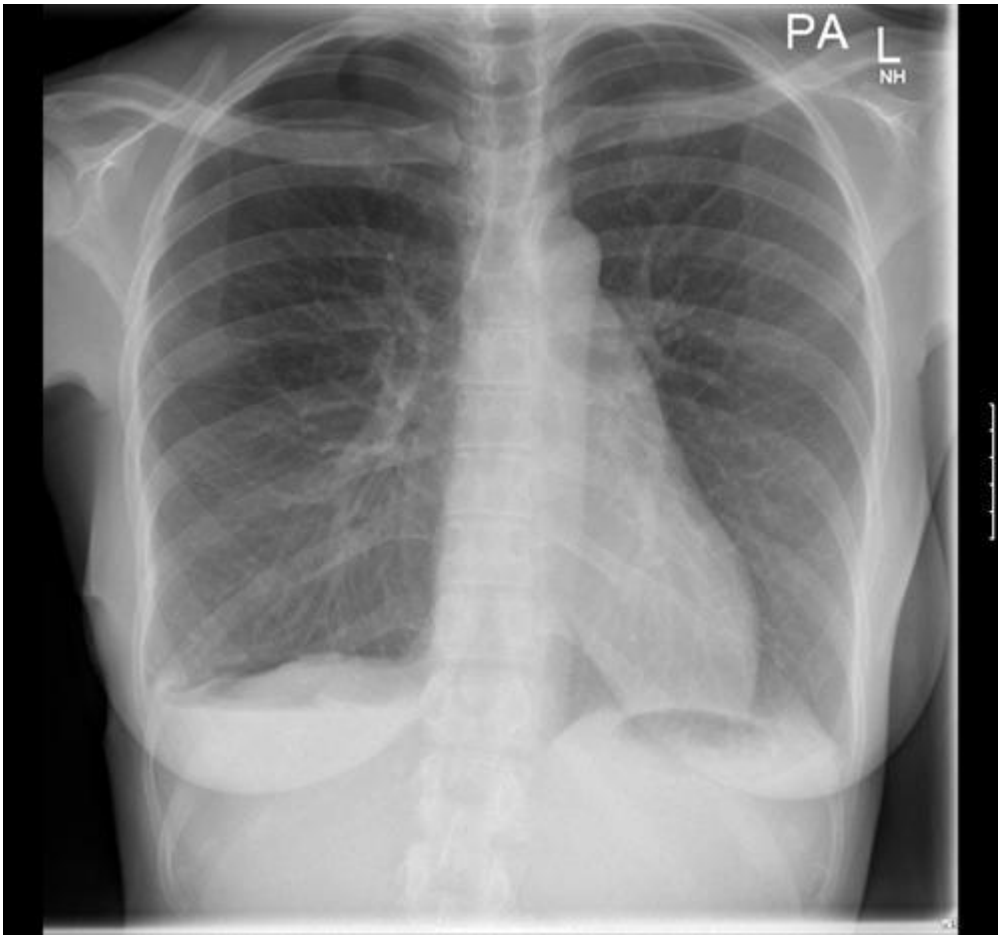
The symptoms, size of the pneumothorax and history of previous respiratory disease mean that conservative management or discharge is not appropriate.

[D]

3/A 29-year-old woman is admitted to hospital with pleuritic chest pain and shortness of breath. She has a past medical history of asthma which is usually well controlled.

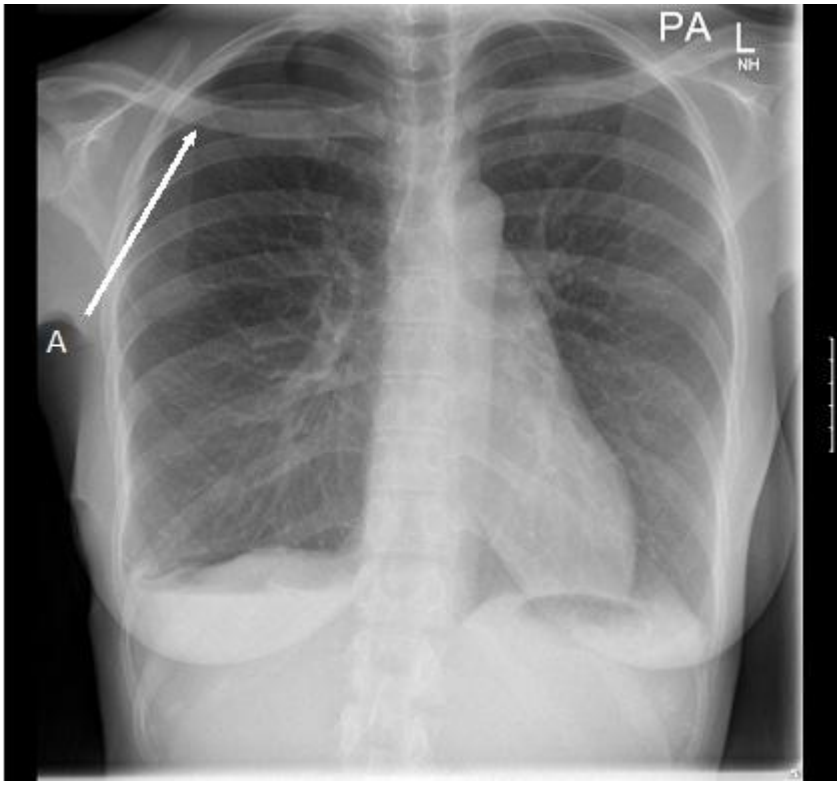
She drinks 24 units of alcohol per week and smokes 15 cigarettes a day. Physical examination is unremarkable. A chest x ray is performed and the film is shown below.

What is the underlying diagnosis?



(Please select 1 option)

○	Acute exacerbation of asthma
○	Pleural effusion
○	Pneumonia
○	Pneumothorax
○	Pulmonary embolus



The chest x ray demonstrates a right-sided pneumothorax.

The examination findings do not support a diagnosis of asthma exacerbation and the positive chest x ray makes this even less likely.

There is no evidence of a pleural effusion as both costophrenic angles can be clearly visualised.

There is no consolidation on the film to suggest pneumonia.

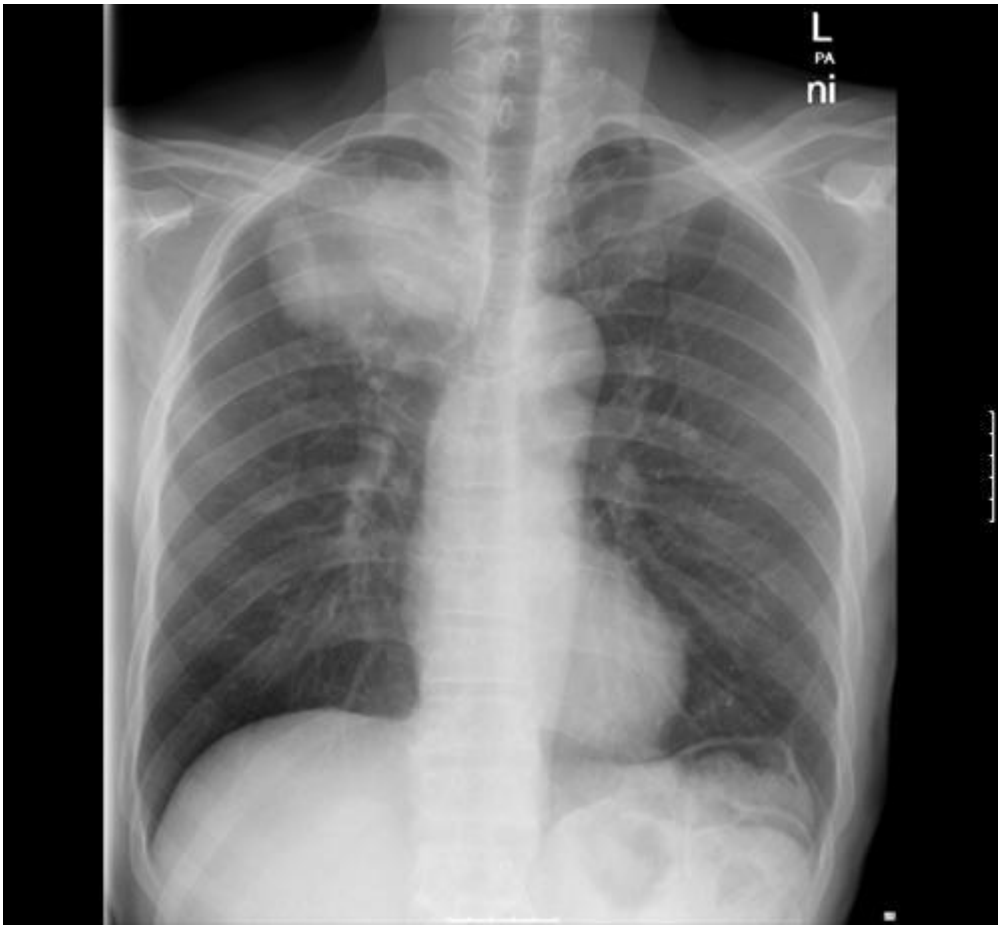
The chest x ray is usually normal in pulmonary embolism (unless there is concomitant lung pathology).

[D]

4/A 75-year-old man presents with a chronic productive cough and shortness of breath. He has a previous history of chronic obstructive airways disease.

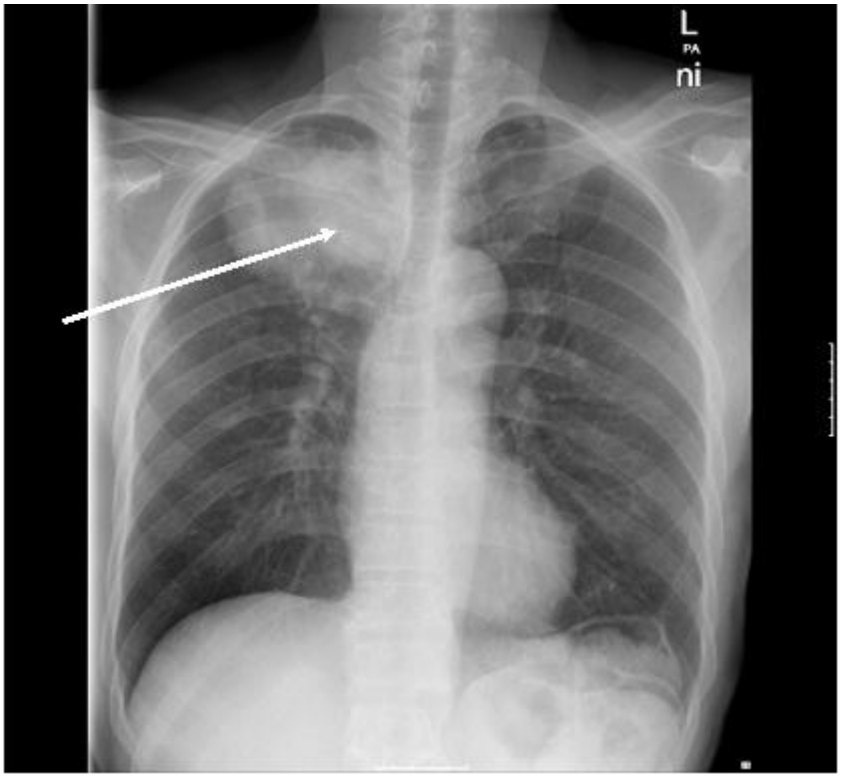
On examination he is cachectic and clubbed; there are coarse crackles in the right upper zone. A chest x ray is performed and the image is shown below.

What is the most likely diagnosis?



(Please select 1 option)

○	Lung cancer
○	Pleural effusion
○	Pulmonary haemorrhage
○	Right upper lobe collapse
○	Right upper lobe pneumonia



The chest x ray demonstrates a large density in the right upper lobe (A). In combination with the history the most likely diagnosis is a lung malignancy.

Whilst a loculated pleural effusion may have a similar appearance there is nothing in the history to suggest this as a cause and it would be unlikely to explain the history and examination findings without another simultaneous pathology.

Pulmonary haemorrhage tends to have a diffuse and 'fluffy' appearance.

There is no loss of volume to indicate collapse and the appearance is not indicative of right upper lobe collapse (straight bordered rather than round bordered).

There are no air bronchograms to suggest consolidation.

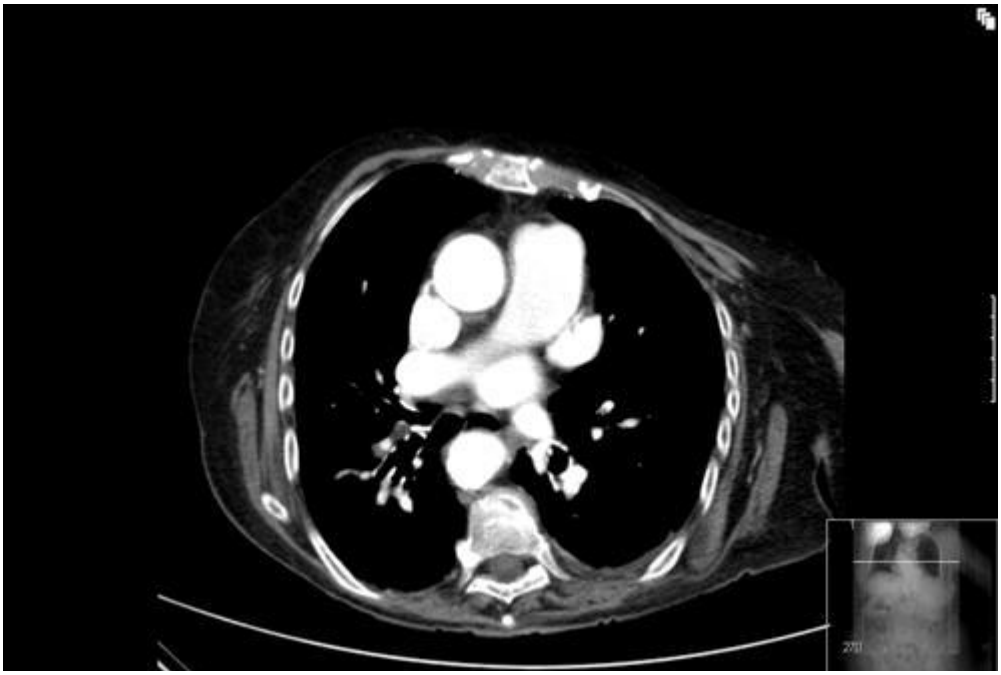
[A]

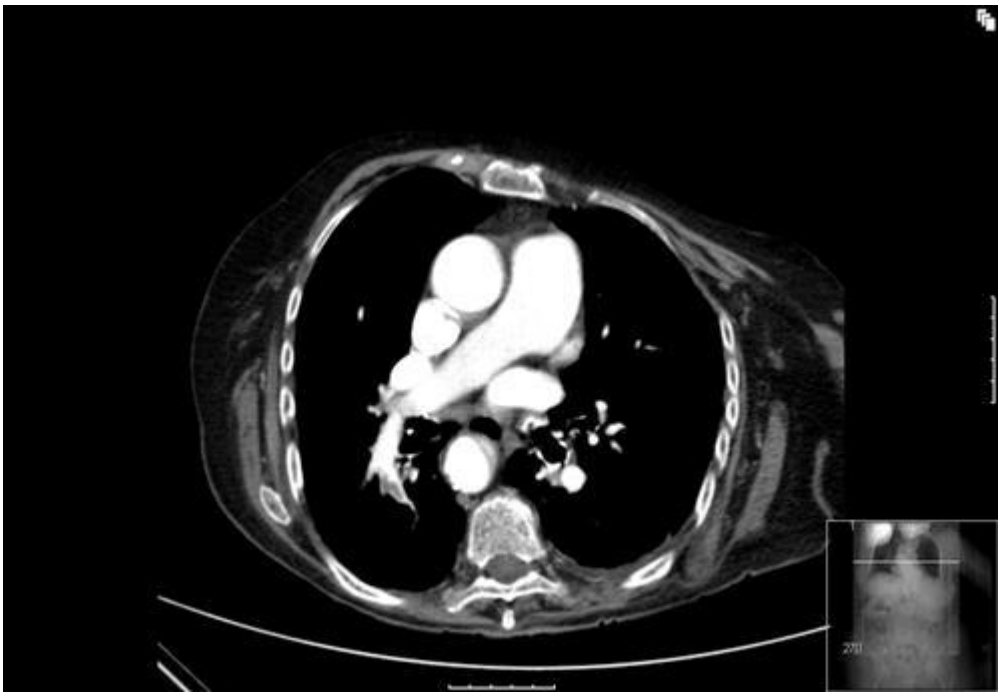
5/A 65-year-old man presents with chest pain, cough and shortness of breath. He has lost some weight recently.

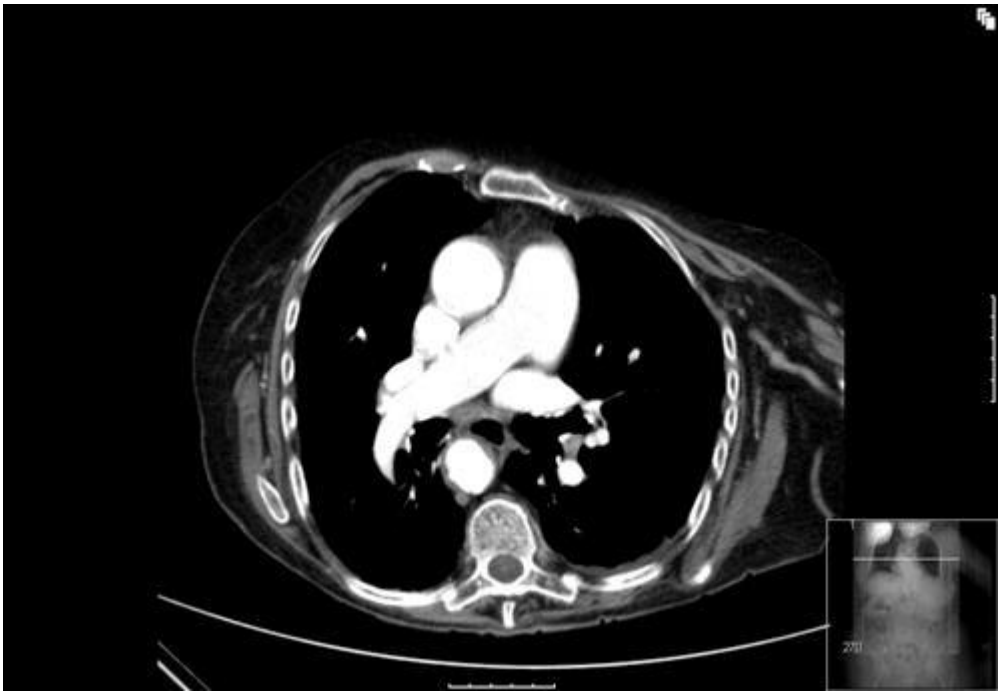
He has a past medical history of breast cancer and chronic obstructive pulmonary disease.

On examination he has a temperature of 37.8℃, some coarse crepitations in the right mid-zone and mild expiratory wheeze throughout both lungs. A CT of the chest is performed and three of the sections are shown below.

What is the most appropriate initial treatment?

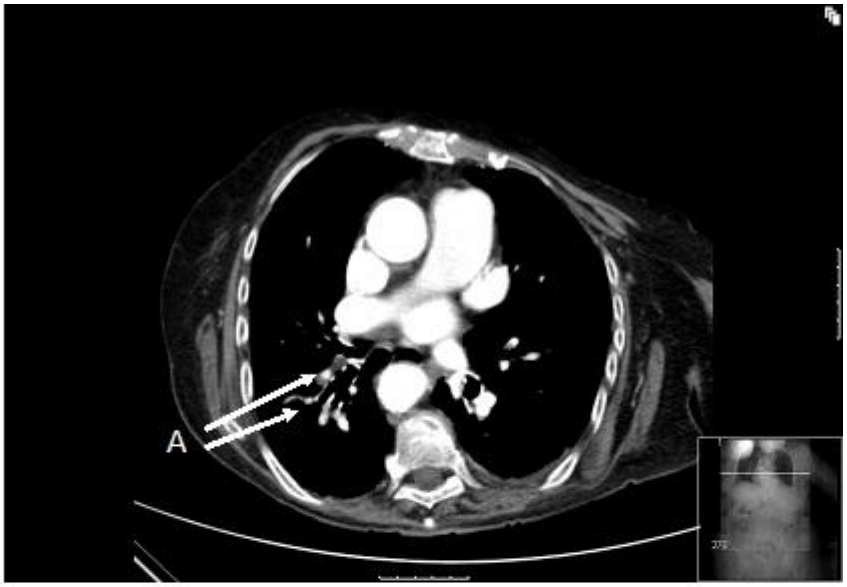


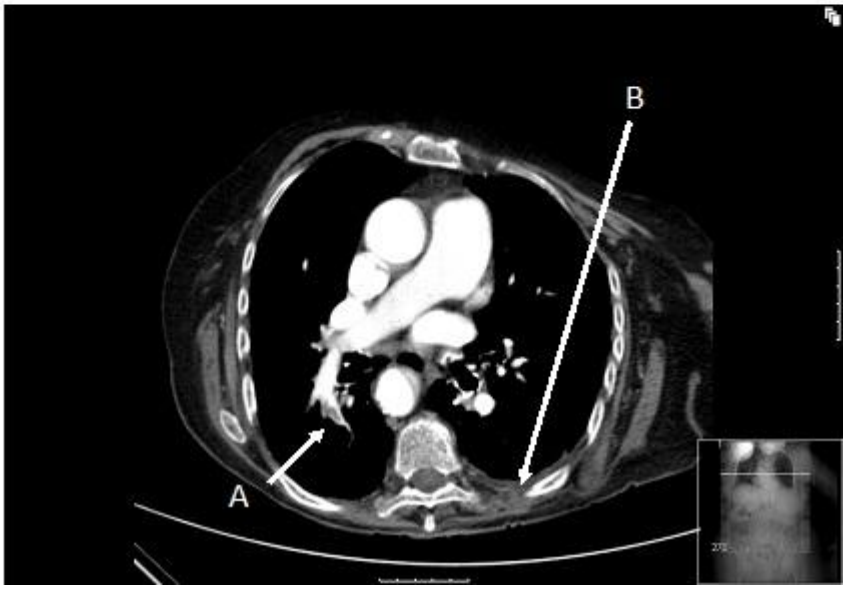


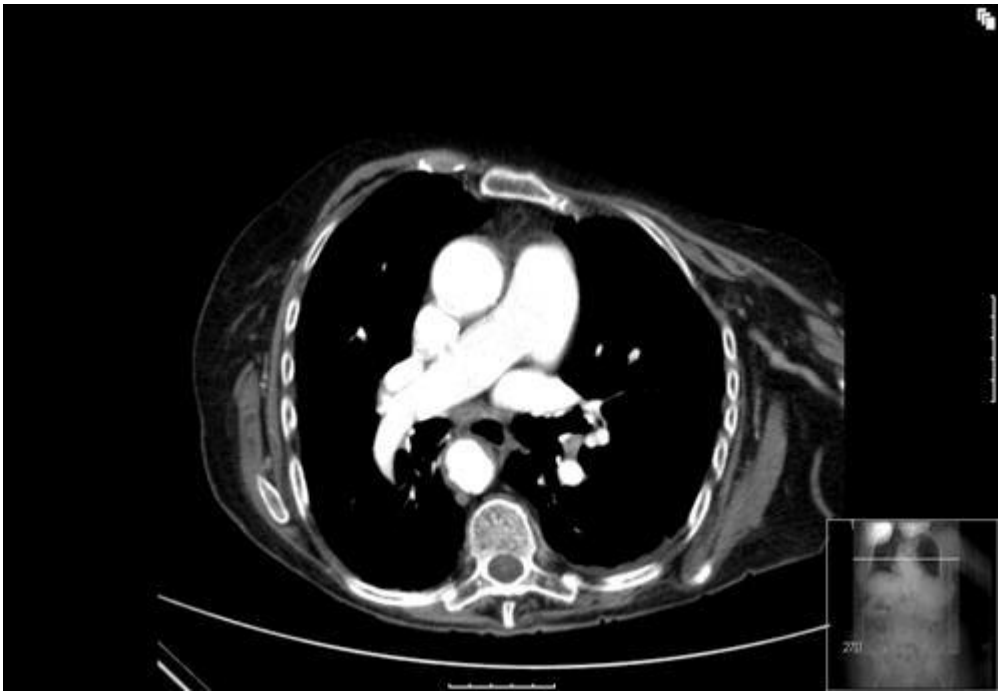


(Please select 1 option)

◌	Co-amoxiclav
◌	Non-steroidal anti-inflammatory drugs
◌	Radiotherapy
◌	Treatment dose low molecular weight heparin
◌	Ultrasound guided drainage


A


B
A



This is a contrast enhanced CT and demonstrates multiple filling defects in the right middle lobe branch pulmonary arteries (A) consistent with pulmonary emboli. The appropriate treatment is therapeutic anticoagulation with a low molecular weight heparin in the first instance before warfarinisation. There is a very small left-sided rim of pleural fluid.

There are no significant masses or lymphadenopathy to suggest a diagnosis of lung cancer and thus an indication for radiotherapy.

No significant parenchymal changes are visualised to suggest a pneumonia and the need for antibiotics.

The pleural effusion is small and very unlikely to be clinically significant, additionally the presentation is not consistent with an empyema so there is no indication for drainage.

The presentation is not consistent with pericarditis and there is no pericardial thickening or effusion to suggest the diagnosis on imaging.

Non-steroidal anti-inflammatories may be useful in managing associated pleuritic chest pain.

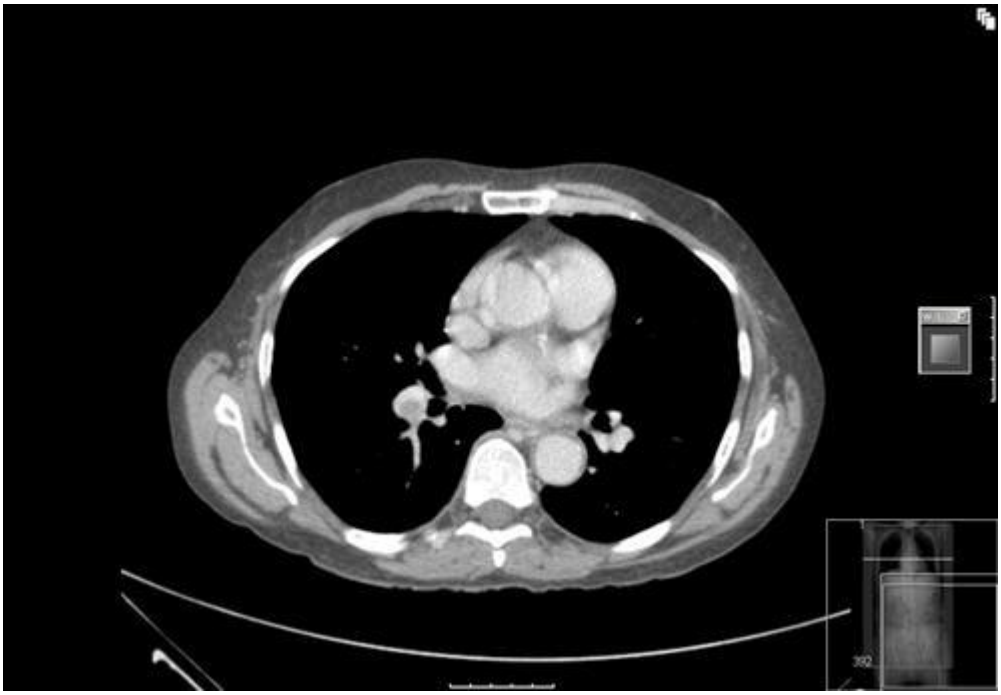
[D]

6/A 65-year-old woman presents with chest pain, cough and shortness of breath. She has lost some weight recently.

She has a past medical history of breast cancer and chronic obstructive pulmonary disease.

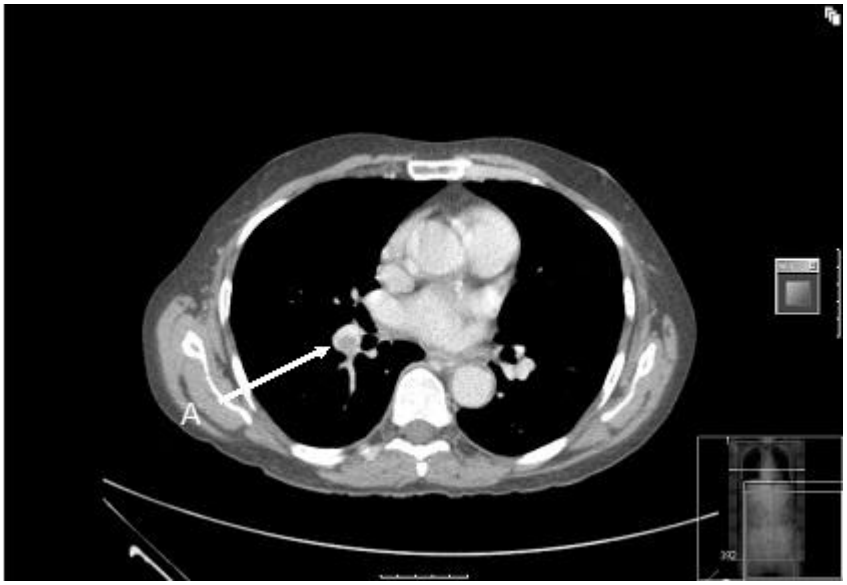
On examination she has a temperature of 37.8°C, some coarse crepitations in the right mid-zone and mild expiratory wheeze throughout both lungs. A CT of the chest is performed and one of the sections is shown below.

What is the most likely diagnosis?



(Please select 1 option)

○	Lung cancer
○	Pericarditis
○	Pleural effusion
○	Pneumonia
○	Pulmonary embolus



This is a contrast enhanced CT and demonstrates a filling defect in the right middle lobe pulmonary artery (A) consistent with a pulmonary embolus.

There are no significant masses or lymphadenopathy to suggest a diagnosis of lung cancer.

Pleural effusions are peripheral and gravity dependent (unless loculated). The presentation is not consistent with pericarditis and there is no pericardial thickening or effusion to suggest the diagnosis on imaging.

No significant parenchymal changes are visualised to suggest a pneumonia however these changes are often best seen when the images are viewed on 'lung windows'.

[E]

7/A 31-year-old gentleman presented over the Christmas period with shortness of breath and cough, which has been increasing over the last two weeks.

He was previously fit and well. When questioned however he did report occasional fluctuating joint pains and had a couple of episodes of sinusitis over the last one year. He had no family history of note. He smoked 10 cigarettes/day. He and his partner had recently bought a dog.

On examination he was dyspnoeic and tachycardic. His oxygen saturations were 92% on 10 L oxygen.

Chest examination revealed coarse crackles bilaterally.

Investigations are as follows:

Hb	12.8 g/dL	(13.0 - 18.0)

Na+	136 mmol/L	(137 - 144)
WBC	11.5 $\times 10^9$/L	(4 - 11)
K+	5.7 mmol/L	(3.5 - 4.9)
Platelets	452 $\times 10^9$/L	(150 - 400)
Urea	42.7 mmol/L	(2.5 - 7.5)
CRP	56 mg/L	(< 10)
Creatinine	650 µmol/L	(60 - 110)

Urinalysis
Protein ++
Blood +++
Nitrates negative
Leucocytes negative

Chest x ray	bilateral infiltrates, more dense at the bases

Anti-GBM antibodies	negative
c-ANCA	positive
p-ANCA	negative

He deteriorates and is intubated on ITU and also received renal replacement therapy.

Given the most likely diagnosis, what course of treatment should be commenced?

(Please select 1 option)

○	Cyclophosphamide and prednisolone
○	IV co-amoxiclav and clarithromycin
○	Plasmapheresis and prednisolone
○	Plasmapheresis, cyclophosphamide and prednisolone
○	Tamiflu

This patient has renal and pulmonary involvement. Given the upper respiratory tract symptoms, Wegener's granulomatosis is the most likely diagnosis.

Wegener's granulomatosis is a rare, autoimmune multi-system disease. It involves the upper airways, the lungs, renal and occasionally neurological system.

Wegener's can be diagnosed via a positive c-ANCA, although biopsies are often also required.

Treatment is with IV cyclophosphamide and steroids. However in severe life threatening Wegener's, or in patients with primarily renal involvement needing dialysis, plasmapheresis is used to rapidly remove the immune complexes.

It should be noted that although cyclophosphamide has revolutionised the treatment of this condition, it has a number of reported side effects (including bladder cancer and infertility).

His respiratory symptoms are caused by pulmonary haemorrhage and although Goodpasture's can present similarly the inclusion of his upper respiratory tract symptoms leans more to a diagnosis of Wegener's.

Although severe respiratory infection can result in acute kidney injury, his other symptoms (and urinalysis) would be in keeping with a vasculitis.

[D]

8/A 31-year-old gentleman presented over the Christmas period with shortness of breath and cough, which has been increasing over the last two weeks.

He was previously fit and well. When questioned, however, he did report occasional fluctuating joint pains, and had a couple of episodes of sinusitis over the last one year. He had no family history of note. He smoked 10 cigarettes/day. He and his partner had recently bought a dog.

On examination he was dyspnoeic and tachycardic. His oxygen saturations were 92% on 10L oxygen.

Chest examination revealed coarse crackles bilaterally.

Investigations are as follows:

Hb	12.8 g/dL	(13.0 - 18.0)
Na+	136 mmol/L	(137 - 144)
WBC	11.5 $\times 10^9$/L	(4 - 11)
K+	5.7 mmol/L	(3.5 - 4.9)
Platelets	452 $\times 10^9$/L	(150 - 400)

Urea	42.7 mmol/L	(2.5 - 7.5)
CRP	56 mg/L	(< 10)
Creatinine	650 µmol/L	(60 - 110)

Urinalysis
Protein ++
Blood +++
Nitrates negative
Leucocytes negative

Chest x ray - bilateral infiltrates, more dense at the bases.

Given the most likely diagnosis, what investigation would confirm the diagnosis?

(Please select 1 option)

○	ANCA
○	Anti-GBM antibodies
○	H1N1 virology
○	*Legionella* antigen
○	Sputum for MCS

This patient has renal and pulmonary involvement. Given the upper respiratory tract symptoms, Wegener's granulomatosis is the most likely diagnosis.

Wegener's granulomatosis is a rare, autoimmune multi-system disease. It involves the upper airways, the lungs, renal and occasionally neurological system.

Wegener's can be diagnosed via a positive c-ANCA, although biopsies are often also required.

His respiratory symptoms are caused by pulmonary haemorrhage, and although Goodpasture's can present similarly the inclusion of his upper respiratory tract symptoms leans more to a diagnosis of Wegener's.

Although severe respiratory infection can result in acute kidney injury, his other symptoms (and urinalysis) would be in keeping with a vasculitis.

[A]

9/A 60-year-old stone mason attends respiratory clinic with a two year history of increasing breathlessness.

He is an ex-smoker with a 40 pack year history.

On examination he looked well. His chest was hyperexpanded and had bibasal inspiratory crackles. He was not clubbed nor had he any lymphadenopathy.

His spirometry shows:

FEV_1	39%	predicted
FVC	72%	predicted
K_{co}	53%	predicted

Chest x ray reported hilar eggshell calcification and some upper zone fibrotic changes.

What is the most likely diagnosis?

(Please select 1 option)

○	Asbestosis
○	Berylliosis
○	Byssinosis
○	Silicosis
○	Simple pneumoconiosis

This patient has lung fibrosis and his employment is vital in the diagnosis.

Silicosis is a fibrotic lung disease associated with the inhalation of silicon dioxide (silica). It is usually found in quarry workers or miners and also sandblasters, pottery workers and stone masons (if the dust contains quartz).

Diagnosis is made on industrial history and typical chest x ray changes.

The pathognomic radiological changes are hilar eggshell calcification.

[D]

10/A 72-year-old retired stone mason under follow up for silicosis and mild COPD attends the Emergency department with increasing shortness of breath, productive cough and wheeze.

On examination his respiratory rate is 32, oxygen saturations 63% on air, pulse 126 bpm and BP 135/57 mmHg. A blood gas is performed on 28% oxygen, revealing type II respiratory failure.

He is moved to ITU for level 2 care with non-invasive ventilation. He initially improves but then acutely deteriorates with dropping oxygen saturations. It is noted his BP is 126/56 mmHg.

On examination there are diminished breath sounds on the left, but increased resonance on percussion and reduced expansion. Trachea is central.

What management should be initiated?

(Please select 1 option)

○	Aspirate left lung with a green needle (maximum 2L)
○	Decrease IPAP
○	Emergency decompression with 14G cannula
○	Insert a left interpleural chest drain
○	Remove non-invasive ventilation

This patient has clinical evidence of a left sided pneumothorax but at present not a tension pneumothorax and needs managing appropriately.

The treatment of choice for a pneumothorax in a ventilated patient is insertion of a chest drain.

If the patient developed a tension pneumothorax it would need emergency decompression with a cannula followed by chest drain.

This patient needs to continue on NIV but needs a drain urgently.

[D]

11/A 72-year-old retired stone mason under follow up for silicosis and mild COPD attends the Emergency department with increasing shortness of breath, productive cough and wheeze.

On examination his respiratory rate is 32, oxygen saturations 63% on air, pulse 126 bpm and BP 135/57 mmHg.

A blood gas is performed on 28% oxygen:

pH	7.21	(7.36 - 7.44)

P_{O2}	7.5 kP_a	(11.3 - 12.6)
P_{co2}	8.0 kP_a	(4.7 - 6.0)
Bicarbonate	10 mmol/L	(20 - 28)
Base excess	−3	

What do the blood gases show?

(Please select 1 option)

○	Metabolic acidosis
○	Metabolic acidosis with respiratory compensation
○	Mixed metabolic and respiratory acidosis
○	Respiratory acidosis
○	Respiratory acidosis with metabolic compensation

Interpreting blood gases is essential in the management of acutely unwell patients.

This gentleman is obviously acidotic. His PCO_2 is elevated indicating a respiratory cause for his acidosis, however his bicarbonate is reduced which is contributing to the acidosis.

He has not had time to compensate for his hypercapnia, indicating that this is likely to be an acute event.

[D]

12/A 75-year-old lady is referred to the rapid access chest clinic with a four month history of progressive breathlessness, lethargy, anorexia and one stone weight loss. She reports a dull right-sided chest pain that has been present for the last one month, and is partially relieved with 'low dose' co-codamol prescribed by her GP.

She is a housewife and smokes 15 cigarettes a day. Her husband, a retired plumber, recently died from a chest problem.

On examination she is dyspnoeic and cachetic. Examination of her chest demonstrates reduced expansion, vocal fremitus, breath sounds and dull percussion note throughout the right lung.

Chest x ray reports a medium sized right-sided pleural effusion. The pleura of the right hemi-thorax are thickened.

What is the most likely diagnosis?

(Please select 1 option)

	Asbestosis
	Adenocarcinoma
	Mesothelioma
	Pleural adenocarcinoma
	Squamous cell carcinoma

This lady has constitutional symptoms as well as a unilateral pleural effusion, so malignancy should be excluded. It is her husband's occupation that is the clue to her underlying diagnosis.

The inclusion of her husband's occupation as a plumber is indicative of possible asbestos exposure to this lady through washing her husband's clothes.

The weight loss, lethargy and chest pain coupled with the probable asbestos exposure and the chest x ray findings would make mesothelioma the most likely diagnosis.

She may be eligible for compensation if it is shown to be a mesothelioma.

Further investigation including CT and biopsy are required.

[C]

13/A 75-year-old lady is referred to the rapid access chest clinic with a four month history of progressive breathlessness, lethargy, anorexia and one stone weight loss.

She is a housewife and smokes 15/cigarettes a day. Her husband, a retired plumber, recently died from a 'chest problem'. She reports a dull right-sided chest pain that has been present for the last one month, and is partially relieved with 'low dose' co-codamol prescribed by her GP.

On examination she is dyspnoeic and cachectic. Examination of her chest demonstrates reduced vocal fremitus, percussion note and breath sounds throughout the right lung.

Chest x ray reports a medium sized right-sided pleural effusion. The pleura of the right hemithorax are thickened.

What investigation is the most likely to yield the diagnosis?

(Please select 1 option)

	Chest drain and pleural fluid sent for cytology
	CT thorax

	CT guided lung biopsy
	Pleural aspiration and fluid for cytology
	Video-assisted thoracoscopic surgery (VATS) biopsy

This lady has constitutional symptoms as well as a unilateral pleural effusion, and given her possible second-hand exposure to asbestos, mesothelioma needs to be excluded.

The inclusion of her husband's occupation as a plumber is indicative of possible asbestos exposure in this lady, potentially through washing her husband's clothes.

The weight loss, lethargy and chest pain coupled with the probable asbestos exposure and the chest x ray findings would make mesothelioma the most likely diagnosis.

She may be eligible for compensation if it is shown to be a mesothelioma.

To aid the choice of treatment, as well as future compensation claims, a histological diagnosis is key. VATS biopsy is the diagnostic tool of choice as it provides the highest diagnostic yield, as well as allowing the operator to gain a macroscopic view of the abnormal pleura. Prophylactic radiotherapy is often given to minimise the risk of track seeding.

Further investigations including CT and histopathology are required.

[E]

14/A 15-year-old boy attends outpatients accompanied by his parents, following an episode of small volume haemoptysis.

He reports feeling a little breathless. His mother reports that throughout childhood he had repeated chest infections and has always been the smallest of her children. The patient's siblings are all well. They have a pet cat at home.

On examination he looks well and is not dyspnoeic. He has nasal polyps and on auscultation coarse crackles are heard throughout both lung fields.

Investigations are as follows:

Hb	11.3 g/dL	(13.0 - 18.0)
WCC	12.9 $\times 10^9$/L	(4 - 11)
Platelets	438 $\times 10^9$/L	(150 - 400)
Na+	138 mmol/L	(137 - 144)
K+	4.1 mmol/L	(3.5 - 4.9)

Urea	5.3 mmol/L	(2.5 - 7.5)
Creatinine	69 µmol/L	(60 - 110)
Bilirubin	37 µmol/L	(1 - 22)
AST	49 U/L	(1 - 31)
ALP	228 U/L	(45 - 105)

Spirometry:

FEV_1	1.8 L/min (62% predicted)
FVC	3.1 L (83% predicted)
K_{co}	72% predicted

CXR demonstrates patchy haziness throughout both lung fields, with tramlines noted at the bases.

Weight 40 kg

ECG; sinus rhythm, nil of note.

What is the most likely diagnosis?

(Please select 1 option)

	Allergic bronchopulmonary aspergillosis
	Asthma
	Cystic fibrosis
	Goodpasture's syndrome
	Kartagener's syndrome

This patient presents relatively late with symptoms and signs of bronchiectasis, relatively poor nutritional status, hepatic derangement. This is in keeping with a diagnosis of cystic fibrosis.

This patient has cystic fibrosis, as evidenced by the repeated chest infections (secondary to bronchiectasis, as seen clinically and on the chest x ray), his small stature, and nasal polyps and deranged LFTS (secondary to biliary ductule obstruction). The diagnosis of cystic fibrosis can be made by performing a sweat test. A sweat concentration of >60 mmol/L is diagnostic.

The cilia in Kartagener's syndrome are abnormal. This condition is associated with bronchiectasis, situs inversus (there was no note made of a right sided apex beat and the ECG did not show signs of dextrocardia), and infertility.

Goodpasture's syndrome may be diagnosed through immunological testing (positive anti-GBM antibodies), but usually occurs in adults over 16 years.

His presentation is not in keeping with asthma.

[C]

15/A 15-year-old boy attends outpatients accompanied by his parents, following an episode of small volume haemoptysis.

He reports feeling a little breathless. His mother reports that throughout childhood he had repeated chest infections, and has always been the smallest of her children. The patient's siblings are all well. They have a pet cat at home.

On examination he looks well and is not dyspnoeic. He has nasal polyps and on auscultation coarse crackles are heard throughout both lung fields.

Investigations are as follows:

Haemoglobin	11.3 g/dL	(13.0 - 18.0)
WCC	12.9 $\times 10^9$/L	(4 - 11)
Platelets	438 $\times 10^9$/L	(150 - 400)
CRP	41 mg/L	(<10)
Serum Sodium	138 mmol/L	(137 - 144)
Serum Potassium	4.1 mmol/L	(3.5 - 4.9)
Urea	5.3 mmol/L	(2.5 - 7.5)
Creatinine	69 µmol/L	(60 - 110)
Bilirubin	37 µmol/L	(1 - 22)
AST	49 U/L	(1 - 31)
ALP	228 U/L	(45 - 105)

Spirometry:

FEV_1	1.8L/min (62% predicted)
FVC	3.1 L (83% predicted)
K_{co}	(72% predcited)

CXR demonstrates patchy haziness throughout both lung fields, with tramlines noted at the bases.

ECG; sinus rhythm, nil of note.

Given the likely diagnosis, which of the below is the most suitable test to confirm diagnosis?

(Please select 1 option)

○	Anti-GBM antibodies
○	*Aspergillus* precipitins
○	Electron microscopy of the cilia
○	Methacholine challenge test
○	Sweat test

This patient has cystic fibrosis, as evidenced by:

The repeated chest infections (secondary to bronchiectasis, as seen clinically and on the chest x ray)
His small stature
Nasal polyps
Deranged LFTs (secondary to biliary ductule obstruction)

The diagnosis of cystic fibrosis can be made by performing a sweat test. A sweat concentration of greater than 60 mmol/L is diagnostic.

The cilia in Kartagener's syndrome are abnormal. This condition is associated with bronchiectasis, situs inversus (there was no note made of a right sided apex beat and the ECG did not show signs of dextrocardia), and infertility.

Goodpasture's syndrome may be diagnosed through immunological testing (positive anti-GBM antibodies), but usually occurs in adults over 16 years.

[E]

16/A 53-year-old lady is referred to the chest clinic with a history of recurrent chest infection and episodic wheezing.

The only other medical history she provides is that she thinks she is in menopause as she is having frequent flushing attacks. She has a 10 pack year history.

A chest x ray was requested by the GP and the report is as follows:

"There is right upper lobe collapse. The remaining lung fields are clear, with no masses identified. The heart is unremarkable."

What is the most likely diagnosis?

(Please select 1 option)

○	Bronchial carcinoid
○	Churg-Strauss syndrome
○	COPD
○	Lymphoma
○	Phaeochromocytoma

This lady may have an obstructive lesion as evidenced by the right upper lobe collapse and recurrent chest infections.

She is likely to have bronchial carcinoid, a form of neuroendocrine tumour. They usually arise from the large bronchi and secrete serotonin. They may present with obstructive symptoms caused by the growing mass in the airway - wheezing, recurrent chest infections, haemoptysis - although they are often asymptomatic.

Systemic symptoms are usually found when there are metastases in the liver - carcinoid syndrome, but can very occasionally occur in localised bronchial carcinoid as the bronchial tree drains into the systemic circulation.

COPD is less likely given the history of flushing and lobar collapse.

Phaeochromocytoma may account for the flushing but not the other symptoms.

Lymphoma can result in obstructive lesions but one would expect enlarged mediastinal lymph nodes, as well as palpable lymphadenopathy.

[A]

17/A 67-year-old gentleman presents to the Emergency department with a four day history of increasing breathlessness, productive cough and fever.

On examination he is disoreiented. His observations are as follows: HR 132 (irregular), BP 89/61 mmHg, RR 29, and temperature 38.7℃.

His laboratory investigations are listed below:

Hb	11.1 g/dL	(13.0 - 18.0)
WBC	22.5 $\times 10^9$/L	(4 - 11)
Platelets	567 $\times 10^9$/L	(150 - 400)
Na+	136 mmol/L	(137 - 144)
K+	4.5 mmol/L	(3.5 - 5.0)
Urea	8.9 mmol/L	(2.5 - 7.5)

Creatinine	114 µmol/	(60 - 110l)
CRP	345 mg/L	(<10)

Chest x ray - right lower lobe consolidation.

Using current guidelines, what is his CURB-65 score and hence on which antibiotic regime should he be started empirically?

(Please select 1 option)

	CURB-65 score 3 and amoxicillin 1 g TDS and clarithromycin 500 mg BD
	CURB-65 score 4 and benzylpenicillin 1.2 g QDS and levofloxacin 500 mg BD
	CURB-65 score 4 and co-amoxiclav 1.2 g TDS and clarithromycin 500 mg BD
	CURB-65 score 5 and co-amoxiclav 1.2 g TDS and clarithromycin 500 mg BD
	CURB-65 score 3 and benzylpenicillin 1.2 g QDS and levofloxacin 500 mg BD

BTS has issued Guidelines for the Management of Community Acquired Pneumonia in Adults (2009). These discuss the most appropriate antibiotic regime to treat community-acquired pneumonia (CAP), according to the severity, which is often based on the CURB-65 score.

This patient has a CURB-65 score of 4 and as such co-amoxiclav and clarithromycin should be used (these antibiotics should be reserved for severe community acquired pneumonia [CURB-65 3-5]).

BTS guidelines recommend the use of amoxicillin and clarithromycin as the first line antibiotic regime in the treatment of moderate severity CAP. If the oral route is not possible, benzylpenicillin and clarithromycin should be used.

Amoxicillin alone is recommended for low severity CAP (CURB-65< 2) whether treated at home or in hospital.

Doxycycline may be used as an alternative antibiotic regime, but is not the preferred treatment according to these guidelines.

[C]

18/A 65-year-old gentleman with a BMI of 37 is referred to the respiratory clinic with increasing shortness of breath. He has a 40 pack year history.

Initial investigations reveal:

pH	7.36	(7.36 - 7.44)
pO_2	10.6 kP_a	(11.3 - 12.6)
pCO_2	6.7 kP_a	(4.7 - 6.0)

HCO_3	37 mmol/L	(20 - 28)

Spirometry shows:

	Actual	% predicted
FVC (l)	3.63	59
FEV1(l)	3.01	60
FRC (l)	1.46	37
RV (l)	1.08	48
TLC (l)	4.99	61

What is the likely diagnosis?

(Please select 1 option)

○	Chronic bronchitis
○	Emphysema
○	Interstitial lung disease
○	Neuromuscular disorder
○	Obesity hypoventilation syndrome

This patient has evidence of a restrictive lung disease based on his spirometry readings and type II respiratory failure (compensated) on his blood gases.

Coupled with his raised BMI, this would be in keeping with obesity hypoventilation syndrome.

Neuromuscular disease and interstitial lung disease may also cause restrictive lung defects, but the inclusion of the patient's BMI is key here.

[E]

19/A 68-year-old woman with a significant smoking history is referred to the rapid access chest clinic with a six month history of weight loss, a cough and two episodes of haemoptysis. She was previously fit and well.

On questioning she has a troublesome cough, some mild right sided chest pain which she describes as an ache and finds she is getting increasingly fatigued and is having to spend a few hours of the day in either bed or on the sofa. However she is still able to complete most of her activities of daily living with some assistance from her husband.

According to the World Health Organisation (WHO) classification, what is her performance status?

(Please select 1 option)

	0
	1
	2
	3
	4

Assessing a patient's performance status is important when evaluating the most appropriate treatment options. It is commonly used by cancer MDTs, but has a role in assessing patients with chronic illnesses including COPD.

WHO Scale	Description
0	Asymptomatic
1	Symptomatic but ambulatory (can carry out light work)
2	In bed <50% of the day. Unable to work but can live at home with some assistance
3	In bed >50% of the day but unable to care for self
4	Bedridden

As taken from:

NICE. Lung cancer (CG121).

[C]

20/A 74-year-old man with known metastatic carcinoma of the pancreas presents with an acute episode of dyspnoea and pleuritic-sounding chest pain.

On examination his heart rate is 118 bpm and his oxygen saturations on pulse oximetry are 84% on 2 L of oxygen. Auscultation of his chest reveals clear lung fields.

What is his Wells score?

(Please select 1 option)

	3
	4.5

	5
	5.5
	6

Although all the above answers are features associated with pulmonary embolism, only haemoptysis forms part of the Wells score. The Wells scoring system devised in 1995 by Wells et al. is a prediction tool based on clinical criteria.

The Wells score:

Criteria	Points
Clinically suspected DVT	3
PE is #1 diagnosis, or equally likely	3
Tachycardia	1.5
Immobilisation or surgery in the previous four weeks	1.5
History of DVT or PE	1.5
Haemotypsis	1
Malignancy (treatment for within six months, palliative)	1

Traditional interpretation:

High score: >6.0
Moderate score: 2.0 to 6.0
Low score: <2.0

Alternate interpretation score:

>4 - PE likely. Consider diagnostic imaging
≤4 - PE unlikely. Consider D-dimer to rule out PE.

[D]

21/A 67-year-old smoker who lives in a men's hostel attends the Emergency department with back pain on a background of a cough which has been present for approximately nine months and intermittently has haemoptysis.

He is cachetic and looks unkempt. He smokes 40 cigarettes a day, and drinks a bottle of cider daily. He also reports urinary frequency and hesitancy, but no haematuria.

He undergoes a number of investigations and the results are listed below:

Hb	10.4 g/dl	13-18
WBC	11.9 × 10^9/L	4-11
Plt	342 × 10^9/L	150-400)
PSA	3µg/L	<4

CXR: cavitating lesions in both upper lobes.

Lumbar spine x ray: suspicious lesions in L3 and L5.

Sputum microscopy: multiple red rods on a blue background.

Bone biopsy: caseating granuloma.

What is the most likely diagnosis?

(Please select 1 option)

◌	Lung cancer
◌	Multiple myeloma
◌	Prostate cancer
◌	*Staphylococcus aureus* infection
◌	Tuberculosis

Cavitating apical lesions are characteristic of tuberculosis but differentials such as lung cancer should be considered for this appearance.

The sputum described is the classical AAFB appearance using the Ziehl-Neelsen stain.

Biopsy of the affected site may show the characteristic caseating granuloma.

His PSA is normal which excludes metastatic prostate cancer.

Staphylococcal infection may cause cavitating lung lesions, but the remaining investigations are in keeping with a diagnosis of tuberculosis.

[E]

22/A 67-year-old gentleman attends hospital with a one week history of severe flu-like symptoms and is now complaining of a cough productive of green sputum with shortness of breath.

A chest x ray demonstrates right upper lobe consolidation and pneumatocele formation.

What is the likely causative organism?

(Please select 1 option)

	Chlamydia pneumoniae
	Haemophilus influenzae
	Klebsiella pneumoniae
	Mycoplasma pneumoniae
	Staphylococcus aureus

Staphylococcal pneumonia is often a sequel to influenza infection and so anti-staphylococcal antibiotics need to be administered in patients who develop pneumonia following influenza.

It may produce a severe illness with a high mortality.

The productions of toxins may cause tissue necrosis resulting in cavitation, pneumatocele and pneumothoraces

[E]

23/A single 39-year-old man presents to the Emergency department with a one week history of a non-productive cough and increasing breathlessness. He reports his breathing is much worse on exertion.

His past medical history includes migraines and childhood asthma (but he has not used any inhaler in almost 30 years). He works in the pharmaceutical industry and often travels to India and Africa with work.

Examination reveals mild pyrexia of 37.8°C.

Investigations are as follows:

Hb	13.4 g/dL	(13-18)
WBC	3.3 $\times 10^9$/L	(1.5 - 7)
Plt	177 $\times 10^9$/L	(150 - 400)
CRP	78 mg/L	(<10)

pH	7.35	(7.35 - 7.45)
pO_2	6.5 kPa	(11.3-12.6)
pCO_2	5.8 kPa	(4.7-6.0)
LDH	310 U/L	(10-250)

CXR - bilateral patchy infiltrates

Given the likely diagnosis, which of the following would be the most appropriate initial treatment regime?

(Please select 1 option)

○	Amphotericin B
○	Clarithromycin and co-amoxiclav
○	Clindamycin and primaquine
○	Rifampicin, ethambutol, isoniazid, pyrazinamide
○	Trimethoprim sulfamethoxazole (TMP-SMX)

The gentleman is profoundly hypoxic with a slightly reduced WBC count and raised LDH - which is commonly seen in*Pneumocystis* pneumonia with underlying HIV infection. The chest x ray findings are also in keeping with this diagnosis.

Pneumocystis is a yeast-like fungal organism that can cause opportunistic infection in those who are immunocompromised but does not respond to typical anti-fungal treatment.

Initial treatment is with trimethoprim sulfamethoxazole (TMP-SMX / co-trimoxazole) or with intravenous pentamidine.

Adjunctive steroid therapy is recommended in patients with underlying HIV infection.

Clindamycin and primaquine are used in trimethoprim sulfamethoxazole resistant cases.

[E]

24/A patient who was seen in rapid access chest clinic undergoes a CT thorax. The report is as follows:

"There is a mass arising from the left main bronchus, 1.5 cm from the carina and not directly involving the carina. It is causing almost complete obstruction of the left main bronchus, and is likely to represent a primary lung tumour. There are several left hilar lymph nodes, the largest measuring 2 cm. The TNM staging is T3N1MX."

Using the above information what is the staging?

(Please select 1 option)

◯	IB
◯	IIB
◯	IIIA
◯	IIIB
◯	IV

Stage grouping by TMN subset is useful in the management of patients with lung cancer and is often utilised by physicians to assess treatment options.

Table 2: Stage grouping by TNM subsets

Nodes \ Tumour	T1	T2	T3	T4
N0	IA	IB	IIB	IIIB
N1	IIA	IIB	IIIA	IIIB
N2	IIIA	IIIA	IIIA	IIIB
N3	IIIB	IIIB	IIIB	IIIB

Key	
	Patient should be offered surgery if no medical contraindications and adequate lung function
	Surgery may be suitable for some patients, based on clinical judgement
	Not suitable for surgery

Stage IV = M1

Table taken from NICE guidance on Lung cancer (CG121).

[C]

25/A single 39-year-old man presents to the Emergency department with a one week history of a non-productive cough and increasing breathlessness. He reports his breathing is much worse on exertion.

His past medical history includes migraines and childhood asthma (but he has not used any inhaler in almost 30 years). He works in the pharmaceutical industry and often travels to India and Africa with work.

Examination reveals mild pyrexia of 37.8°C.

Investigations are as follows:

Hb	13.4 g/dl	13-18
WBC	3.3 x 10^9/L	1.5 - 7
Plt	177 x 10^9/L	150 - 400

CRP	78 mg/L	< 10
pH	7.35	7.35 - 7.45
pO_2	6.5 kPa	11.3-12.6
pCO_2	5.8 kPa	4.7-6.0
LDH	310 U/L	10-250

CXR - bilateral patchy infiltrates

What is the likely causative organism for his acute illness?

(Please select 1 option)

○	*Chlamydia pneumoniae*
○	*Legionella pneumophilia*
○	*Mycoplasma* tuberculosis
○	*Pneumocystis carinii*
○	*Pneumocystis jiroveci*

The gentleman is profoundly hypoxic with a slightly reduced WBC count and raised LDH - which is commonly seen in*Pneumocystis* pneumonia with underlying HIV infection.

Pneumocystis is yeast-like fungal organism that can cause opportunistic infection in those who are immunocompromised. The organism was initially described as *Pneumocystis carinii*, but this is the variant that causes infection in animals and not humans. The human variant of the organism is*Pneumocystis jiroveci*. Classically patients with*Pneumocystis* pneumonia (PCP) have a dry cough, profound hypoxia and bilateral infiltrates on chest x ray.

Given the travel to TB endemic areas this should be considered however the history and chest x ray findings are more in keeping with PCP.

The other two are atypical pathogens.

[E]

26/A 45-year-old salesman presents with a one week history of fever, cough, headache and dyspnoea. He has no past medical history of note. He has no recent overseas travel history but does travel widely within the United Kingdom with his work.

He has a temperature of 40.1°C, heart rate is 121 bpm, and saturations are 92% air.

Investigations are as follows:

Na	128 mmol/L	137 - 144
Urea	6.8 mmol/L	2.5 - 7.5
Creatinine	109 µmol/L	60 - 110
WBC	11.7 g/dl	13-18
CRP	73	< 10 mg/L
Urine	2+ protein	2+ blood
Sputum	mainly leukocytes	no organims seen

What is the most likely diagnosis?

(Please select 1 option)

○	Legionnaires' disease
○	*Mycoplasma* pneumonia
○	*Pneumocystis* pneumonia
○	Pulmonary embolism
○	Streptococcal pneumonia

Many of the findings in this patient are in keeping with a diagnosis of legionnaires' disease (*Legionella*infection plus pneumonia).

Hyponatraemia secondary to syndrome of inappropriate antidiuretic hormone secretion (SIADH) is more common in*Legionella* infection than other pathogens. Deranged liver function tests and microscopic haematuria may also be seen. Up to 40% of patients may also have proteinuria. Sputum sampling is often unhelpful on its own.

The *Legionella* bacterium was first identified in 1976 during the annual convention of the American Legion which was held at a hotel in Philadelphia. Infection was presumed to be spread by contamination of the water in the hotel's air conditioning system.

The mortality rate may approach 100% in patients with underlying disease. In untreated patients, the mortality rate may be as high as 80%.

Further information

[A]

27/A patient who was seen in rapid access chest clinic undergoes a CT thorax. The report is as follows;

"There is a mass arising from the left main bronchus, 1.5 cm from the carina and not directly involving the carina. It is causing almost complete obstruction of the left main

bronchus, and is likely to represent a primary lung tumour. There are several left hilar lymph nodes, the largest measuring 2 cm".

What is the TNM (tumour, nodes, metastases) staging of this lung tumour?

(Please select 1 option)

○	T2N1MX
○	T2N2MX
○	T2N2M1
○	T3N1MX
○	T3N2M1

The TNM Classification of Malignant Tumours (TNM) is a cancer staging system that describes the extent of cancer in a patient's body.

T describes the size of the tumor and whether it has invaded nearby tissue,
M describes distant metastasis (spread of cancer from one body part to another),
N describes regional lymph nodes that are involved.

Primary Tumor (T)	
TX	Primary tumor cannot be assessed, or tumor proven by the presence of malignant cells in sputum or bronchial washings but not visualized by imaging or bronchoscopy
T0	No evidence of primary tumor
Tis	Carcinoma in situ
T1	Tumor 3 cm or less in greatest dimension, surrounded by lung or visceral pleura, without bronchoscopic evidence of invasion more proximal than the lobar bronchus (for example, not in the main bronchus)
T1a	Tumor 2 cm or less in greatest dimension
T1b	Tumor more than 2 cm but 3 cm or less in greatest dimension
T2	Tumor more than 3 cm but 7 cm or less or tumor with any of the following features (T2 tumors with these features are classified T2a if 5 cm or less): • involves main bronchus, 2 cm or more distal to the carina; • invades visceral pleura (PL1 or PL2); • associated with atelectasis or obstructive pneumonitis that extends to the hilar region but does not involve the entire lung

T2a	Tumor more than 3 cm but 5 cm or less in greatest dimension
T2b	Tumor more than 5 cm but 7 cm or less in greatest dimension
T3	Tumor more than 7 cm or one that directly invades any of the following: parietal pleural (PL3), chest wall (including superior sulcus tumors), diaphragm, phrenic nerve, mediastinal pleura, parietal pericardium; or tumor in the main bronchus less than 2 cm distal to the carina1 but without involvement of the carina; or associated atelectasis or obstructive pneumonitis of the entire lung or separate tumor nodule(s) in the same lobe
T4	Tumor of any size that invades any of the following: mediastinum, heart, great vessels, trachea, recurrent laryngeal nerve, esophagus, vertebral body, carina, separate tumor nodule(s) in a different ipsilateral lobe
Distant Metastasis (M)	
M0	No distant metastasis
M1	Distant metastasis
M1a	Separate tumor nodule(s) in a contralateral lobe, tumor with pleural nodules or malignant pleural (or pericardial) effusion
M1b	Distant metastasis (in extrathoracic organs)
Regional Lymph Nodes (N)	
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastases
N1	Metastasis in ipsilateral peribronchial and/or ipsilateral hilar lymph nodes and intrapulmonary nodes, including involvement by direct extension
N2	Metastasis in ipsilateral mediastinal and/or subcarinal lymph node(s)
N3	Metastasis in contralateral mediastinal, contralateral hilar, ipsilateral or contralateral scalene, or supraclavicular lymph node(s)

[D]

28/A 35-year-old male attends the HIV clinic complaining of a productive cough. Sputum analysis is as follows:

"Gram positive (weakly) bacilli, stains red with Ziehl Neelsen stain."

Given these findings what is the mostly likely organism responsible for his symptoms?

(Please select 1 option)

	Aspergillus fumigatus
	B. anthracis
	B. cereus
	M. tuberculosis
	Pneumocystis jirovecii

The sputum analysis is in keeping with *Mycobacterium tuberculosis*, which is a small aerobic non-motile bacillus. It is classified as a Gram positive organism but stains very weakly on testing. When using the Ziehl-Neelsen test it stains bright red against a blue background.

B. anthracis and *B. cereus* are two other bacilli that are pathogenic; *B. anthracis* is responsible for anthrax.

Aspergillus and *Pneumocystis* are both pathogens that are associated with respiratory illnesses in immunocompromised patients, but the sputum findings are not in keeping with these organisms.

[D]

29/A 24-year-old man presents to the Emergency department with a two hour history of pleuritic chest pain. He denies feeling breathless, with oxygen saturations of 96% on air.

A chest x ray is performed and an observant CT1 recognises a small apical pneumothorax, which when measured is 1.8 cm.

Accordingly to current guidelines, what intervention should be undertaken?

(Please select 1 option)

	Aspirate and admit for 24 hours
	Aspirate and discharge home after 12 hours if well
	Discharge with advice to return if symptoms worsen and follow up chest x ray in 48 hours
	Discharge with advice to return if symptoms worsen and follow up chest x ray in two weeks
	Intercostal chest drain insertion (Seldinger technique)

This question requires an understanding of the different types of pneumothorax and the current British Thoracic Society (BTS) guidelines for management.

This patient has a small pneumothorax with no known underlying lung disease and as such can be classed as a spontaneous pneumothorax. According to current BTS guidelines, if the patient is not breathless and the rim of air is less than 2 cm they can be considered for early discharge and repeat chest x ray in two weeks.

A secondary pneumothorax in contrast always requires intervention.

Aspiration is less likely to be effective in a secondary pneumothorax and so if it fails or does not meet the above criteria, a chest drain needs to be inserted.

The Seldinger technique using a 16G is the preferred method for this.

[D]

30/A 65-year-old man with a 50 pack/year history presents with cough and haemoptysis.

A chest x ray shows a right hilar cavitating mass. Bronchoscopy and biopsy confirm the diagnosis of carcinoma of bronchus. His calcium levels are 3.89, and the parathyroid hormone related peptide (PTHrH) is raised.

What is the most likely histological diagnosis?

(Please select 1 option)

○	Adenocarcinoma
○	Alveolar cell carcinoma
○	Fibrosarcoma
○	Small cell carcinoma
○	Squamous cell carcinoma

Sixty per cent to 80% of squamous cell carcinomas arise in the proximal portions of the tracheobronchial tree. Squamous cell carcinomas are associated with extensive necrosis with resulting cavitation.

Hypercalcaemia and elevated parathyroid hormone related peptide levels are most commonly associated with squamous cell carcinomas.

Small cell carcinomas may be also be associated with hypercalcaemia but not with increased PTHrH levels.

[E]

31/A 25-year-old stable hand presents with 12 hour history of cough, shortness of breath, fever and headache.

On examination his heart rate is 114 bpm, with a respiratory rate of 26 and temperature of 37.8°C. His oxygen saturations are 92%, dropping to 88% when he walks across the ward.

His chest x ray demonstrates a diffuse interstitial micronodular pattern.

Based on the information what is the most likely diagnosis?

(Please select 1 option)

	Chlamydia trachomatis
	Hamman-Rich syndrome
	Hypersentivity pneumonitis
	Pneumocystis pneumonia
	Respiratory syncytial virus

This patient has evidence of inflammation of the alveoli and lung interstitium. All the above conditions may be classed as interstitial lung disease, in keeping with the x ray findings.

The inclusion of this patient's occupation as a stable hand (and hence potential mold allergen exposure) is key to answering this question

This patient's signs and symptoms are in keeping with acute hypersensitivity pneumonitis - most likely as a result of exposure to mouldy hay in his line of work (farmer's lung).

Hypersensitivity pneumonitis may be acute, subacute or chronic. Acute hypersensitivity pneumonitis symptoms may develop four to six hours after exposure to allergen and last 12 hours up to several days following removal of the antigen.

In a young person with these findings *Pneumocystis*pneumonia (with underlying undiagnosed HIV) should be considered.

[C]

32/A 35-year-old woman with a two year history of rhinitis, develops asthma-like symptoms and a purpuric rash.

She is referred to the hospital for further investigations. The results are as follows:

Hb	11.5	11.5-16.5
MCV	93 fl	80 - 96
WBC	10.2 x 10^9/L	4 - 11
Neutrophils	4. 7 x 10^9/L	1.5 - 7

Eosinophils	1.8 x 10^9/L	0.04 - 0.4
Urine	Nil	
ESR	45 mm/1st hr	0 - 20
Na+	144 mmol/L	137 - 144
Urea	5.8 mmol/L	2.5 - 7.5
ANCA	detected perinuclear pattern	
MPO	detected	
CXR	patchy shadowing in both lung fields	

What is the most likely diagnosis?

(Please select 1 option)

○	Churg-Strauss syndrome
○	Microscopic polyangiitis
○	Polyarteritis nodosa
○	Systemic lupus erythematosus
○	Wegener's granulomatosis

The positive antineutrophil cytoplasmic antibody (ANCA) indicates this patient likely has a vasculitis (non-vasculitic causes of positive ANCA have negative anti-myeloperoxidase antibodies). The skin lesions indicate cutaneous involvement.

The absence of renal and nasal involvement suggest a diagnosis of either Churg-Strauss or polyarteritis nodosa.

The presence of asthma symptoms is more in keeping with Churg-Strauss.

[A]

33/A 23-year-old man presents to the Emergency department with a severe exacerbation of asthma. He is treated with nebulised salbutamol and ipratropium bromide and 15L oxygen.

His results are as follows:

	Pre-treatment	Post-Treatment
PO2 (kP_a)	6.6	7.9
PCO2 (kP_a)	3.4	3.5
pH	7.35	7.32

PEFR (L/min)	100	160

According to BTS guidelines, which is the next step in his management?

(Please select 1 option)

○	Intravenous aminophylline
○	Intravenous hydrocortisone
○	Intravenous magnesium
○	Intravenous salbutamol
○	Intravenous suxamethonium and ventilation

BTS guidelines recommend that all patients with acute exacerbations of asthma receive steroids in appropriate doses.

Although the effects may not be seen for several hours it is important to initiate this management step.

Obviously the other treatments may all be required, however this question focussed on the guidelines.

[B]

34/A 67-year-old gentleman presents to the Emergency department with a four day history of increasing breathlessness, productive cough and fever.

On examination his observations are as follows: HR 132 (irregular), BP 89/61mmHg, RR 29, and temperature 38.7°C. He appears a little disorientated.

His laboratory investigations are listed below:

Hb	11.1 g/dL	(13.0 - 18.0)
WBC	22.5 $\times 10^9$/L	(4 - 11)
Platelets	567 $\times 10^9$/L	(150 - 400)
Na+	136 mmol/L	(137 - 144)
K+	4.5 mmol/L	(3.5 - 4.9)
Urea	8.9 mmol/L	(2.5 - 7.5)
Creatinine	114 µmol/L	(60 - 110)
CRP	345 mg/L	(<10)

Chest x ray right lower lobe consolidation

Using current guidelines, which empirical antibiotic regime should be commenced?

(Please select 1 option)

◯	Amoxicillin 500 mg TDS
◯	Amoxicillin 1 g TDS and clarithromycin 500 mg BD
◯	Benzylpenicillin 1.2 g QDS and Levofloxacin 500 mg BD
◯	Co-amoxiclav 1.2g TDS and clarithromycin 500 mg BD
◯	Doxycycline 200 mg loading dose and then 100 mg OD

The British Thoracic Society has issued Guidelines for the Management of Community Acquired Pneumonia in Adults. These discuss the most appropriate antibiotic regime to treat community-acquired pneumonia (CAP) according to the severity, which is often based on the CURB-65 score.

This patient has a CURB-65 score of 4 and as such co-amoxiclav and clarithromycin should be used (these antibiotics should be reserved for severe community acquired pneumonia [CURB-65 3-5]).

BTS guidelines recommend the use of amoxicillin and clarithromycin as the first line antibiotic regime in the treatment of moderate severity CAP.

If the oral route is not possible, benzylpenicillin and clarithromycin should be used.

Amoxicillin alone is recommended for low severity CAP (CURB-65< 2) whether treated at home or in hospital.

Doxycycline may be used as an alternative antibiotic regime, but is not the preferred treatment according to these guidelines.

[D]

35/A 55-year-old former shipyard worker complained of increasing breathlessness.

He undergoes pulmonary function tests and the results are as follows:

Test	Predicted	Actual
FEV (L)	4.4	3.5
FVC (L)	4.9	4.0
TL_{CO} (mmol/min/kP_a)	10.3	6.8
K_{CO} (mmol/min/kP_a/L)	1.9	2.3

What is the most likely diagnosis?

(Please select 1 option)

◯	Asbestosis

	Chronic bronchitis
	Emphysema
	Mesothelioma
	Pulmonary fibrosis

FVC is reduced much more than FEV1, which would be in keeping with a restrictive defect. This may arise from either pulmonary fibrosis or extrapulmonary cause such as pleural disease.

The raised K_{co} would be in keeping with extrapulmonary disease with unimpaired gas transfer (pulmonary fibrosis has both reduced TL_{co} and K_{co}).

This gentleman's occupational history is key, making him at risk from both mesothelioma and asbestosis. In the question the absence of other features of mesothelioma (pain, weight loss, etc.) makes asbestosis the most likely diagnosis. Asbestosis specifically refers to the pneumoconiosis caused by inhalation of asbestos fibers. The disease is characterized by slowly progressive, diffuse pulmonary fibrosis.

The spectrum of pulmonary disorders associated with asbestos exposure includes:[1]

Asbestosis
Pleural disease (focal and diffuse benign pleural plaques)
Malignancies (non-small cell and small cell carcinoma of the lung as well as malignant mesothelioma)

Other causes of raised K_{co} include

Pulmonary haemorrhage
Polycythaemia
Left to right shunts, and
Neuromuscular weakness.

[A]

36/A 69-year-old man is referred into hospital by his GP with a 5/7 history of shortness of breath and productive cough. He is talking appropriately in full sentences.

His observations are as follows; HR 98, BP 110/79 mmHg, RR 27 and temperature 38.2°C.

His laboratory investigations are as follows;

Hb	12.5 g/dL	(13.0 - 18.0)
WBC	18.7 $\times 10^9$/L	(4 - 11)

Neutrophils	16.1 $\times 10^9$/L	(1.5 - 7.0)
Platelets	479 $\times 10^9$/L	(150 - 400)
Sodium	123 mmol/L	(137 - 144)
Potassium	3.8 mmol/L	(3.5 - 4.9)
Urea	8.1 mmol/L	(2.5 - 7.5)
Creatinine	115 µmol/L	(60 - 110)
CRP	210 mg/L	(<10)

Using the information available and the severity index CURB-65, what severity of pneumonia does this patient demonstrate?

(Please select 1 option)

○	High severity
○	Life threatening
○	Low severity
○	Moderate severity
○	Very low severity

The CURB-65 score is based on five parameters:

C - Confusion (new onset)
U - Urea >7 mmol/l
R - Respiratory rate >30
B - BP <90 mmHg systolic or diastolic< 60 mmHg
65 - Age equal to or greater than 65 years.

This patient scores 2 on the CURB-65 scoring index (for his age and elevated urea). As such he would be classed as moderate severity.

High severity is for CURB-65 scores 3-5.

For this scoring system there is no life threatening or very low severity indexes, although clinical acumen should always be used in conjunction with any severity indexes to ensure effective management of the patient

[D]

37/A 75-year-old man is referred into hospital by his GP with a provisional diagnosis of pneumonia. He is talking appropriately in full sentences.

His observations are as follows; HR 98, BP 110/79 mmHg, RR 27 and temperature 38.2°C.

His laboratory investigations are as follows:

Hb	12.5 g/dL	(13.0 - 18.0)
WBC	18.7 $\times 10^9$/L	(4 - 11)
Neutrophils	16.1 $\times 10^9$/L	(1.5 - 7.0)
Platelets	479 $\times 10^9$/L	(150 - 400)
Sodium	123 mmol/L	(137 - 144)
Potassium	3.8 mmol/L	(3.5 - 4.9)
Urea	8.1 mmol/L	(2.5 - 7.5)
Creatinine	115 µmol/L	(60 - 110)
CRP	210 mg/L	(<10)

Based on the above information what is his predicted mortality according to current BTS guidelines?

(Please select 1 option)

	<3%
	5-7%
	9%
	20%
	40%

This question requires an understanding of the pneumonia severity index (CURB-65) and its associated mortality rates.

This gentleman has a CURB-65 score 2 based on his age and elevated urea. This places him in moderate severity of community-acquired pneumonia and according to current BTS guidelines this is associated with 9% mortality.

CURB-65 scoring is a quick method of assessing a patient's disease severity and using this to initiate appropriate management.

Low severity - CURB65 0-1 mortality <3%
Moderate severity - CURB-65 2, mortality 9%
High severity - CURB-65 3-5, mortality 15-40%

[C]

38/A 19-year-old man with recurrent admissions to hospital for exacerbations of his asthma attends the Emergency department with a short history of increasing breathlessness and cough.

On examination he is obviously dyspnoeic and wheezy and becoming exhausted. His respiratory rate is 16 breaths per minute, his HR is 125 bpm (sinus tachycardia) and his PEFR is 30% predicted.

An arterial blood gas is taken and the results are as follows:

pH	7.43	(7.36 - 7.44)
pO_2	7.3 kPa	(11.3 - 12.6)
pCO_2	5.2 kPa	(4.7 - 6.0)

Using the details above what severity is this patient's exacerbation?

(Please select 1 option)

○	Acute severe exacerbation
○	Life threatening exacerbation
○	Mild exacerbation
○	Moderate exacerbation
○	Near fatal exacerbation

A life threatening exacerbation of asthma can be diagnosed based on one of the following:

PEF <33% best or predicted
SpO_2 <92%
PaO_2 <8 kPa
Normal $PaCO_2$ (4.6-6.0 kPa)
Silent chest
Cyanosis
Poor respiratory effort
Arrhythmia
Exhaustion, altered conscious level.

[B]

39/A 24-year-old woman with recurrent admissions to hospital for exacerbations of her asthma attends the Emergency department at 8 am with a short history of increasing breathlessness.

On examination she is obviously dyspnoeic and wheezy. Her respiratory rate is 27 breaths per minute, her HR is 118 bpm (sinus tachycardia) and her PEFR is 34%.

An arterial blood gas is taken and the results are as follows;

pH	7.52	(7.36 - 7.44)
pO_2	8.64 kPa	(11.3 - 12.6)
pCO_2	3.1 kPa	(4.7 - 6.0)

Using the details above what severity is this patient's exacerbation?

(Please select 1 option)

○	Acute severe exacerbation
○	Life threatening exacerbation
○	Mild exacerbation
○	Moderate exacerbation
○	Near fatal exacerbation

An acute severe exacerbation of asthma can be diagnosed based on one of the following:

PEF 33-50% best or predicted
Respiratory rate ≥25/min
Heart rate ≥110/min
Inability to complete sentences in one breath

[A]

40/A 60-year-old lady undergoing treatment for acute leukaemia attends the haematology clinic complaining of cough, wheeze, occasional haemoptysis and fever.

A chest x ray demonstrates the classical air crescent sign. A galactomannan test is performed which is positive.

Based on the above information, what is the most likely diagnosis?

(Please select 1 option)

○	Aspergillosis
○	*Pneumocystis* pneumonia

	Staphylococcal pneumonia
	Streptococcal pneumonia
	Tuberculosis

Aspergillosis is a fungal infection, and develops mainly in individuals who are immunocompromised. It is a leading cause of death in acute leukaemia and haemopoietic stem cell transplantation.

Signs and symptoms include cough, haemoptysis, chest wall pain, fever and shock.

It is often seen on chest x rays and CT scan, and demonstrates an air crescent sign. Other investigations include microscopy and the galactomannan test.

Pneumocystis pneumonia and tuberculosis are also found in immunocompromised individuals however the information provided is more in keeping with a diagnosis of aspergillosis.

[A]

41/A 55-year-old woman undergoing treatment for acute leukaemia attends the haematology clinic complaining of cough, wheeze, occasional haemoptysis and fever.

A chest x ray demonstrates the classical air crescent sign. A galactomannan test is performed which is positive.

Given the most likely diagnosis, what treatment should be commenced?

(Please select 1 option)

	Amphotericin B
	Caspofungin
	Co-amoxiclav
	Co-trimoxazole
	Rifampicin, isoniazid, ethambutol and pyrazinamide

Aspergillosis is a fungal infection and develops mainly in individuals who are immunocompromised. It is a leading cause of death in acute leukaemia and haemopoietic stem cell transplantation.

Signs and symptoms include cough, haemoptysis, chest wall pain, fever and shock.

It is often seen on chest x rays and CT scan, and demonstrates an air crescent sign. Other investigations include microscopy and the galactomannan test.

The current treatments include voriconazole and liposomal amphotericin B. Caspofungin is also used as part of combination therapy.

Co-trimoxazole is used in the treatment of *Pneumocystis*pneumonia.

Rifampicin, isoniazid, ethambutol and pyrazinamide are used in the treatment of TB.

[A]

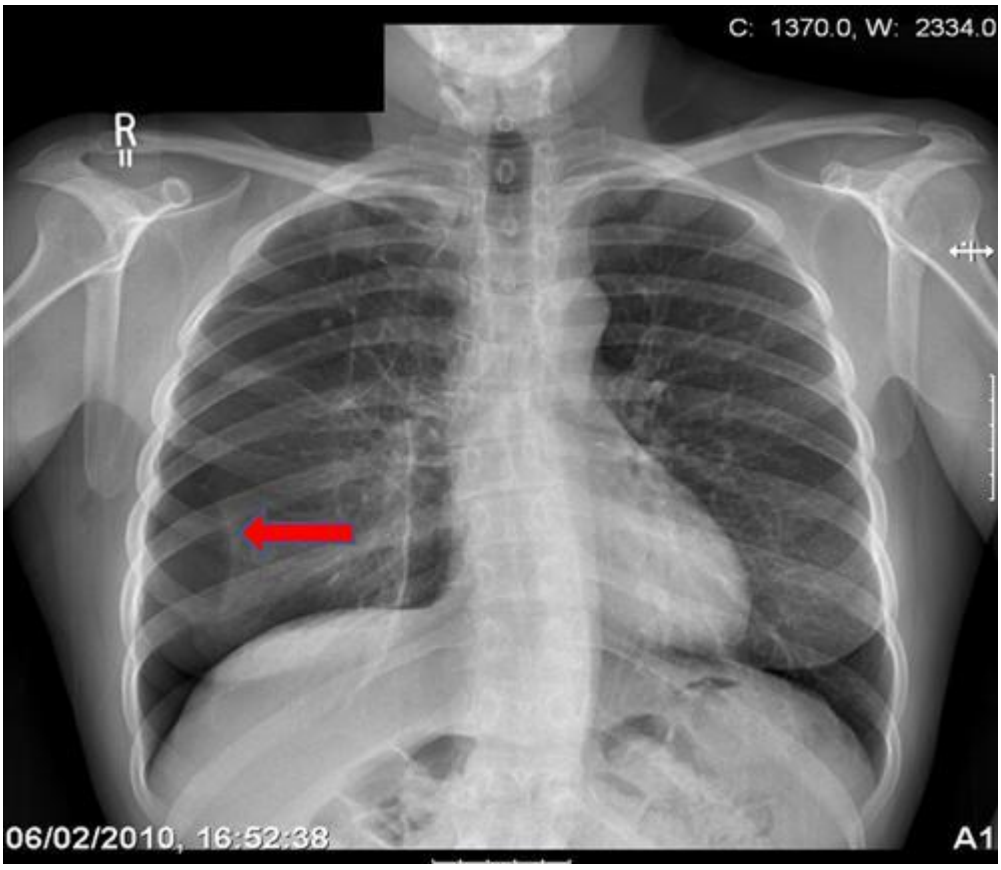
42/A 45-year-old lady presents with a sudden onset of shortness of breath and right-sided chest pain.

She has a history of recurrent pneumothoraces and has had a pleurodesis in 2005. There is no history of any recent flights or trauma and she is a non-smoker. Past medical history includes endometriosis for which she takes depot progesterone injections every three months. On examination she is of average height and stature.

Observations: oxygen saturations 98% on air, blood pressure 138/90 mmHg, heart rate 65/min, RR 30.

Examination of the chest reveals a hyper-resonant note and decreased air entry in the right base; the trachea is central.

Chest x ray reveals a 3.2 cm rim of air around the lung.



How would you like to manage this patient?

(Please select 1 option)

	Admit and insert chest drain with underwater seal
	Admit, aspirate, re-x ray and monitor
	Admit, aspirate under ultrasound guidance as localised pneumothorax
	Admit, monitor, high flow oxygen
	Discharge with advice, for outpatient follow up

This is a secondary pneumothorax.

Although this lady is haemodynamically stable, she has a respiratory rate of 30 and is complaining of pain, indicating she was symptomatic from her pneumothorax and intervention is required.

As per British Thoracic Society (BTS) guidance, a pneumothorax with a rim of greater than 2 cm requires either aspiration or chest drainage.

High flow oxygen should be given in all cases of pneumothorax, as it facilitates re-absorption of the pleural air, which is predominantly composed of nitrogen.

[A]

43/A 58-year-old man presents with weight loss and haemoptysis. He has smoked most of his life.

On examination he is clubbed and has clinical evidence of right pleural effusion. His serum calcium is 3.2 mM (2.2-2.6 mmol/l). A bone scan is normal.

Which of the following histological type of lung cancer is he most likely to suffer from?

(Please select 1 option)

	Adenocarcinoma
	Large cell carcinoma
	Mesothelioma
	Small cell carcinoma
	Squamous cell carcinoma

Hypercalcaemia in absence of bony metastases occurs in about 15% of squamous cell lung carcinoma from parathyroid hormone related protein (PTHrP) production. This is a feature of non-metastatic manifestation of malignancy.

Inappropriate antidiuretic hormone (ADH) secretion (hyponatraemia) and ectopic adrenocorticotropic hormone (ACTH) production (Cushing's syndrome) occur with small cell lung cancer.

Clubbing is predominantly associated with squamous cell cancers and occasionally adenocarcinoma.

[E]

44/A 60-year old man with a history of non-small cell lung cancer was treated with a right lower lobectomy 12 months ago.

He had an chest and abdominal CT scan one month ago which revealed hepatic mass lesions and hilar lymphadenopathy. He now presents with malaise and fatigue.

His results show:

Urinalysis	Protein +++
24 hour urine protein	2.7 g/24hr
Serum urea	30 mmol/L (2.5-7.5)
Serum creatinine	450 µmol/L (60-110)

A renal biopsy shows focal deposition of IgG and C3 with a granular pattern.

What is the most likely diagnosis?

(Please select 1 option)

○	Goodpasture's syndrome
○	Membranous glomerulonephritis
○	Minimal change glomerulonephritis
○	Nodular glomerulosclerosis
○	Rapidly progressive glomerulonephritis

Membranous GN is associated with:

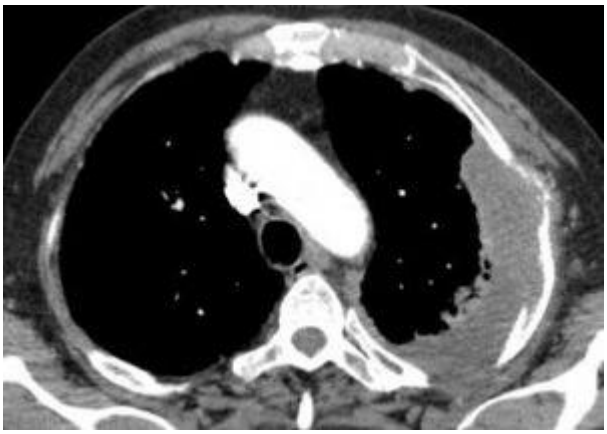
Malignancy
Elderly patients, male more than female
Medications: penicillamine, GOLD, captopril, and heavy metals: mercury and cadmium
Basement membrane thickening
Rheumatoid arthritis
Autoimmune disease: systemic lupus erythematosus (SLE), thyroid
Nephrotic syndrome is the main presentation
Hepatitis B
Odd infections - like syphilis, leprosy, HIV, schistosomiasis, malaria
Immune complex deposition with IgG and C3
Sickle cell disease.

Forty per cent remit without treatment, 30% develop endstage renal failure (ESRF).

[B]

45/A 70-year-old man complains of pain in the chest over the last three months associated with a 7 kg weight loss. He is a smoker of 10 cigarettes daily.

After initial chest x rays a CT scan was performed.



What is the diagnosis?

(Please select 1 option)

◯	Mesothelioma
◯	Neurofibroma
◯	Pleural effusion
◯	Pleural plaque
◯	Pneumonic consolidation

The diagnosis is a mesothelioma.

There is a large rind of soft tissue related to the left chest wall.

This is a malignant process as there is destruction of the associated rib excluding the other diagnoses.

[A]

46/A 61-year-old man with moderately severe COPD was admitted to the Emergency department after collapsing at home in front of his wife. Whilst in the ambulance en route to the hospital he had two episodes of ventricular tachycardia with loss of cardiac output, both of which revert to sinus rhythm with a single 200 joule shock.

On arrival at the hospital, the medical staff were informed by his wife that he has

been unwell for the past week with a lower respiratory tract infection, but did not want to bother his general practitioner. He had been breathless, wheezy and coughing up copious amounts of sputum. Over the past 24 hours his symptoms had deteriorated markedly and he had become drowsy.

He had a 40 pack year smoking history but had given up for over 10 years. Although disabled by his breathlessness he led an active life and had a good quality of life. He had never been admitted to hospital before.

On clinical examination he was drowsy with a GCS of 8/15. He was tachypnoeic with a respiratory rate of 40 breaths per minute and was taking very shallow breaths. His pulse was measured at 150 per minute and the cardiac monitor showed him to be in sinus rhythm. His oxygen saturations were 68% on 40% inhaled oxygen. Auscultation of his chest reveals feeble breath sounds bilaterally with a polyphonic wheeze.

His arterial blood gases on 40% oxygen are shown below:

pH	7.18	(7.36-7.44)
pO_2	5.8 kPa	(11.3-12.6)
pCO_2	9 kPa	(4.7-6.0)
HCO_3	16 mmol/L	(20-28)

He is commenced on nebulised bronchodilators and given 200 milligrams of hydrocortisone.

What is the most appropriate management for this gentleman?

(Please select 1 option)

○	Commence intravenous aminophylline
○	Commence intravenous magnesium
○	Decrease inspired oxygen to 28% via venturi mask and repeat the blood gas analysis in 30 minutes
○	Increase his inspired oxygen to 60% via venturi mask and repeat the blood gas in 30 minutes
○	Request immediate intubation and invasive ventilation.

This patient is critically unwell with type 2 respiratory failure and needs immediate intubation and ventilation.

Non-invasive ventilation is not indicated as he is too acidotic and drowsy given his GCS.

In addition copious sputum production and life-threatening arrhythmias are contraindications to non-invasive positive pressure ventilation (NIPPV).

Increasing his FiO_2 would not be helpful as he is in type 2 respiratory failure.

There is no role for intravenous magnesium or aminophylline in acute exacerbations of chronic obstructive pulmonary disease (COPD).

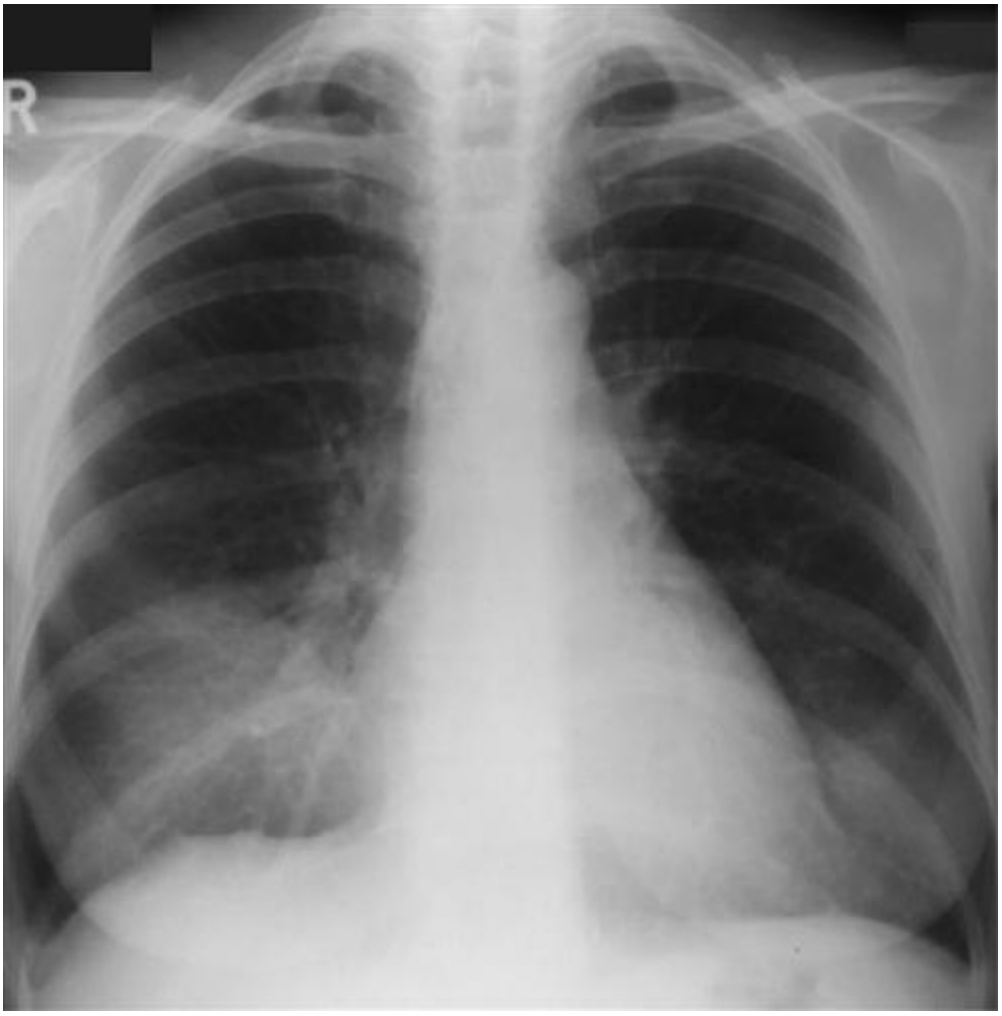
[E]

47/A 46-year-old asthmatic woman was admitted to the emergency department with severe breathlessness.

Her symptoms started four days previously after a coryzal illness. At first she had a cough with thick purulent sputum, but the cough subsequently became more productive. Her symptoms of breathlessness and wheeze had progressively worsened over the past 24 hours despite her general practitioner commencing prednisolone 40 mg and regular inhaled salbutamol.

On clinical examination she was distressed and was able to talk only in three-word sentences. Temperature was 36.9°C. Her respiratory rate was 30 per minute and pulse 130 beats per minute. Her oxygen saturations were 95% on 10 litres of oxygen. Auscultation of her chest reveals multiple high pitched polyphonic wheezes and diminished breath sounds bilaterally.

The chest radiograph is shown below:



What is the most likely cause for her deterioration?

(Please select 1 option)

◯	Bronchogenic carcinoma
◯	Invasive aspergillosis
◯	Mucous plugging
◯	Pneumococcal pneumonia
◯	Staphylococcal pneumonia

The chest x ray shows an indistinct right heart border with an adjacent shadow. This appearance, in conjunction with the clinical presentation, is typical of right middle lobe collapse due to mucous plugging.

Mucous plugging is not uncommon in asthmatic patients and can occasionally cause lobar collapse.

Treatment involves adequate hydration and chest physiotherapy.

Bronchoscopy with lavage may be required if this is unsuccessful.[C]

48/A 58-year-old South African man attended the respiratory outpatient clinic with severe breathlessness. He had recently moved to the United Kingdom to live with his children after working in the mining region of Pretoria.

His symptoms of breathlessness started two years previously and had progressively worsened. When reviewed, he had an MRC dyspnoea score of IV and needed to stop after walking 100 yards. He had no history of cough, sputum or wheeze. He had never been diagnosed with a respiratory condition in the past and was a lifelong non-smoker.

On examination he was clubbed and clearly breathless at rest. Pulse 100 beats per minute and regular with blood pressure 140/80 mmHg. The jugular venous pulse was not elevated. Auscultation of his heart revealed normal heart sounds with no added sounds and no audible murmurs. Examination of his respiratory system reveals symmetrically diminished breath sounds at his lung bases with fine end inspiratory crepitations. Abdominal examination was normal.

The chest radiograph showed irregular reticular shadowing affecting the periphery of the lower lobes. In addition there are multiple pleural plaques and areas of pleural thickening.

His full pulmonary function tests are shown below:

	Actual	% predicted
FVC (l)	2.5	40
FEV1(l)	2.4	44
FEV1/FVC(%)	96	113
TLC (l)	4.5	56
FRC(l)	1.6	54
RV(l)	1.4	70
Kco(ml/m/mm Hg)	2.30	51

What is the most likely cause of his symptoms?

(Please select 1 option)

○	Asbestosis
○	Asbestos-related pleural thickening
○	Bronchiolitis obliterans organising pneumonia (BOOP)

	Idiopathic pulmonary fibrosis
	Malignant mesothelioma

This patient has a restrictive defect secondary to pulmonary fibrosis based on his pulmonary function tests and chest radiograph.

The fact that he has pleural plaques on his chest radiograph also means that he has been exposed to asbestos. Blue asbestos is still mined in South Africa with little protection given to workers.

Asbestosis, defined as diffuse interstitial fibrosis secondary to asbestos inhalation is the most likely answer, although idiopathic pulmonary fibrosis is still a possibility.

The chest radiograph appearance is not typical of BOOP, and neither malignant mesothelioma nor pleural thickening are consistent with the pulmonary function tests shown.

Medical Research Council dyspnoea scale:

Grade	Degree of breathlessness related to activities
1	Not troubled by breathlessness except on strenuous exercise
2	Short of breath when hurrying or walking up a slight hill
3	Walks slower than contemporaries on level ground because of breathlessness, or has to stop for breath when walking at own pace
4	Stops for breath after walking about 100 m or after a few minutes on level ground
5	Too breathless to leave the house, or breathless when dressing or undressing

[A]

49/A 55-year-old woman was referred to the respiratory outpatient clinic with a two-year history of shortness of breath and a daily cough with purulent sputum. She reported that her symptoms had progressed slowly and she that she was now breathless on climbing two flights of stairs. She had been prescribed a salbutamol inhaler by her GP, which provided some short-term relief from her symptoms.

She had a 50-pack year smoking history and gave up smoking two months previously with the aid of nicotine replacement therapy. There was no occupational history of note and she had never kept pets at home.

On clinical examination there is no evidence of cyanosis, clubbing or lymphadenopathy. Pulse 80 beats per minute and regular, blood pressure 135/75 mmHg. Heart sounds were normal. Her chest wall expansion is symmetrically diminished and there is evidence of static hyperinflation. Auscultation reveals slightly

diminished breath sounds with a prolonged expiratory phase and a polyphonic wheeze. Abdominal examination was unremarkable.

Her spirometry and ear lobe blood gas analysis are shown below

Spirometry:

FVC	2.4 Litres	(88% predicted)
FEV1	1.5 Litres	(60% predicted)
FEV1/FVC	0.625	

Ear lobe blood gas analysis:

pH	7.46	(7.36-7.44)
pO_2	8.5 kPa	(11.3-12.6)
pCO_2	3.8 kPa	(4.7-6.0)
HCO_3	24 mmol/L	(20-28)

What is the most appropriate next step in her management?

(Please select 1 option)

○	Precribe a long acting anticholinergic inhaler
○	Prescibe an inhaled corticosteroid
○	Prescribe an oral theophylline
○	Prescribe regular nebulised ventolin
○	Refer for formal long term oxygen therapy assessment

This patient has moderate COPD based on her symptoms, smoking history and obstructive spirometry. The severity of COPD is based on FEV1, which in her case is moderate or GOLD stage 2.

Any patients with COPD who are symptomatic should receive a long acting bronchodilator either an anticholinergic or a beta-2 agonist. Inhaled steroids should only be used in patients with an FEV1<50% predicted and who have had two or more exacerbations in the last year. There is little evidence for the beneficial effect of theophylline in COPD and there is no evidence for the superior efficacy of nebulised as opposed to inhaled salbutamol. He does not require long term oxygen therapy based on these current blood gas results.

[A]

50/A 21-year-old Asian medical student presented to the emergency department with a month long history of fever, night sweats and a cough productive of purulent sputum.

On examination, he was febrile (38.5°C) with pulse 110 beats per minute and a respiratory rate of 22 breaths per minute. Coarse crackles were heard over the right lung apex. His chest x ray showed right upper lobar consolidation with a single cavitating lesion.

Investigations revealed:

Haemoglobin	11.5 g/dl	(13.0-18.0)
White cell count	12.5 $x10^9$/l	(4-11)
Platelets	425 $x10^9$/l	(150-400)
Serum sodium	140 mmol/l	(137-144)
Serum potassium	4.2 mmol/l	(3.5-4.9)
Serum urea	3.1 mmol/L	(2.5-7.5)
Serum creatinine	76 µmol/l	(60-110)
Serum C reactive protein	254 mg/l	(<10)

A sputum sample showed acid-alcohol fast bacilli.

He was started on anti-tuberculous therapy with rifampicin, isoniazid, pyrazinamide and ethambutol. Within seven days his fever had settled, he was feeling better and his inflammatory markers were settling.

Contact tracing reveals that he lived with his parents and two sisters. His older sister, who had lived in the United Kingdom all her life, subsequently has a strongly positive Mantoux test. She otherwise feels well and has no symptoms of anorexia, weight loss, fever, night sweats or cough and her chest radiograph is normal.

What is the most appropriate management for his sister?

(Please select 1 option)

○	Arrange to see her in clinic in one month with a repeat chest radiograph and clinical assessment
○	Ignore the Mantoux test as she is physically well and has no signs of active tuberculosis
○	Prescribe a three month course of rifampicin and isoniazid
○	Prescribe quadruple tuberculosis treatment

◯	Repeat her Mantoux test after one month

Prevention of cross infection is extremely important in the management of tuberculosis, and is much more likely in patients who are smear positive. The people most likely to be infected are close contacts, such as family and house mates.

People with disease should be fully treated, whereas those with infection but no evidence of disease (as is the case here), should receive prophylaxis with isoniazid for six months or rifampicin and isoniazid for three months.

[C]

51/A 24-year-old-man was referred to the respiratory outpatient clinic with symptoms of cough, shortness of breath and wheeze.

His symptoms started two months previously with a nocturnal cough. His general practitioner had commenced him on two puffs of combination Flixotide 100 micrograms and Serevent 50 micrograms (Seretide 50/100) twice a day via a metered dose inhaler.

His nocturnal symptoms improved after starting the inhalers, but he was still symptomatic whenever he exercised. He also had a history of perennial rhinitis which was not adequately controlled with antihistamine medication. There was no other past history of note.

His peak flow chart shows a suboptimal average peak expiratory flow rate of 420 l/min (80% predicted) with a variation of 20%. Examination of his nasal passage reveals erythematous and oedematous mucosa with no evidence of polyps. Examination of his respiratory system reveals a mild polyphonic wheeze.

During the consultation he stated that he was not keen to increase his inhaled steroid medication as he has heard this may predispose him to future diabetes and osteoporosis.

Which is the next most appropriate step in his management?

(Please select 1 option)

◯	Add in a leukotriene antagonist tablet
◯	Commence a long acting anticholinergic inhaler
◯	Increase the steroid dose of his Seretide inhaler
◯	Prescribe an oral theophylline
◯	Replace Seretide with sodium cromoglicate

Leukotriene antagonists are licensed for use in asthma with allergic rhinitis and have been shown to be as effective as doubling the dose of inhaled steroid. They are also particularly useful in exercise-induced asthma and adult onset aspirin sensitive asthma.

Sodium cromoglicate is a useful suggestion for exercise-induced asthma but should not replace an inhaled steroid.

Long acting anticholinergics are not licensed and should not be used in asthma.

Oral theophyllines should be considered only if patients are on maximal doses of inhaled steroids and all other treatment avenues have been explored.

[A]

52/A 60-year-old woman was referred to the rapid access lung cancer clinic after having a chest x ray which she was told was abnormal. She reported feeling unwell for the past six weeks with lethargy, intermittent fever and myalgia. She noticed that she became more breathless on exertion and had difficulty climbing steep hills.

She had one episode of haemoptysis which she attributed to nose bleeds, which she was experiencing frequently. She had a 40 pack/year smoking history and kept no pets at home. She was a retired nurse and had worked in the NHS for 40 years.

On examination she looked unwell and had a temperature of 38.4°C. Her blood pressure was elevated at 160/100 mmHg, pulse 100 beats per minute and oxygen saturations of 94% on room air. On palpation of the carotid pulse, she had marked tenderness of her right carotid artery. There was no palpable lymphadenopathy in her cervical, axillary or inguinal regions. Her heart sounds were normal with no audible murmurs. Respiratory examination revealed vesicular breath sounds which were of normal intensity. There was no audible wheeze or crepitations. Abdominal examination was normal.

A repeat chest radiograph revealed alveolar shadowing in the right upper lobe. The previous chest radiograph, which she brought with her to clinic, showed alveolar shadowing in the right mid-zone.

Investigations revealed:

Haemoglobin	11.5 g/dl	(11.5-16.5)
White cell count	10.0 $x10^9$/l	(4-11)
Neutrophils	6.4 $x10^9$/l	(1.5-7)
Lymphocytes	2.0 $x10^9$/l	(1.5-4.0)
Monocytes	0.8 $x10^9$/l	(0-0.8)

Eosinophils	0.7 $x10^9$/l	(0.04-0.4)
Basophils	0.1 $x10^9$/l	(0-0.1)
Platelets	500 $x10^9$/L	(150-400)
ESR (Westergren)	100 mm/1^{st} hour	(0-15)
Serum sodium	140 mmol/l	(137-144)
Serum potassium	4.2 mmol/l	(3.5-4.9)
Serum urea	13.7 mmol/l	(2.5-7.5)
Serum creatinine	170 µmol/l	(60-110)
Anti-nuclear antibody	Positive	
Perinuclear-ANCA (pANCA)	Negative	
Cytoplasmic-ANCA (cANCA)	Positive	
Anti-GBM	Negative	

A Heaf test showed a Grade 2 reaction.

What is the most likely diagnosis?

(Please select 1 option)

○	Bronchoalveolar cell carcinoma
○	Bronchiolitis obliterans organising pneumonia
○	Goodpasture's syndrome
○	Lymphangitis carcinomatosa
○	Wegener's granulomatosis

The symptoms, clinical signs and laboratory findings are classical of Wegener's granulomatosis.

In addition to causing vasculitis (causing carotid artery tenderness), Wegener's is also a cause of migrating alveolar shadowing. The raised ESR, renal dysfunction and positive C-ANCA are also in keeping with the diagnosis.

P-ANCA can also be raised in Wegener's granulomatosis.

[E]

53/A 66-year-old woman presented to the outpatient clinic with a one year history of non-productive cough.

On further questioning she reported that the cough disappeared at night and was worse on laughing and talking. She also complained of an intermittent hoarse voice. There were no other associated respiratory symptoms such as wheeze or breathlessness.

She had a past medical history of essential hypertension and depression and had been taking an ACE inhibitor. However, because of the dry cough her general practitioner had discontinued the ACE inhibitor six months previously, with no improvement in her symptoms.

She had diligently kept an accurate peak flow diary over the past eight weeks. This shows a 5% diurnal variability.

On clinical examination blood pressure was 90/55 mmHg, pulse 80 beats per minute, and oxygen saturations of 99% on air. Heart sounds were of normal quality and there were no audible murmurs. Auscultation of the chest revealed vesicular breath sounds with no added wheeze or crepitations. Abdominal examination was normal, and a chest radiograph was reported as normal.

What is the most likely diagnosis?

(Please select 1 option)

○	Asthma
○	Cough secondary to ACE inhibitor therapy
○	Gastro-oesophageal reflux disease
○	Globus hystericus
○	Psychogenic cough

Gastro-oesophageal reflux disease (GORD) is one of the three commonest causes of chronic cough, the other two being asthma and postnasal drip.

Her clinical presentation is typical of cough secondary to GORD, with symptoms worse on talking and laughing. In all but the most severe cases, the lower oesophageal sphincter closes at night leading to a cessation of symptoms.

Asthma is also a possibility, but a peak flow variability of at least 20% is required for an accurate diagnosis to be made.

Cough secondary to angiotensin-converting enzyme (ACE) inhibitors should take a maximum of three months to disappear after the drug is stopped.

Globus hystericus usually gives symptoms of dysphagia and psychogenic cough is a diagnosis of exclusion.

[C]

54/A 52-year-old male presented to the respiratory outpatient clinic with a history of progressive breathlessness on exertion.

He was a keen rambler, and had first noticed his symptoms some twelve months previously when he was struggling to keep up with his wife.

He had no associated cough, sputum production, wheeze or chest pain. His past medical history included a transient ischaemic attack (TIA) three months previously, seasonal allergic rhinitis and hypercholesterolaemia. He was a lifelong non-smoker and had been started on aspirin and simvastatin since his TIA. He admitted that his diet was poor and contained a significant amount of saturated fats.

On clinical examination he was found to be obese (BMI 30) and plethoric. His blood pressure was 100/80 mmHg, Temperature 36.8°C, pulse 96 beats per minute and oxygen saturations of 90% on room air. On auscultation there was an ejection systolic murmur loudest in the left second intercostal space.

His ECG showed right axis deviation with right bundle branch block.

His full pulmonary function tests are shown below:

	Actual %	**Predicted**
FVC	4.30 L	87%
FEV1	3.62 L	84%
FEV1/FVC	84%	99%
TLC	6.90 L	86%
RV/TLC	30.0 %	110%
DLCO (ml/m/mm Hg)	56 mL /m/mm Hg	163%

What is the most likely underlying diagnosis?

(Please select 1 option)

○	Atrial septal defect
○	Chronic pulmonary emboli
○	Cor pulmonale

	Obstructive sleep apnoea
	Pulmonary restriction secondary to obesity

The history of a recent TIA, along with the clinical and ECG findings, are consistent with an atrial septal defect.

The grossly elevated $D_L$co is secondary to the left-right shunt and increased pulmonary blood flow. In contrast, chronic pulmonary emboli will cause a low $D_L$co. Although the patient has a mild ventilatory defect secondary to obesity, this does not explain the clinical findings.

Other causes of a raised $D_L$co include asthma, obesity, exercise, polycythaemia and any cause of alveolar haemorrhage (Goodpasture's syndrome, Wegener's granulomatosis, etc).

[A]

55/A 55-year-old man presented to the outpatient clinic with a six month history of breathlessness, wheeze and a non-productive cough. He had been given a salbutamol inhaler by his GP which had not significantly improved his symptoms.

He was presently unemployed, but had worked for 20 years in a nearby coal mine. He had smoked since the age of 20 and had a twenty pack per year smoking history. He was particularly concerned as his father had died of emphysema at the age of 65.

Clinical examination revealed a hyperinflated chest with reduced breath sounds, scattered rhonchi throughout the chest and a prolonged expiratory phase. His heart sounds were quiet but no murmurs could be heard. Examination of his abdomen was normal.

Spirometry revealed:

Actual (L)	Predicted (%)
FVC 3.2	98
FEV1 2.0	70
FEV1/FVC	62.5

Which one of the following options most strongly supports a diagnosis of COPD?

(Please select 1 option)

	An increase in FEV1 of 12% post bronchodilator
	No variability in peak expiratory flow rate over the past two months

○	Occupational exposure to coal dust
○	Positive family history of COPD
○	Twenty pack year smoking history

Chronic obstructive pulmonary disease (COPD) is characterised by progressive decline in lung function as a result of airflow obstruction. Symptoms and peak flow values do not change markedly from day to day, in marked contrast to asthma.

Although the majority of patients with COPD will have a significant history, many patients with asthma will also be smokers.

Coal dust exposure has not been definitively linked to COPD, but this association is difficult to prove since most coal workers were smokers.

[B]

56/A 75-year-old South African woman with known rheumatoid arthritis was admitted to hospital with a three week history of weight loss, night sweats and cough.

Her general practitioner had been unsuccessfully treating her with amoxicillin for the past week and had decided to admit her when she developed haemoptysis. She was on maintenance prednisolone 10 mg once per day and four weeks earlier she had received infliximab for a flare up of rheumatoid arthritis.

She lived with her husband, who was usually her main carer, but had been admitted to hospital himself with influenza three days earlier. She was a lifelong non-smoker and worked most of her life as a missionary in South Africa and Zimbabwe.

On examination she looked cachexic and was pyrexial with a temperature of 38.4°C. Her blood pressure was 180/100 mmHg, pulse 120 beats per minute and oxygen saturations of 89% on room air. Her heart sounds were normal and there were no audible murmurs. Auscultation of her lung fields revealed bronchial breath sounds in the left upper zone. Examination of her abdomen was normal.

Mantoux test	< 5 mm	(after 48 hours)

A chest radiograph revealed cavitating left upper lobe consolidation.

What is the most likely diagnosis?

(Please select 1 option)

○	*Klebsiella* pneumonia
○	*Pneumocystis jirovecii* pneumonia

	Post-primary tuberculosis
	Squamous cell carcinoma
	Staphylococcal pneumonia

Treatment with tumour necrosis factor (TNF) alpha antagonists significantly increases the risk of post-primary tuberculosis. In this case the Mantoux test will be unreliable since the patient is on maintenance steroids.

Although immunosuppressed patients are at risk of pneumocystis pneumonia (PCP), the chest radiograph appearance described is not typical of this.

The Heaf test involves the multipuncture of skin using 100,000 tuberculin units/ml. It is read after one week and graded according to the coalescence of the puncture dots.

The Mantoux test involves the intradermal injection of 0.1 ml of 1 in 1000 tuberculin units and is read after 48 hours. An induration of less than 5 mm is considered negative.

[C]

57/An 87-year-old man was admitted to the Emergency department with a two hour history of sharp central chest pain and breathlessness.

One week ago he noticed his left leg had become swollen, but had decided not to consult medical advice. Apart from essential hypertension, for which he was taking bendroflumethiazide, he had no other medical conditions. He was a lifelong non-smoker.

On clinical examination he was dyspnoeic at rest with a respiratory rate of 28 breaths per minute. His blood pressure was 90/60 mmHg, pulse 110 beats per minute and regular with oxygen saturations of 86% on room air. Auscultation of his chest revealed a loud second heart sound over the right second intercostal space.

Analysis of arterial blood gases on air showed:

pH	7.35	(7.36-7.44)
PaO_2	7.0 kPa	(11.3-12.6)
$PaCO_2$	3.8 kPa	(4.7-6.0)
Standard HCO_3	24 mmol/L	(20-28)

A CT pulmonary angiogram showed a large saddle embolism affecting his pulmonary trunk.

What is the most appropriate management?

(Please select 1 option)

○	Continuous infusion of unfractionated heparin
○	Low molecular weight (LMW) heparin
○	Urgent placement of inferior vena cava (IVC) filter
○	Urgent referral for endarterectomy
○	Urgent thrombolysis

In the presence of shock, the treatment of choice in sub-massive pulmonary embolism is thrombolysis and oxygen therapy.

Thrombolysis is indicated in the presence of shock (hypotension, acidosis, etc.). However, the mortality in elderly patients who are thrombolysed is significant (up to 5% in some series).

Endarterectomy is never performed in the acute setting and an IVC filter is not the appropriate management in this case.

The outcome for unfractionated heparin is no better than LMW heparin, and is associated with a higher risk of haemorrhage.

[E]

58/A 14-year-old boy with a known nut allergy was admitted to the Emergency department following the accidental ingestion of a pistachio nut biscuit.

On examination he had audible stridor and looked peripherally shut down. His blood pressure was 70/50 mmHg, oxygen saturations 86% on room air and respiratory rate of 26 breaths per minute. His pulse was feeble.

What is the most appropriate immediate management?

(Please select 1 option)

○	Intramuscular adrenaline
○	Intravenous adrenaline
○	Intravenous antihistamine
○	Intravenous hydrocortisone
○	Subcutaneous adrenaline

Anaphylactic shock requires urgent treatment with adrenaline, which is absorbed at a faster rate intramuscularly than subcutaneously.

In this scenario, waiting to obtain intravenous access in a peripherally shutdown patient could prove fatal.

Although hydrocortisone and antihistamine therapy are indicated in this patient, they will take hours to work and will have no effect on shock.

[A]

59/A 33-year-old pregnant woman was admitted to the emergency department agitated and confused following a seizure.

She awoke one hour earlier to find her husband lying dead in the bed next to her. An ultrasound scan confirmed that her 26-week-old fetus had also died. She informed the admitting staff that both she and her husband had been unwell with 'flu like symptoms for the past week. They had spent the previous day painting their bedroom and had eaten re-heated Chinese food that night.

Her past medical history included asthma which was well controlled on inhaled corticosteroids. She had a 15 pack year smoking history, but had given up when she learnt that she had become pregnant.

On clinical examination she was tachypnoeic with a respiratory rate of 24 breaths per minute, blood pressure of 90/60 mmHg, pulse of 120 beats per minute and oxygen saturations of 98% on air. There was no rash or purpura visible on her body. Heart sounds were normal with no murmurs or added sounds. Auscultation of her chest revealed vesicular breath sounds with no added sounds. Abdominal examination was normal.

What is the most likely cause of this tragic event?

(Please select 1 option)

○	*Bacillus cereus* septicaemia
○	Carbon monoxide poisoning
○	Lead poisoning
○	Methaemoglobinaemia
○	Meningococcal septicaemia

Carbon monoxide poisoning still accounts for 75 deaths per year and often presents with non-specific symptoms such as headache, malaise, myalgia and weakness.

The woman in the question survived because the fetal haemoglobin of her unborn child preferentially bound to the poisonous carbon monoxide gas.

Lead poisoning does not present so acutely, and there is nothing in the history to suggest methaemoglobinaemia.

Although septicaemia secondary to *Bacillus cereus* or meningococcus are possible, the clinical findings do not support this.

[B]

60/Which of the following patients with lobar pneumonia has the worst prognosis?

(Please select 1 option)

○	70-year-old patient with a white cell count of 20 × 10^9/L and a serum C-reactive protein of 250 IU/L.
○	64-year-old patient with a white cell count of 1 × 10^9/L, serum urea of 18 mmol/L, serum C-reactive protein of 200 IU/L and a pO2 of 7kPa on air
○	66-year-old confused patient with a respiratory rate of 30 per minute, blood pressure of 80/40 mmHg and a serum urea of 16 mmol/L
○	84-year-old patient with a white cell count of 16 × 10^9/L, blood pressure of 90/50 mmHg and a past medical history of ischaemic heart disease
○	A 29-year-old with confirmed Legionella pneumophilia pneumonia.

Prognosis for patients with pneumonia is now assessed using the validated CURB 65 Score, where Confusion, Uraemia, high Respiratory rate, low Blood Pressure and age >65 years are all given one point, to make up a total possible score of 5. Patients with higher CURB65 scores have a worse 30 day mortality.

[C]

61/A 60-year-old farmer presented to the Emergency department with a two week history of progressively worsening cough, breathlessness and myalgia. His symptoms had become particularly worse after he recently finished baling hay indoors, ready for the winter.

He had no other previous medical history of note and was a lifelong non-smoker. He recently purchased a budgerigar from an internet auction site.

On examination his temperature was 38.4°C, blood pressure 160/100 mmHg, pulse 130 beats per minute and oxygen saturations of 86% on air. His heart sounds were normal and there were no audible murmurs. Auscultation of the chest revealed bronchial breath sounds at the right base. Abdominal examination revealed a soft and non-tender abdomen with no palpable masses or abnormal enlargement of organs.

Laboratory investigations revealed:

Haemoglobin	14.5 g/dL	(13.0-18.0)
White cell count	20.0 $x10^9$/L	(4-11 $x10^9$)
Neutrophils	18.0 $x10^9$/L	(1.5-7 $x10^9$)
Lymphocytes	1.0 $x10^9$/L	(1.5-4.0 $x10^9$)
Monocytes	0.8 x 10^9/L	(0-0.8 $x10^9$)
Eosinophils	0.2 $x10^9$/L	(0.04-0.4 $x10^9$)
Basophils	0.01 $x10^9$/L	(0-0.1 $x10^9$)
Platelets	390 $x10^9$/L	(150-400 $x10^9$)
ESR (Westergren)	90 mm/1st hour	(0-15 mm/1st hour)
Serum sodium	140 mmol/L	(137-144)
Serum potassium	4.2 mmol/L	(3.5-4.9)
Serum urea	13.7 mmol/L	(2.5-7.5)
Serum creatinine	120 mol/L	(60-110)
Serum avian precipitins	Positive	
Micropolyspora faeni precipitins	Negative	

A chest radiograph revealed patchy consolidation in the right lower zone.

What is the most likely diagnosis?

(Please select 1 option)

○	Avian influenza
○	*Chlamydia psittaci* pneumonia
○	*Coxiella burnetii* pneumonia
○	Extrinsic allergic alveolitis
○	Farmer's lung

This patient has acute pneumonia with a high white cell count, high inflammatory markers and chest radiograph changes suggestive of an infective process. The most likely pathogen in this case is*Chlamydia psittaci*, a pathogen acquired most commonly from birds.

Extrinsic allergic alveolitis would be less likely to cause such acute symptoms, and chest radiograph changes are usually bilateral.

Positive serum avian precipitins are not diagnostic of extrinsic allergic alveolitis (EAA), and only suggest the patient has had exposure to birds.

Farmer's lung, a form of hypersensitivity pneumonitis, is now very rare since the majority of farmers no longer bale hay in wet conditions. When present, it is most commonly associated with positive *Micropolyspora faeni* antibodies.

[B]

62/A 54-year-old man was admitted to casualty with a three-day history of increasing dyspnoea and cough productive of purulent sputum.

There was no associated haemoptysis. He was known to suffer from chronic obstructive pulmonary disease, which usually caused him to be breathless on exertion, but his normal exercise tolerance was one mile walking on the flat at his own pace. He gave up smoking five years previously, but had smoked up to twenty cigarettes a day for over thirty years. He was a retired plumber and lived with his wife. His only medicatiotion was Tiotropium 18 mcg once daily.

On examination he was breathless at rest and found it difficult to talk in complete sentences. He was sweaty and in some distress. His temperature was 38.2°C. His peak flow was 210 L/min. His pulse rate was 120 beats per minute and blood pressure is 90/60 mmHg. His respiratory rate was 24 breaths per minute. He had reduced expansion bilaterally. Percussion note was resonant. His breath sounds were vesicular with occasional scattered expiratory wheeze.

Investigations revealed:

Haemoglobin	15.8 g/dL	(13.0-18.0)
White cell Count	16.5 $\times 10^9$/L	(4-11)
Platelets	470 $\times 10^9$/L	(150-400)
Sodium	133 mmol/L	(137-144)
Potassium	3.4 mmol/L	(3.5-4.9)
Urea	12.1 mmol/L	(2.5-7.5)
Creatinine	134 µmol/L	(60-110)
Troponin T	<0.03 U/L	(<0.03)

Arterial blood gases on air:

PaO_2	6.8 kPa	(11.3-12.6)

$PaCO^2$	6.8 kPa	(4.7-6.0)
HCO_3^-	24 mmol/L	(20-28)
pH	7.32	(7.36-7.44)

His electrocardiogram showed as sinus tachycardia with a ventricular rate of 130 beats/minute. There were no ST segment changes. A chest radiograph demonstrated hyperinflated lung fields with no pneumothorax and no focal areas of consolidation.

He was treated with nebulised bronchodilators, oral prednisolone and 24% oxygen. However, despite treatment his condition deteriorated: he became agitated and more breathless. On examination his respiratory rate was 25 breaths per minute with a pulse is 130 beats per minute and blood pressure is 80/50 mmHg.

Repeat arterial blood gases on 24% oxygen showed:

PaO_2	6.3 kPa	(11.3-12.6)
$PaCO_2$	7.9 kPa	(4.7-6.0)
HCO_3^-	27 mmol/L	(20-28)
pH	7.28	(7.36-7.44)

What is the most appropriate next step in this patients management?

(Please select 1 option)

○	Doxapram infusion
○	Increase inspired oxygen concentration to 28%
○	Initiate non-invasive ventilation
○	Intubation with endotracheal tube and formal ventilation
○	Stop 24% oxygen and allow patient to breathe room air

This patient has an acute exacerbation of chronic obstructive pulmonary disease (AECOPD). He is in type two respiratory failure. He needs respiratory support. The choices are either non-invasive ventilation (NIV) through a full face or nasal mask or endotracheal intubation (ETI) and ventilation on an intensive care unit.

In selected patients with type 2 respiratory failure due to AECOPD NIV has been shown to decrease mortality and length of hospital stay over ETI. However there are contraindications to NIV:

Contraindications to NIV:

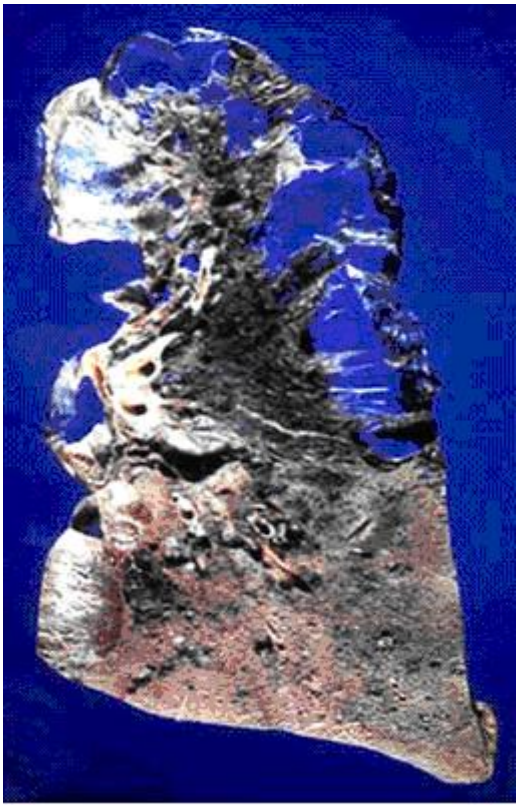
Haemodynamically unstable
Confusion / impaired consciouness
Vomiting
Inability to protect airway
Facial burns/traums
Fixed obstruction of the upper airway
Undrained pneumothorax.

It should be noted however NIV may be used despite many of these contraindications if NIV is to be the ceiling of treatment. This patient is haemodynamically unstable and agitated and therefore should be treated with ETI in an ITU.

[B]

63/A 67-year-old lady with a longstanding history of respiratory disease was admitted to hospital acutely breathless. Despite appropriate treatment, she died.

The picture below shows the macroscopic appearance of her lung at post-mortem examination.



One month before her admission to hospital, she had been reviewed in the respiratory outpatient clinic and had lung function tests performed. The normal range of pulmonary function tests for this lady's age and height are given below.

Forced expiratory volume in 1 second (FEV1)	1.80 - 3.02 litres

Forced vital capacity (FVC)	2.16 - 3.58 litres
Diffusion capacity	5.91 - 9.65 mmol/min/kPa
Total lung capacity	4.25 - 6.22 litres
Residual volume	1.46 - 2.48 litres

What would have been the most likely result of her pulmonary function tests from her outpatient review?

(Please select 1 option)

○	FEV1 0.90 litres, diffusion capacity 2.80 mmol/min/kPa, residual volume 2.73 litres
○	FEV1 0.90 litres, diffusion capacity 6.50 mmol/min/kPa, residual volume 2.73 litres
○	FEV1 0.90 litres, FVC 1.10 litres, diffusion capacity 2.80 mmol/min/kPa
○	FEV1 0.90 litres, FVC 1.10 litres, diffusion capacity 6.50 mmol/min/kPa
○	FEV1 1.90 litres, FVC 2.90 litres, diffusion capacity 2.80 mmol/min/kPa

This specimen of lung shows large upper lobe bullae associated with emphysema.

This would produce an obstructive picture on spirometry with a reduced FEV1.

The FVC is often reduced as well but not to the same degree so that the FEV1/ FVC ratio is less than 70%.

There is hyperinflation resultant from air trapping which produces an increased residual volume and total lung capacity.

In addition in emphysema there is a reduction in diffusion capacity.

In asthma the diffusion capacity is normal although the transfer co-efficient may be increased during an acute exacerbation

[A]

64/A 28-year-old, 225 kg male was referred for investigation of breathlessness prior to undergoing a gastropexy operation.

He admitted to being breathless on walking 100 yards and also complained of a non-productive cough on waking each morning.

His past medical history included type 2 diabetes mellitus and a history of childhood asthma and rhinitis. He smoked twenty cigarettes per day and drank at least ten units of alcohol every evening. He lived with his father who had kept pigeons for the past five years.

As part of his investigations he underwent full pulmonary function tests the results of which are shown below.

	Actual	% predicted
FVC (l)	3.72	61
FEV1(l)	3.05	64
FRC (l)	1.42	34
RV (l)	1.01	45
TLC (l)	4.94	60
DLCO (ml/m/mm Hg)	29.13	61
DLCO/VA	4.95	94

What is the most likely cause of his breathlessness?

(Please select 1 option)

○	Asthma
○	Bronchiolitis obliterans
○	Extrinsic allergic alveolitis
○	Left ventricular failure
○	Obesity

These tests are consistent with obesity, that is, extra-thoracic restriction.

For obesity to cause a reduced total lung capacity (TLS) the weight (kg) / height (cm) must generally exceed 1.

Reductions in functional residual capacity (FRC), vital capacity (VC) and residual volume (RV) can occur with lesser degrees of obesity.

The reduced gas transfer corrects to normal when alveolar volume (AV) is incorporated. This implies normal pulmonary gas exchange and the low DL_{CO} is probably due to basal hypoventilation as a direct consequence of obesity.

[E]

65/A 25-year-old man was admitted to the Emergency department after being found unconscious by his girlfriend. His past medical history included anxiety and depression for which he was taking a combination of benzodiazepines and tricyclic antidepressants.

His girlfriend estimates that he has ingested fifty milligrams of diazepam and five hundred milligrams of amitriptylline. Next to his body she also discovered a suicide note and an empty bottle of vodka. His past medical history included asthma, which was well controlled with high dose inhaled corticosteroids.

On examination he was drowsy, with a Glasgow coma score of 7. His temperature was 34.8°C, pulse 120 per minute and blood pressure 80/50 mmHg. Bronchial breath sounds were heard over the right upper zone.

A chest x ray revealed right upper lobe consolidation.

His arterial blood gases on 15L of oxygen per minute via a reservoir bag mask showed:

pH	7.2	(7.36-7.44)
$PaCO_2$	9.5 kPa	(4.7-6.0)
PaO_2	12.0 kPa	(11.3-12.6)
HCO_3	27.3 mmol/l	(20-28)

What is the most appropriate management?

(Please select 1 option)

○	Administration of intravenous flumazenil and maintain high flow oxygen
○	Change his oxygen to a 28% venturi mask and repeat the ABG
○	Continue with high flow oxygen and fast bleep the on-call anaesthetist for an ETT
○	Continuous positive pressure ventilation (CPAP) via mask
○	Nasal intermittent positive pressure ventilation (NIPPV) via mask

Intubation is the only correct option here as his GCS is less than 8 and the patient is suffering with an unprotected airway.

The reason this patient has ventilatory failure is a combination of things secondary to a reduced concious level, aspiration pneumonia and respiratory suppressant drugs.

He is not a chronic CO_2 retainer and needs high concentration oxygen until he is intubated.

Non-invasive ventilation would be inappropriate since he is not protecting his airway.

Flumazenil is contraindicated since the tricyclic antidepressant drugs he has taken will significantly reduce his seizure threshold.

[C]

66/A 54-year-old man with a previous history of allergic rhinitis and adult onset asthma presented to the respiratory outpatients clinic with a six month history of worsening breathlessness, wheeze and intermittent abdominal pain. His asthma had previously been well controlled on high dose inhaled corticosteroids.

One month previously he had been admitted with acute coronary syndrome following a prolonged bout of chest pain. A subsequent coronary angiogram was normal.

On examination, he had a purpuric rash over his abdomen and lower extremities. Auscultation of his chest reveals bilateral polyphonic wheeze. Abdominal examination revealed some generalised tenderness around his umbilical region but no evidence of peritonism.

His laboratory investigations are as follows:

Haemoglobin	14.5 g/dL	(13.0-18.0)
White blood cells	16.1 $\times 10^9$/L	(4-11)
Neutrophils	4.5 $\times 10^9$/L	(1.5-7)
Lymphocytes	0.8 $\times 10^9$/L	(1.5-4)
Monocytes	0.8 $\times 10^9$/L	(0-0.8)
Eosinophils	10 $\times 10^9$/L	(0.04-0.4)
Platelets	390 $\times 10^9$/L	(150-400)
Erythrocyte sedimentation rate	90 mm/1st hour	(0-20)
Serum sodium	140 mmol/L	(137-144)
Serum potassium	4.2 mmol/L	(3.5-4.9)
Serum urea	13.7 mmol/L	(2.5-7.5)
Serum creatinine	170 µmol/L	(60-110)
Serum IgE	Elevated	
P-ANCA	Positive	

C-ANCA	Negative

What is the most likely diagnosis?

(Please select 1 option)

	Allergic bronchopulmonary aspergillosis
	Churg-Strauss syndrome
	Goodpasture's syndrome
	Polyarteritis nodosa
	Wegener's granulomatosis

Churg-Strauss syndrome is characterised by:

Asthma
Rhinitis, and
Peripheral blood.

Eosinophilia and vasculitis affects organs, such as:

Lungs
Kidneys
Peripheral nerves
Gastrointestinal tract, and
Heart.

Peri-nuclear staining antineutrophil cytoplasmic antibodies are present in 50% of affected patients, but their presence or absence is not diagnostic.

Serum IgE is very commonly elevated and correlates with disease severity.

[B]

67/A 19-year-old male was admitted to hospital with a two day history of nausea and vomiting.

He had been diagnosed with asthma since the age of 4 years and had been admitted to hospital one year previously with acute severe asthma that had required admission to level 2 care, though he did not require mechanical ventilation.

He was also under the care of the obesity clinic because of his weight, and on admission to hospital weighed over 220 kg. He lived with his parents and was unemployed. He was a non-smoker.

His prescribed medications included beclamethasone 500 mcg bd., salmeterol 50 mcg bd and sustained release theophylline. He has been well controlled on this regime for over nine months, but had developed increasing wheeze and breathlessness with a fever and a cough productive of purulent sputum five days previously. He had seen his general practitioner, who gave him a course of tablets, but his symptoms progressed despite this treatment.

On examination he was agitated and breathless at rest. His peak expiratory flow rate was 190L/min. He was febrile with a temperature of 37.8°C. His pulse was 120 beats per minute in sinus rhythm and his blood pressure was 120/88 mmHg.

What was the most likely treatment prescribed by his general practitioner?

(Please select 1 option)

○	Amoxicillin
○	Erythromycin
○	Montelukast
○	Prednisolone
○	Tetracycline

The patient has symptoms and signs typical of theophylline toxicity.

Phyllocontin is a slow release aminophylline. It is converted to theophylline which is then metabolised to inactive xanthine derivatives. The average half life is six hours but this is affected by a number of factors:

Factors decreasing theophylline clearance

Disease
Hepatic cirrhosis
Congestive cardiac failure
Chronic obstructive pulmonary disease
Acute pulmonary oedema
Pneumonia
Acute febrile illnesses

Drugs
Cimetidine
Oral contraceptive pill
Erythromycin
Ciprofloxacin
Fluvoxamine

Diet
Obesity
High carbohydrate intake

High methylxanthine intake (for example, tea, coffee)

Factors increasing theophylline clearance

Diet
Low carbohydrate
High protein intake

Social
Cigarette smoking

Drugs
Rifampicin
Carbamazepine.

[B]

68/A 62-year-old man presented to the Emergency department with sudden onset of left-sided chest pain and breathlessness. The pain came on suddenly while he was sitting in an armchair in front of the television and was described as sharp. The pain was followed almost immediately by a sensation of breathlessness.

His general health had been good, though he reported that he was usually breathless only on exertion, particularly walking up hills and walking up the stairs in his house. For several years he has had a productive cough in the morning, producing clear or white sputum which he attributes to a smoker's cough. He had never noticed any blood in his sputum. His appetite was good and his weight has been steady.

He is a retired plumber who lived with his wife and two cats. He was a smoker of 20 cigarettes a day and had been since the age of 16 years; he drank approximately twelve units of alcohol per week. There was no family history of note and he had no known allergies. His regular prescribed medications included salmeterol 50 mg BD, tiotropium 18 mg OD, and Combivent PRN.

Investigations showed:

Haemoglobin	14.8 g/dl	(13.0-18.0)
White cell count	9.7 x 10^9/l	(4-11)
Platelets	197 x 10^9/l	(150-400)
Troponin T	<0.03 U/l	(< 0.03)

The ECG showed sinus rhythm, with a large P wave, but was otherwise unremarkable.

The chest radiograph showed hyperinflated lung fields with a small apical left pneumothorax with a 3 cm margin between the lung surface and the chest wall. His oxygen saturations by pulse oximetry were 88% on room air. He was given 24% oxygen in the Emergency department, but remained dyspnoeic with oxygen saturations of 91% on 24% oxygen.

What should be the next step in his management?

(Please select 1 option)

○	Arrange CTPA
○	Discharge the patient home with repeat chest radiograph in seven days
○	Increase the concentration of inspired oxygen to 28%
○	Insert an intercostal drainage tube
○	Undertake a pleural aspiration

This patient has underlying chronic obstructive pulmonary disease (COPD) as evidenced by his history of exertional dyspnoea and persistent productive cough, together with his smoking history and the bronchodilators he has been prescribed.

He has now developed a pneumothorax of the left lung, which therefore can be classified as a secondary spontaneous pneumothorax. (Primary spontaneous pneumothoraces occur in otherwise healthy patients without pre-existing lung disease.)

The pneumothorax is significant (more than 2 cm rim of air on chest radiograph), has resulted in increased breathless, and in addition he is hypoxic. The treatment of secondary pneumothoraces is insertion of an intercostal tube and drainage.

Pleural aspiration is only recommended in secondary pneumothoraces if the pneumothorax is less than 2 cm, patient has minimal breathlessness and the patient is less than 50-years-old.

Its success rate is variable (33-67%) but is significantly less than that of primary pneumothorax (59-83%).

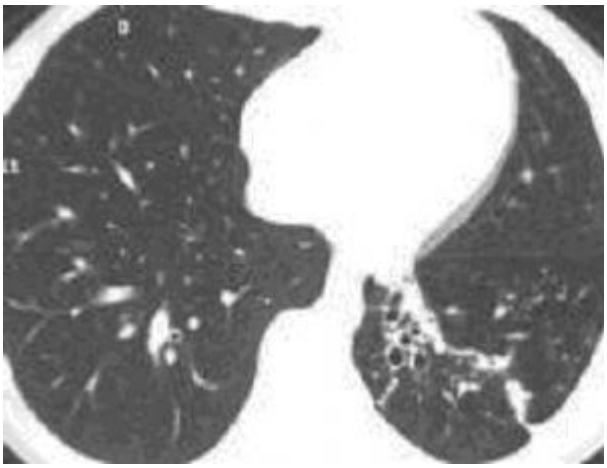
[D]

69/A 63-year-old lady with known rheumatoid arthritis was referred by her general practitioner to the outpatient clinic with a twelve-month history of increasing breathless and cough.

The breathlessness was most notable on exertion, although there was no limitation to her exercise capacity. She said that the cough was generally worse in the morning and was productive of mucoid white sputum. The sputum was occasionally yellow or green but she had never seen any blood in it. She lived with her husband and their disabled son. She had never smoked.

On examination, she appeared well and was apyrexial. Her pulse was 80 beats per minute in sinus rhythm with blood pressure 115/65 mmHg. On auscultation of her chest, the breath sounds were vesicular with bilateral inspiratory basal crepitations.

The chest radiograph showed increased interstitial shadowing at the left base and a computed tomography scan of the thorax was undertaken (shown below).



What is the most likely cause of her respiratory symptoms?

(Please select 1 option)

○	Bronchiectasis
○	Bronchiolitis obliterans and organising pneumonia ('BOOP')
○	Caplan's syndrome
○	Extrinisic allergic alveolitis
○	Pulmonary fibrosis

There are numerous respiratory complications of rheumatoid arthritis.

The commonest is pleural effusion which is often asymptomatic and may resolve spontaneously.

Other pulmonary complications include

Usual interstitial pneumonia
Non-specific interstitial pneumonia

Cryptogenic organising pneumonia
Pulmonary nodules.

Caplan's syndrome is the association of simple coal workers' pneumoconiosis with pulmonary nodules seen on the chest radiograph.

There is a well recognised association between rheumatoid arthritis and bronchiectasis with the reported prevalence in studies using high resolution computed tomography (HRCT) of the chest of between 20-30%.

The HRCT clearly demonstrates bronchiectasis with fluid filled dilated bronchi in the left lower lobe.

[A]

Bronchiectasis is more common in patients with rheumatoid disease who do not smoke in contrast to other pulmonary complications of the disease. It has been suggested that bronchiectasis may be a trigger for the development of rheumatoid arthritis although the exact mechanism for this is unclear.

70/A 69-year-old retired miner was referred to the outpatient clinic with a two month history of persistent productive cough. He also complained of exertional dyspnoea and left sided chest pain.

His appetite had been poor for the preceeding four months and he had lost over one stone in weight. He had retired aged 45 years after working underground for over thirty years. He lived with his wife, drank 10 units of alcohol per week and was an ex-smoker of forty cigarettes a day.

On examination he was apyrexial. Pulse regular at 90 beats per minute, blood pressure 135/75 mmHg. There was no finger clubbing. Chest expansion was reduced on the left with dullness to percussion and diminished breath sounds at the left base and mid-zone. A chest radiograph showed bilateral calcified pleural plaques and a large left pleural effusion.

Which investigation is most likely to yield a diagnosis?

(Please select 1 option)

○	Bronchoscopy
○	Computed tomography
○	Pleural aspiration
○	Pleural biopsy
○	Sputum cytology

This man has bilateral pleural plaques and undoubtably therefore has been exposed to asbestos in the past. In addition he has a left pleural effusion, is a smoker and has symptoms of progressive disease including weight loss.

Although asbestos exposure can cause benign pleural effusions malignancy is the most likely cause of his signs and symptoms, either a bronchial carcinoma or a malignant mesothelioma.

Bronchoscopy is unlikely to be helpful in the absence of a central lesion on radiology.

Computed tomography may identify pleural thickening and other abnormalities and features such as nodular pleural thickening, mediastinal pleural thickening, parietal pleural thickening greater than 1 cm and circumferential pleural thickening are characteristic of malignancy.

The sensitivity of pleural fluid cytology is around 60%. Pleural biopsy however allows histological confirmation of the diagnosis. When done blindly using an Abrams' needle it may increase the diagnostics yield by only 10% over pleural cytology but when undertaken either under CT guidance or thorascopically it has a sensitivity of 90-95%.

[D]

71/A 23-year-old male with asthma presented to the emergency department with acute breathlessness and wheeze following a coryzal illness. He has been treated with high flow oxygen, regular nebulised bronchodilators and 200 milligrams of intravenous hydrocortisone.

On examination he was pale, clammy and unable to record a peak flow reading. His pulse is 140 per min, temperature 37.3°C, and had oxygen saturations of 86% on 15L of oxygen. Auscultation of his chest reveals poor breath sounds bilaterally with a faint polyphonic wheeze.

His arterial blood gas on 15L of oxygen reveals:

pH	7.30	(7.36-7.44)
PO_2	8.0 kPa	(11.3-12.6)
pCO_2	7.8 kPa	(4.7-6.0)
HCO_3	16 mmol/L	(20-28)

What is the most appropriate management for this patient?

(Please select 1 option)

○	Intravenous aminophylline
○	Intravenous antibiotics

	Intravenous magnesium
	Intubation and invasive positive pressure ventilation
	Non-invasive ventilation

Acute severe asthma is still responsible for a large number of deaths in the United Kingdom each year.

Hypercapnia and signs of fatigue are indications for immediate intubation and ventilation.

Although intravenous aminophylline and magnesium may have a place in the treatment of severe asthma, they should not delay intubation when it is indicated.

Non-invasive ventilation is not recommended in acute severe asthma.

[D]

72/A 36-year-old woman presented to the Accident & Emergency department with an episode of loss consciousness whilst out walking her dog.

There was no history of tongue biting or loss of continence. There were no witnesses to the collapse. On direct questioning she admitted to being increasingly breathless on exertion and feeling fatigued. She had no history of respiratory disease but was unable to comment on her family history as she was adopted at birth. Although she took no regular medication she did admit to recreational drug use including ecstasy.

On examination she was mildly cyanosed but was alert and orientated. Her pulse was 90 beats per minute regular, temperature 36.5°C, blood pressure 100/80 mmHg and oxygen saturations 88% on room air. Auscultation of her chest revealed an accentuated second heart sound, a pansystolic murmer at the left sternal edge and an early diastolic murmur at the second left space.

An Electrocardiogram revealed a sinus tachycardia.

What is the most likely diagnosis?

(Please select 1 option)

	Acute Pulmonary Embolism
	Epilepsy
	Hypertrophic Cardiomyopathy
	Mixed Aortic Valve disease

	Pulmonary Arterial Hypertension

The symptoms of pulmonary arterial hypertension include dyspnoea, fatigue, weakness and syncope. Drugs associate with pulmonary arterial hypertension include appetite suppressants and amphetamines. The physical signs of include a left parasternal heave, loud second pulmonary sound, a pansystolic murmur of tricuspid regurgitation and an early diastolic murmur of pulmonary insufficiency. The ECG is not uncommonly normal in cases of pulmonary arterial hypertension.

Although a pulmonary embolism is a possible diagnosis, her symptoms are more suggestive of a chronic condition. Neither epilepsy, hypertrophic cardiomyopathy nor mixed aortic valve disease are consistent with her presentation or clinical signs.

[E]

73/A 35-year-old man presented with a fever and a cough productive of green sputum.

His illness began four days earlier with a headache and general malaise. On the day of presentation he had developed a high fever and had pain in his left side. He was married with two children and worked as an accounts clerk. He was usually fit and well. He was a non-smoker and drank approximately 10 units of alcohol per week.

On examination he was febrile 38.5°C. His blood pressure was 105/65 mmHg, pulse 105 beats per minute and regular, with a respiratory rate of 18 breaths per minute. His heart sounds were normal. Coarse crepitations were heard at the base of the left lung.

Investigations revealed:

Haemoglobin	15.0 g/dL	(13.0-18.0)
White cell count	18.2 $\times 10^9$/L	(4-11)
Neutrophils	12.9 $\times 10^9$/L	(1.5-7)
Platelets	505 $\times 10^9$/L	(150-400)
Serum sodium	139 mmol/L	137-144)
Serum potassium	3.4 mmol/L	(3.5-4.9)
Serum urea	8.7 mmol/L	(2.5-7.5)
Serum creatinine	98 µmol/L	(60-110)

The chest x ray showed left lower lobar consolidation.

Which of the following features is an adverse prognostic marker in this patient?

(Please select 1 option)

	Blood pressure 105/65 mmHg
	Chest x ray showing left lower lobar consolidation
	Respiratory rate of 18 breaths per minute
	Serum urea 8.7 mmol/L
	White cell count more than 18.0 $\times 10^9$/L

The British Thoracic Society (BTS) guidelines on the management of community-acquired pneumoniahighlight several adverse prognostic markers:

Pre-existing adverse prognostic features:

Age 50 years and over
Presence of coexisting disease.

Core clinical adverse prognostic features (CURB):

Confusion: new mental confusion (defined as an abbreviated mental test score of 8 or less)
Urea: raised more than 7 mmol/l (for patients being seen in hospital)
Respiratory rate: raised more than 30/min
Blood pressure: low blood pressure (systolic blood pressure less than 90 mm Hg and/or diastolic blood pressure less than 60 mm Hg).

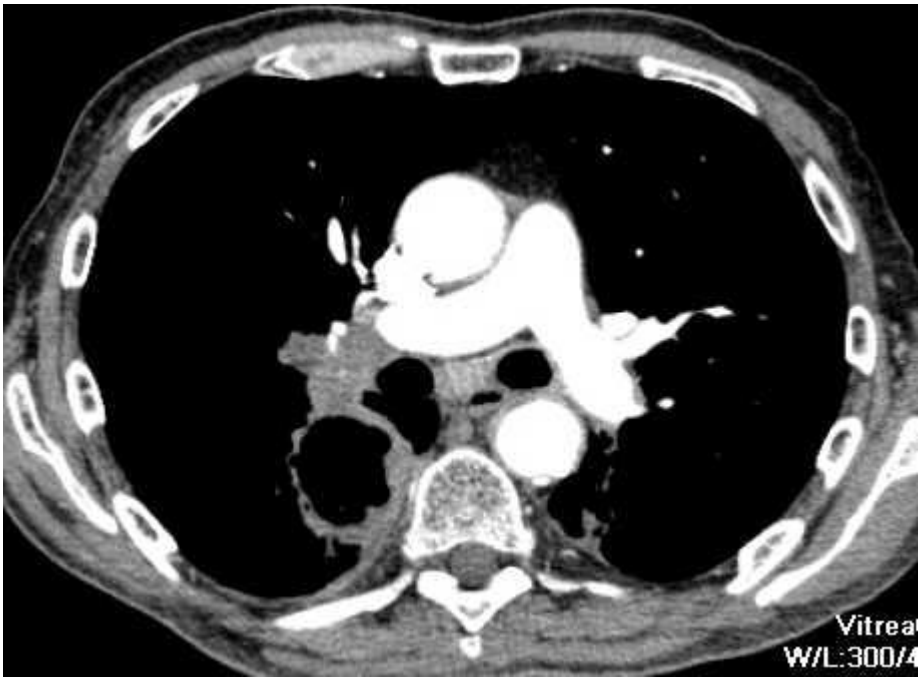
Additional clinical adverse prognostic features:

Hypoxaemia (SaO_2 less than 92% or PaO_2less than 8 kPa) regardless of FiO_2. Oxygen saturation measurements may be available to some general practitioners in the community who have oximeters.
Bilateral or multilobar involvement on the chest radiograph.

Thus, the raised serum urea is the only adverse prognostic marker in this patient.

[D]

74/A 70-year-old is investigated for weight loss.



What is the diagnosis?

(Please select 1 option)

	Aspergilloma
	Cystic bronchiectasis
	Lung carcinoma
	Pneumonic consolidation
	Pulmonary embolus

The diagnosis is a cavitating lung carcinoma.

This is a thick walled irregular cyst with an associated enlarged right hilum responsible for the soft tissue adjacent to the right pulmonary artery.

[C]

75/A 30-year-old pet shop owner presents with a 10 day history of non-productive cough, fever, chills, malaise and lethargy. On questioning he also complains of increasing dyspnoea and sore throat. There is no other relevant medical history of note.

On examination, his pulse is 88 bpm regular, he has a blood pressure of 122/76 mmHg and a respiratory rate of 22/min. He has a temperature of 37.6°C. He has a mild pharyngitis together with bilateral basal crepitations on auscultation of his chest. Abdominal palpation reveals a two finger breadths splenomegaly.

Investigations reveal:

Full blood count	Normal
U+Es	Normal
Glucose	5.6 mmol/L (3.0-6.0)
ESR	95 mm/hr (<10)

The chest x ray shows left lower lobe consolidation.

What is the most probable diagnosis?

(Please select 1 option)

○	Allergic bronchopulmonary aspergillosis
○	Extrinsic allergic alveolitis
○	Legionellosis
○	Psittacosis
○	Streptococcal pneumonia

The patient is a pet shop worker with features of an atypical pneumonia.

All the mentioned features are typical of psittacosis although other causes of atypical pneumonias cannot be excluded completely from the information provided.

[D]

76/A 25-year-old gentleman, a known case of asthma, presents to the Emergency Department with an acute exacerbation of asthma.

He is usually well controlled on inhaled fluticasone BD and salbutamol inhaler as required.

The patient admits to having been getting progressively short of breath over the last few days, requiring several doses of inhaled salbutamol per day. He is distressed at rest with a respiratory rate of 26 breaths per minute and he is unable to complete sentences in one breath. Examination reveals bilateral expiratory polyphonic wheezes and poor air entry.

Arterial blood gases on air show:

$PaCO_2$	3.6 kPa/27 mmHg	(4.7-6.0)

PaO_2	10 kPa/75 mmHg	(11.3-12.6)
pH	7.45	(7.36-7.44)
Standard bicarbonate	24 mmol/l	(20-28)

The casualty officer starts the patient on high flow oxygen and continuous nebulised salbutamol. He also gives the patient 200 mg of hydrocortisone IV and one dose of nebulised ipratropium bromide. No improvement is seen after 30 minutes of treatment.

Which of the following treatments would be most appropriate at this stage?

(Please select 1 option)

	Helium/oxygen mixture
	Intubation and ventilation
	IV aminophylline
	IV magnesium sulphate
	IV salbutamol

The diagnosis is acute severe asthma (according to the British Thoracic Society Guidelines).

Of all the mentioned treatments the one that is most appropriate is magnesium sulphate.

"In acute asthma, IV aminophylline is not likely to result in any additional bronchodilation compared to standard care with inhaled bronchodilators and steroids." *BTS Guidelines on the Management of Asthma*.

Intubation is not indicated at this stage, though it is to be considered if features of respiratory failure develop.

[D]

77/An 81-year-old man was admitted to hospital from a residential home after developing chest pain. He had a past history of ischaemic heart disease, having suffered a myocardial infarction eight years previously. Following his previous infarction, he had developed congestive cardiac failure which was well controlled with medication.

He had no other past history of note, apart from an injury during his time in the armed services sixty years previously when he sustained a chest injury when he was hit by shrapnel while serving in North Africa. His medications included aspirin, ramipril and

furosemide. He had been born and raised in India, but had lived in the United Kingdom for all of his adult life, where he had worked as a secondary school teacher and latterly a headmaster of a boys' boarding school.

His ECG showed lateral ST segment elevation and his serum troponin T was elevated. His chest x ray showed pleural calcification at the right base.

Select the two most likely causes of pleural calcification in this patient:

(Please select 2 options)

☐	Adenocarcinoma of the bronchus
☐	Chickenpox
☐	Childhood measles
☐	Chronic congestive cardiac failure
☐	Mesothelioma
☐	Occupational exposure to chalk dust
☐	Post-traumatic
☐	Primary hyperparathyroidism
☐	Tuberculosis
☐	Yellow nail syndrome

Unilateral pleural calcification most commonly occurs as a chronic change secondary to pleural infection (particularly tuberculous empyema), pyogenic empyema, or haemothorax. Unilateral pleural calcification rarely is related to asbestos exposure.

Bilateral calcified pleural plaques are usually considered asbestos-related.

Other rarer causes of bilateral disease include radiation exposure, hyperparathyroidism, pulmonary infarction, and pancreatitis.

The most likely explanation for pleural calcification in this patient is either old TB empyema or as a result of haemothorax from his war injury.

[G][I]

78/A 32-year-old man was referred to clinic with a ten year history of recurrent chest infections. He was diagnosed with common variable immunodeficiency.

Laboratory investigations showed:

IgG	6.5 g/l	(6.0-13.0)
IgA	0.8 g/l	(0.8-3.0)
IgM	0.5 g/l	(0.4-2.5)

Which of the following options would best prevent recurrent chest infections?

(Please select 1 option)

○	Chest physiotherapy and postural drainage
○	Heart-lung transplantation
○	Intermittent courses of antibiotics when the patient feels he is developing a chest infection
○	Intravenous immunoglobulin
○	Lifelong prophylactic antibiotics

Common variable immunodeficiency (CVID) is the most prevalent of the primary immunodeficiency diseases. The basic defect in CVID is a failure of B lymphocyte differentiation into plasma cells that produce the various immunoglobulin (Ig) subtypes.

Patients with CVID often have marked reduction in serum levels of both IgG and IgA; approximately 50% of these patients also have reduced IgM. However, some patients have normal IgG levels but functional antibody deficiency may be present despite normal IgG subclass levels.

The diagnosis is based on exclusion of known causes of defects of the humoral immune system. Most cases are sporadic, although familial cases exist that have various inheritance modes (including autosomal dominant with variable penetrance, autosomal recessive, and X linked).

Regular intravenous immunoglobulin (IgG) infusions greatly reduce the frequency of recurrent respiratory tract infections.

Clinical manifestations of CVID include:

Recurrent infections:

Principally recurrent pyogenic infections of upper and lower respiratory tract. This is the main clinical manifestation of CVID. Symptoms may appear during childhood or, more often, after adolescence. Bronchiectasis may develop if optimal therapy is delayed. *Haemophilus influenzae*,*Moraxella catarrhalis*, *Streptococcus pneumoniae*, and *Staphylococcus aureus* are the organisms most commonly involved. Severe, recurrent infection with herpes simplex is also common.

Autoimmune diseases:

Patients may develop

Rheumatoid arthritis
Haemolytic anaemia
Thrombocytopenia
Neutropenia
Thyroid abnormalities
Vitiligo
Keratoconjunctivitis sicca.

About 20% of these patients have a severe gastroenteropathy with severe malabsorption resembling coeliac sprue, nodular lymphoid hyperplasia, and chronic inflammatory bowel disease such as ulcerative colitis and Crohn's disease. A smaller number of patients develop

Achlorhydria and pernicious anaemia
Autoimmune hepatitis
Primary biliary cirrhosis
Alopecia totalis
Hyperthyroidism
Vasculitis
Lymphoid interstitial pneumonia.

Lymphoid hyperplasia and granulomatous diseases:

Atypical lymphoid hyperplasia due to clonal expansion of B or T lymphocytes has been reported in as many as one third of patients with CVID. Granulomas have been reported in approximately 5-10% of patients with CVID. Granulomas are indistinguishable from those of classic sarcoidosis and are found in the

Lung
Liver
Spleen
Conjunctivae.

Predisposition to malignancy:

Patients with CVID have a high risk of developing malignant neoplasms (most commonly lymphoma, especially non-Hodgkin's lymphoma [NHL], and gastrointestinal [GI] carcinoma).

Most lymphomas are of these are of the B cell immunophenotype and are frequently associated with Epstein-Barr virus (EBV). Lymphoma occurs 300 times more frequently in women with CVID than in affected men.

Other malignancies include

Colon cancer
Breast cancer
Prostate cancer
Ovarian cancer
Oral cancer

Melanoma.

[D]

79/A 21-year-old man was admitted to hospital following the onset of sharp, left-sided chest pain and breathlessness.

On examination he was mildly breathless at rest with pulse 100 beats per minute and regular, and blood pressure 125/60 mmHg. His chest x ray showed a left pneumothorax with a 4 cm rim of air visible around the left lung. His oxygen saturation on air was 98%.

What is the most appropriate management?

(Please select 1 option)

○	Admit and repeat chest x ray in 24 hours
○	Discharge from hospital
○	Insertion of intercostal chest drain
○	Needle aspiration
○	Oxygen 24% FiO2

British Thoracic Society guidelines state that in patients with no co-existing lung disease who present with a spontaneous pneumothorax of more than 2 cm and who are mildly symptomatic, the initial management should be needle aspiration.

Young, fit individuals with a pneumothorax of less than 2 cm and who are minimally symptomatic could be discharged home.

If a patient with a pneumothorax requires oxygen, this should be given at 10 L/min.

Intercostal chest drain insertion is reserved for those who fail aspiration, have evidence of tension pneumothorax, or in those who are more severely symptomatic with a history of pre-existing lung disease.

[D]

80/A 26-year-old man was referred to the emergency unit by his general practitioner with a four hour history of exertional dyspnoea.

He experienced undue breathlessness on climbing three flights of stairs associated with mild chest discomfort. The discomfort and breathlessness eased within a few minutes of rest, but recurred when he continued to climb the stairs.

On examination, he appeared well. His pulse was 80 beats per minute in sinus rhythm with blood pressure 120/65 mmHg. On auscultation of his chest there was a loud clicking sound at the lower left sternal border that was synchronised with his heartbeat.

What is the most likely diagnosis?

(Please select 1 option)

○	Acute non-Q wave myocardial infection
○	Mitral valve prolapse
○	Pericarditis
○	Rib fracture
○	Spontaneous pneumothorax

A clicking noise that is synchronised with the heart beat is a less common, but well-recognised finding in patients with spontaneous pneumothorax.

It appears to be commoner in patients with small left-sided pneumothoraces.

The sound is different from the crunching noise that occurs in pneumomediastinum (Hamman's sign).

[E]

81/A 43-year-old man who is known to smoke 20 cigarettes a day for the last 20 years is admitted with a three day history of increasing breathlessness.

He has an associated cough productive of purulent sputum and has noticed some associated left sided chest pain. His appetite has been poor over the last 48 hours and as a result he admits to not drinking or eating very much over this time.

He has a history of hypertension for which he currently taking bendroflumethiazide 2.5 mg once daily. His father died of bronchial carcinoma at the age of 68 years. He works as a plumber. He drinks four pints of beer a night.

On examination he had a temperature of 38.5°C. Respiratory rate 26 breaths per minute. There was an area of bronchial breathing at the left base with associated coarse crackles.

Investigations revealed:

Haemoglobin	15.8 g/dL	(13.0-18.0)

White cell count	3.8 $\times 10^9$/L	(4-11)
Platelets	100 $\times 10^9$/L	(150-400)
Serum sodium	142 mmol/L	(137-144)
Serum potassium	4.2 mmol/L	(3.5-4.9)
Serum urea	14.8 mmol/L	(2.5-7.5)
Serum creatinine	160 µmol/L	(60-110)

The chest x ray showed an area of dense consolidation in the left lower zone.

Which one of the following is not associated with a poorer prognosis in community acquired pneumonia?

(Please select 1 option)

○	Multilobar involvement on chest x ray
○	Platelet count of less than 100 $\times 10^9$/L
○	Serum creatinine of greater than 400 µmol/L
○	Temperature greater than 40°C
○	Total white cell count of less than 4 $\times 10^9$/L

There are numerous predictors of increased severity and risk of death from community acquired pneumonia including

White cell count less than 4 $\times 10^9$/L or greater than 20 $\times 10^9$/L
Co-morbidity such as renal disease
Multi-lobar involvement on CXR and
Temperature less than 35°C or more than 40°C.

These have been incorporated into severity scores such as the pneumonia severity index (PSI) (1), Modified American Thoracic Society rule (2) and the CURB-65 score (3).

The CURB-65 score is easily used in practise and is derived from:

C	Confusion
U	Urea > 7 mmol/L
R	Respiratory rate >30/min
B	Blood pressure <60 mmHg diastolic or <90 mmHg systoloic

65	Age >65

[B]

82/ A 47-year-old lady was referred to outpatients with a four week history of cough productive of sputum and breathlessness.

The cough had become so severe that she was producing 500 ml of frothy mucoid sputum per day. She had noticed a reduction in appetite but no weight loss.

What is the most likely diagnosis ?

(Please select 1 option)

○	Bronchoalveolar cell carcinoma
○	Carcinoid tumour
○	Langerhan's histiocytosis X
○	Lymphangitis carcinomatosa
○	Small cell carcinoma

Bronchoalveolar cell carcinoma classically presents with progressive breathlessness and the production of large amounts of sputum (bronchorrhoea). They account for up to 1% of all bronchial carcinomas.

The tumour spreads using the alveolar walls as a frame and the alveoli are often filled with mucin.

The differential diagnosis includes heart failure, persistent pneumonia, alveolar proteinosis and cryptogenic organising pneumonia.

Pulmonary Langerhan's cell histiocytosis X and lymphangitis carinomatosis can all present with progressive breathlesness and cough but bronchorrhoea is not a feature.

[A]

83/A drowsy 23-year-old student is brought in to the casualty department by her friend. The patient lives alone in a one bedroom flat. She is normally fit and well although had recently been complaining of difficulty concentrating in lectures.

She smokes 20 cigarettes a day. She was on no medication and had no previous medical history of note. She had vomited.

On examination she was flushed. She had a bounding pulse of 120 beats per minute. Her blood pressure was 180/100 mmHg. Oxygen saturations were normal.

Initial investigations showed:

Haemoglobin	12.8 g/dL	(11.5-16.5)
White cell count	10.5 $\times 10^9$/L	(4-11)
Platelets	280 $\times 10^9$/L	(150-400)
Serum sodium	134 mmol/L	(137-144)
Serum potassium	3.6 mmol/L	(3.5-4.9)
Serum urea	7.4 mmol/L	(2.5-7.5)
Serum creatinine	80 µmol/L	(60-110)
Drug screen	Negative	

Arterial blood gases on air:

pO_2	8.6 kPa	(11.3-12.6)
pCO_2	4.7 kPa	(4.7-6.0)
pH	7.42	(7.36-7.44)

The chest x ray was normal.

What investigation would confirm the diagnosis?

(Please select 1 option)

○	Blood glucose
○	Blood lactate
○	Carboxy haemoglobin
○	Electroencephalogram
○	Lumbar puncture

This girl has classical features of carbon monoxide poisoning.

Carbon monoxide binds with haemoglobin with a greater affinity than oxygen displacing it from the blood causing tissue hypoxia. In addition carbon monoxide shifts the oxygen dissociation curve to the left reducing tissue delivery even more.

Symptoms of mild poisoning (carboxy haemoglobin levels = 10-30%) are headache, tiredness, nausea, dizziness and poor concentration. With increasing levels vomiting and weakness then impaired consciousness may occur with hypertension, tachycardia and flushing.

With severe poisoning (carboxy haemoglobin levels more than 50%) convulsions, coma, respiratory depression and death can occur.

Treatment is with 100% oxygen through a tight fitting, non-re-breathing face mask at a flow rate of 10 L/min.

In severe cases intubation and mechanical ventilation may be required and in these patients there is a place for hyperbaric oxygen.

[C]

84/A 67-year-old man presents with a two month history of persistent cough. The cough is always unproductive and there has been no haemoptysis. It never wakes him from his sleep.

He has no chest pain but has noticed some mild breathlessness on exertion. He is a smoker of 20 cigarettes a day since the age of 17. He has lost a small amount of weight, and has a reduced appetite. He has been constipated over the last two weeks.

Examination was unremarkable. He has no clubbing nor lymphadenopathy. Examining his chest, breath sounds were vesicular with no added sounds.

Investigations showed:

Haemoglobin	12.6 g/dL (13.0-18.0)
White cell count	10.5 $x10^9$/L (4-11 $x10^9$)
Platelets	160 $x10^9$/L (150-400 $x10^9$)
Serum sodium	149 mmol/L (137-144)
Serum potassium	4.4 mmol/L (3.5-4.9)
Serum urea	9.4 mmol/L (2.5-7.5)
Serum creatinine	120 µmol/L (60-110)
Serum corrected calcium	3.26 mmol/L (2.2-2.6)

The chest x ray showed right hilar lymphadenopathy.

What is the next investigation of choice?

(Please select 1 option)

	Bone marrow biopsy

	Bronchoscopy
	CT chest
	Isotope bone scan
	Kveim test

The differential diagnoses of this man's hilar lymphadenopathy and hypercalcaemia include bronchial carcinoma, sarcoidosis and lymphoma.

The most likely diagnosis is squamous cell bronchial carcinoma. Hilar involvement is usually a result of metastatic spread to the hilar nodes.

CT chest is the next investigation of choice as it will help identify any further nodal involvement and allow planning of an approach to histological confirmation of the tumour.

[C]

85/A 65-year-old lady is admitted as an emergency after a choking episode.

She is breathless and has a temperature of 37.8°C. She is a smoker of 20 cigarettes a day, but has no history of previous respiratory disease; she had a stroke six months previously. She was started on oral co-amoxiclav by her general practitioner.

A chest x ray showed a homogenous opacity at the right base with the right hilum pulled downwards.

What is the next investigation of choice?

(Please select 1 option)

	Blood cultures
	Bronchoscopy
	CT chest
	Sputum culture
	Ventilation/perfusion scan

This lady's clinical history suggests aspiration.

Her chest x ray suggests collapse +/- consolidation of the right lower lobe.

The lower lobes are the usual site of aspiration when the patient is upright. She may therefore have aspirated a foreign body during the choking episode and a bronchoscopy will identify this and allow its removal.

[B]

86/A 62-year-old lady was seen in the outpatient department with a six month history of increasing breathlessness on exertion.

Her exercise tolerance was limited to 80 metres on the flat and she had started to become breathless walking up the stairs in her house. She slept with four pillows and had noticed some swelling of both her ankles. She had a cough which was occasionally productive of sputum. She had given up smoking when she first noticed her dyspnoea and had a 40 pack year smoking history.

She was known to have hypertension and ischaemic heart disease and had coronary artery stenting 12 months ago.

Pulmonary funtion testing revealed:

FEV_1	0.82 L	(1.80 - 3.02 predicted)
FVC	1.84 L	(2.16 - 3.58 predicted)
Diffusion capacity	2.40 mmol/min/kPa	(5.91 - 9.65 predicted)
Total lung capacity	4.40 L	(4.25 - 6.22 predicted)
Residual volume	2.69 L	(1.46 - 2.48 predicted)

What is the diagnosis?

(Please select 1 option)

○	Asthma
○	Chronic obstructive pulmonary disease
○	Cryptogenic fibrosing alveolitis
○	Left ventricular failure
○	Sarcoidosis

This lady has a history consistent with chronic obstructive pulmonary disease (COPD).

The diagnosis is confirmed on lung function; she has airways obstruction with a reduced forced expiratory volume in one second ($FEV_{1)}$ and FEV_1/forced vital capacity (FVC).

She has a normal total lung capacity but her residual volume is increased indicating a degree of air trapping.

Finally her diffusion capacity (transfer factor) is decreased which helps differentiate COPD from asthma.[B]

87/A 67-year-old man was referred to the outpatient department with a four month history of increasing cough and dyspnoea.

He coughed most mornings bringing up mucoid sputum. He denied any haemoptysis. In addition he had become increasingly breathless on exertion so that his exercise tolerance was reduced to 100 metres on the flat.

He denied any chest pain but did have pain in his right shoulder that was constant and had started to keep him awake at night over the last couple of months and more recently pain of the medial aspect of his right arm. He was known to have rheumatoid arthritis. His appetite was reduced and he had lost 5 kg in weight. He had a forty pack year smoking history. He was a retired engineer.

On examination there was nicotine staining of the fingers. He had signs of rheumatoid arthritis in his hands. There was bilateral finger clubbing. He had wasting of small muscles of his right hand particularly the thenar and hypothenar eminences.

What is the diagnositic test of choice?

(Please select 1 option)

○	Bronchoscopy
○	CT scan of chest
○	Isotope bone scan
○	Nerve conduction studies
○	Sputum cytology

This patient has a Pancoast or superior sulcus tumour.

The tumour arises in the apex of the lung and infiltrates locally into the brachial plexus, ribs and mediastinum. Patients often have signs of local extension on presentation such as

Neurological signs in the arm and hand (C8,T1 distribution)
Ipsilateral Horner's syndrome or
Radiological evidence of rib destruction.

CT scan is the investigation of choice.

It can often be difficult to get biopsy material as approaches in this area are difficult.

Bronchoscopy is unhelpful as lesions are too peripheral.

Tumours are most commonly squamous cell and are usually inoperable on presentation.[B]

88/A 65-year-old man is referred to the respiratory clinic by his general practitioner. The patient gives a three month history of a productive cough and steady weight loss of 10 kg. His sputum had initially been yellow, but over the past week he has noticed streaks of blood. He also complains of sweats at night and sometimes has had to change his bedclothes.

He has a longstanding history of bronchial asthma that has required admission to hospital on several occasions. On two occasions he has been admitted in respiratory distress and has required intubation and ventilation. His current therapy includes salbutamol and beclomethasone inhalers and prednisolone 5 mg once daily which he has taken for several years. He also has a home nebuliser which he uses as required.

His chest x ray in clinic shows right apical consolidation. The last chest radiograph, taken six months previously on admission to hospital, had been normal. A sputum sample proves to be positive for acid-alcohol fast bacilli. A presumptive diagnosis of pulmonary tuberculosis is made and he is started on antituberculous therapy.

Five days later he presents to casualty with breathlessness and wheeze. On examination he is afebrile. Widespread expiratory wheezes are heard throughout his chest. His peak expiratory flow rate (PEFR) is measured at 110 l/min. His chest x ray does not show any significant change from the last film taken in chest clinic.

Which of the following options would be the most appropriate step in his management?

(Please select 1 option)

○	Continue anti-tuberculous therapy and increase steroid dose
○	Continue anti-tuberculous therapy and reduce steroid dose
○	Continue anti-tuberculous therapy and steroids at current dose
○	Stop anti-tuberculous therapy and continue steroids at current dose
○	Stop anti-tuberculous therapy and steroids and start broad-spectrum antibiotics

The metabolism of corticosteroids is increased by rifampicin.

Patients on long term steroids should have their dose of steroids increased when starting antituberculous therapy.

[A]

89/A 22-year-old woman presents with recurrent epidodes of breathlessness and cough productive of foul smelling sputum.

She has a past history of recurrent episodes of colicky abdominal pain for the last four years.

Investigations show:

Sputum culture	Heavy growth of *Pseudomonas aeruginosa* and*Haemophilus influenzae*
CXR	Tramline and ring shadows

What is the most likely explanation for her symptoms?

(Please select 1 option)

○	Bronchiectasis
○	Chronic granulomatous disease
○	Cystic fibrosis
○	Hypogammaglobulinaemia
○	Primary ciliary dyskinesia

Cystic fibrosis (CF) usually presents in childhood, but it is not unusual for some cases to present in early adult life.

CF presenting in this fashion occurs frequently in the Part 2 examination.

Pseudomonas is never a pathogen in healthy lungs, it is the cause of the foul smelling sputum and implies that there is underlying bronchiectasis.

Similarly, *H. influenzae* is typically a pathogen in chronic lung disease.

Several of the alternative options may be associated with bronchiectasis, but none adequately explains the history of abdominal pain, which is likely to be due to distal intestinal obstruction syndrome (sludging of intestinal contents leading to intestinal obstruction by faecal impaction or intussusception).

Chronic granulomatous disease (CGD) is an inherited disorder of phagocyte cells, caused by an inability of neutrophils to undergo the oxidative burst necessary to kill some bacteria and fungi. This leads to recurrent life-threatening bacterial and fungal infections.

Fungal pneumonias, particularly those caused by*Aspergillus* species are common. Most patients (75%) present within the first five years of life.

Pseudomonas aeruginosa is a rare pathogen in CGD because CGD neutrophils are able to kill *P. aeruginosa*organisms by nonoxidative mechanisms.

T cell deficiencies (such as loss of CD4 T lymphocytes in HIV/AIDS)are typically associated with viral and fungal pathogens.

Patients with hypogammaglobulinaemia are susceptible to recurrent upper and lower respiratory tract infections (otitis media, sinusitis, bronchitis, bronchiectasis, pneumonia).

Encapsulated bacteria (for example, *Streptococcus pneumoniae*, *Streptococcus pyogenes*, *Haemophilus influenzae*, *Staphylococcus aureus*) are the most common pathogens.

Primary ciliary dyskinesia is associated with recurrent upper and lower respiratory tract infections due to inability to clear secretions effectively. Patients are usually infertile.

[C]

90/The orthopaedic surgeons request a medical opinion on a 77-year-old man who is at post-operative day 10 following a total hip replacement. The patient's operation had been uncomplicated and he had been well in the post-operative period to date.

On the day of referral he had suddenly developed left-sided chest pain and shortness of breath. On examination his pulse was 120/minute and his respiratory rate 22/minute. His chest was clear to auscultation and his heart sounds were normal. His ECG showed left bundle branch block (LBBB).

Arterial blood gases on air:

pH	7.44	(7.36-7.44)
pO_2	9.5 kPa	(11.3-12.6)
pCO_2	3.4 kPa	(4.7-6.0)

What is the most likely diagnosis?

(Please select 1 option)

○	Acute left ventricular failure (LVF)
○	Acute myocardial infarction (AMI)
○	Fat embolism
○	Hospital-acquired pneumonia
○	Pulmonary embolism (PE)

This is a common clinical conundrum.

Despite an ECG showing LBBB, PE is the likeliest explanation for this patient's chest pain and dyspnoea. Being post-operative following a total hip replacement there is a significant risk of PE. This is backed up by the blood gases which show hypoxia and hypocapnoea (that is, he is unable to oxygenate despite good ventilation).

There are no clinical signs of either LVF or pneumonia, and the gases are not compatible with either of these options.

Fat embolus is a risk intra-operatively or in the immediate post op period, but is much less likely 10 days afterwards.

The presence of LBBB raises the possibility of MI, but it is not clear whether this conduction defect is new or old. The gases are not in keeping with an AMI, so you have to say that PE is the most likely explanation.

[E]

91/A 19-year-old lady developed increasing breathlessness and hypoxia two days after admission with severe burns. She had no previous history of note.

Her father had had a myocardial infarction at the age of 40.

She was a non-smoker. On examination she had a respiratory rate of 26 breaths per minute and a pulse rate of 110 beats per minute. On listening to her chest there were crackles over both lung fields. A chest x ray revealed bilateral hazy shadowing.

What is the most likely diagnosis?

(Please select 1 option)

○	Adult respiratory distress syndrome
○	Chemical pneumonitis
○	Left ventricular failure (LVF)
○	Nosocomial pneumonia
○	Pulmonary haemorrhage

This lady is likely to have developed adult respiratory distress syndrome (ARDS) as a complication of her severe burns.

The patient becomes tachypnoeic, increasingly breathless and cyanosed and develops refractory hypoxia.

The CXR classically shows bilateral peripheral interstitial and alveolar infiltrates that become progressively more confluent but spare the costophrenic angles.

Normal heart size
Absent septal lines
Air bronchograms and

A peripheral distribution

are helpful in differentiating ARDS from other conditions such as LVF

[A]

92/A 78-year-old lady is admitted with increasing breathlessness and a cough productive of mucoid sputum on most days. She has had exertional dyspnoea for two years but she has become more breathless over the last five days. She was a smoker of 40 cigarettes per day until three months ago. She takes Beclasone 200 mg BD, salmeterol 50 mg BD and Ventolin PRN.

On examination she was obese with a BMI of 32 kg/m^2. She was cyanosed and pale but there was no clubbing nor lymphadenopathy. She was breathless at rest with a respiratory rate of 24/min. Pulse was 110 beats per minute and blood pressure 140/80 mmHg. Her chest was hyperinflated with expiratory wheezes and she had bilateral swollen ankles.

She was treated with nebulised bronchodilators, controlled oxygen therapy, oral prednisolone, antibiotics and commenced on diuretic therapy. She improved and was discharged home five days later.

On review six weeks later investigations revealed the following:

Arterial blood gas analysis (on air)

PaO_2	6.9 kPa	(11.3-12.6)
$PaCO_2$	6.8 kPa	(4.7-6.0)
pH	7.4	(7.36-7.44)

Pulmonary function testing

FEV_1	0.9 l	(3.2 predicted)
FVC	4.2 l	(4.5 predicted)

Which one of the following is the primary indication for long term domiciliary oxygen therapy in this patient?

(Please select 1 option)

○	Cor pulmonale
○	Inability to get out of the house

	Low FEV_1
	Low PaO_2
	Raised $PaCO_2$

This patient has chronic obstructive pulmonary disease (COPD) as a result of her smoking. Her investigations demonstrate that it is severe (FEV_1 less than 40% predicted).

The reason why she is a candidate for long term oxygen therapy (LTOT) is that she is hypoxic. The criteria for LTOT are PaO_2 less than 7.3 kPa (55 mmHg) with or without hypercapnia or PaO_2 less than 8.0 kPa (60 mmHg) if there is evidence of pulmonary hypertension/cor pulmonale/polycythaemia.

LTOT and smoking cessation are currently the only interventions in COPD that have been shown to prolong life.

[D]

93/A 74-year-old man presented to his general practitioner with a chronic cough.

He smokes 10 cigarettes per day starting at the age of 14. The GP had organised a chest x ray which was reported as showing calcification on both hemidiaphragms and clear lung fields. He was therefore referred to the outpatients' department.

An occupational history revealed that he had worked in a shipyard for about eight years fifty years previously. He had no previous medical history of note. Systems review was negative apart from some intermittent heartburn and nocturia. In particular his appetite was normal and there had been no weight loss. He lived alone but kept pigeons.

On examination his pulse was 74 beats per minute, blood pressure 155/75 mmHg and there were no abnormalities found on respiratory examination.

Which one of the following statements is correct?

(Please select 1 option)

	He is at high risk of developing asbestosis
	He is at high risk of developing mesothelioma
	It is likely that the x ray abnormality is related to his previous occupation
	The x ray abnormality is the cause of his cough

This patient's CXR shows calcification on both hemidiaphragms which are most likely to be pleural plaques from previous asbestos exposure.

He would have been exposed to asbestos in his job in the ship yards. Pleural plaques are benign and simply a marker of previous asbestos exposure and therefore are common in anyone who has been exposed to asbestos in the past. They rarely cause symptoms and are therefore unlikely to be the cause of his cough. They require no long term follow up.

He is unlikely to develop asbestosis 50 years after his last exposure to asbestos, most patients developing disease within 20 years of exposure.

Similarly, the average latent period from exposure to diagnosis of mesothelioma is 20 years. It is as a result of this that the incidence of mesothelioma continues to rise and is expected to peak in 2020 at around 1300 cases/year in the United Kingdom.

[C]

94/A 73-year-old lady presented with lethargy and weight loss of one stone over the last four months. She had had an irritating cough and repeated haemoptysis. She had been a smoker of 20 cigarettes a day but gave up four months previously when her symptoms began. She had been treated for pulmonary tuberculosis 15 years previously. She was not sure what medication she was given and for how long.

Investigations revealed:

Haemoglobin	12.0 g/dL	(11.5-16.5)
White cell count	4.1 $\times 10^9$/L	(4-11 $\times 10^9$)
Platelets	140 $\times 10^9$/L	(150-400 $\times 10^9$)
Serum sodium	134 mmol/L	(137-144)
Serum potassium	3.7 mmol/L	(3.5-4.9)
Serum urea	8.5 mmol/L	(2.5-7.5)
***Aspergillus fumigatus* precipitins**	Positive	
RAST	Negative to *Aspergillus*	

Her chest x ray showed a solid lesion at the left lung apex.

What is the most likely diagnosis?

(Please select 1 option)

○	Allergic bronchopulmonary aspergillosis
○	Aspergilloma
○	Bronchial carcinoma

	Invasive aspergillosis
	Reactivation of tuberculosis

The lesion in the left apex is most likely to be an aspergilloma which has developed in an old tuberculous cavity. Aspergillomas are often asymptomatic but can result in haemoptysis in up to three quarters of patients. In some patients haemoptysis can be massive and fatal. Systemic symptoms of weight loss, lethargy and fever are less common. The CXR appearances are of a solid opacity within a cavity often associated with a rim of air. Thses features are seen more clearly on computed tomography. Precipitating antibodies help to confirm the diagnosis and are present in 95% of cases.

[B]

95/A 67-year-old lady presented with a seven month history of exertional breathlessness and cough. She had a reduced appetite and had lost half a stone in weight. She had no previous history of note.

She had worked as a hairdresser. She kept a cat at home. She lived alone but had been coping well until now. She smoked 20 cigarettes a day.

On examination she was clubbed and cyanosed. She was pale. Pulse rate was 80 beats per minute. BP was 138/80 mmHg. Heart sounds were normal. There were bilateral fine inspiratory crackles heard at the lung bases.

Investigations revealed:

FEV1	2.8 l	(3.6 predicted)
FVC	3.1 l	(4.5 predicted)
Diffusion capacity	5.1 mmol/min/kPa	(NR 6.3-11.9)

The chest x ray showed slight increase in basal lung markings.

What is the most likely diagnosis?

(Please select 1 option)

	Bronchiectasis
	Fibrosing alveolitis
	Left ventricular failure (LVF)
	Lymphangitis carcinomatosis

	Sarcoidosis

The history and examination findings are suggestive of interstitial lung disease (ILD).

The pulmonary function tests demonstrate a reduction in both FEV_1 and FVC (FEV_1/FVC = 90%) with a low diffusion capacity, that is, restrictive defect which is consistent with ILD. The most likely diagnosis is CFA (or usual interstitial pneumonia).

Bronchiectasis usually results in a productive cough and an obstructive pattern on lung function.

Sarcoidosis usually affects the mid zones or is distributed more uniformly throughout the lungs.

The history and CXR findings are not suggestive of LVF and lymphangitis carcinomatosis typically produces hilar enlargement with diffuse streaky mid zone infiltrates.

The diagnosis can be confirmed on high-resolution computed tomography HRCT in most cases although lung biopsy may be required.

[B]

96/A 22-year-old lady was planning to emigrate to Australia and had a chest x ray undertaken as part of her visa requirements.

This demonstrated bilateral hilar lymphadenopathy but clear lung fields. It was reported as being suspicious of sarcoidosis. She was referred to the outpatients clinic.

On systems review in outpatients a two month history of arthralgia and a dry cough was elicited. She had no previous history of note but three months ago she had had unprotected intercourse and took an HIV test which was negative. She had no other symptoms. She lived in Zimbabwe until she was 16. She worked as a waitress.

Which one investigation is most likely to confirm the diagnosis?

(Please select 1 option)

	Bronchoalveolar lavage
	High-resolution (HR) CT scan of chest
	Serum angiotensin converting enzyme (ACE) activity
	Transbronchial lung biopsy
	Tuberculin test

A HRCT scan of chest may reveal pulmonary abnormalities in sarcoidosis, such as multiple ill defined opacities running along the bronchovascular bundles, lymphatics and interlobar septa even in the absence of plain CXR abnormalities.

Serum ACE is elevated in about 70% of patients with active sarcoidosis but is not sensitive or specific and is therefore not helpful in diagnosis.

Histological confirmation is required to make the diagnosis with confidence.

Transbronchial lung biopsy will provide positive histology in about 80% of patients, is safe and can be done under sedation with local anaesthesia and is therefore the diagnostic investigation of choice.

[D]

97/A 65-year-old man presented with a four week history of pleuritic chest pain associated with shortness of breath and dry cough. He also reported weight loss of nearly 10 kg in the past six months.

He had a past history of myocardial infarction 20 years earlier from which he had made a good recovery. He did not suffer from any exertional chest pain subsequently.

He lived alone and had not seen his general practitioner for two years. He seldom saw his general practitioner, but had attended the surgery twice recently with mild recurrent pain in his left knee that responded well to treatment with simple analgesia. He was an ex-smoker of 15 cigarettes per day, having given up smoking 10 years previously. His only medication was aspirin.

On examination of his chest he had reduced expansion, dull percussion note and decreased breath sounds on the right. A chest x ray confirmed a right-sided pleural effusion.

Analysis of a pleural aspirate revealed:

Pleural fluid protein content	42 g/L
Pleural fluid glucose	2.0 mmol/L

What is the likely diagnosis?

(Please select 1 option)

○	Bronchial carcinoma
○	Cardiac failure
○	Mesothelioma

	Rheumatoid arthritis
	Tuberculosis

The pleural fluid protein is greater than 35 g/l which demonstrates it is an exudate and effectively excludes cardiac failure.

If pleural fluid protein is 25-35 g/l then Light's criteria are more accurate in determining whether the effusion is an exudate or transudate. It is an exudate if one or more of the following criteria are met

1. Pleural fluid protein divided by serum protein greater than 0.5
2. Pleural fluid lactate dehydrogenase (LDH) divided by serum LDH greater than 0.6
3. Pleural fluid LDH greater than 2/3rds upper limits of normal serum LDH.

The pleural glucose level is low.

Levels less than 3.3 mmol/l are found in

Empyema
Rheumatoid arthritis
Lupus
Malignancy
Oesophageal rupture and
Tuberculosis.

The lowest levels are found in rheumatoid effusions and empyema with pleural glucose in rheumatoid effusions rarely being above 1.6 mmol/l.

The most likely diagnosis would be malignancy and appropriate cytology and other tests should be ordered. There is no history of RA to suggest this as the underlying cause. There is also no history of exposure to TB, pyrexia or night sweats to point to TB as the likely diagnosis. Cardiac failure is more likely to present with a bilateral pleura effusion and a history of progressive worsening of IHD. Mesothelioma is possible but again no exposure to asbestos has been mentioned.

[A]

98/A 68-year-old retired plumber presents with a six month history of dry nocturnal cough and increasing exertional breathlessness. The cough is unproductive and there has been no haemoptysis.

He is comfortable at rest but his breathing limits him to 400 metres on the flat and he is beginning to have difficulty climbing stairs. He sleeps with four pillows. He had a myocardial infarction four years ago and has a 15 year history of hypertension. His current treatment is aspirin, atenolol, simvastatin and bendroflumethiazide.

On examination he has finger clubbing, cyanosis and looks pale. His pulse is 48 beats per minute and is regular. Blood pressure is 158/ 78 mmHg. On examining his chest he has vesicular breath sounds with bilateral basal crackles.

Investigations reveal:

Arterial blood gases on air		
PaO_2	8.2 kPa	(11.3-12.6)
$PaCO_2$	5.1 kPa	(4.7-6.0)
pH	7.41	(7.36-7.44)

Pulmonary function tests		
FEV1	2.3	(predicted 3.0)
FVC	2.8	(predicted 3.8)
FEV1/FVC	82%	-

The ECG showed a sinus bradycardia with Q waves in the anterior chest leads and left ventricular hypertrophy (by voltage criteria).

The chest x ray showed bilateral lower zone shadowing

Which one of the following investigations is most likely to establish the diagnosis?

(Please select 1 option)

○	Bronchoalveolar lavage
○	Echocardiography
○	High resolution CT scan of chest
○	Measurement of diffusion capacity
○	Serum angiotensin-converting enzyme (ACE) activity

		9	/w EPDw UJOTAw	ZWY9li9Bc3Nlc3
NDo1NmRkAgcPI	IVtDREFUQVs8cI	dGggYmlsYXRlcr	aW9yDQpjaGVzc	aWxsIGNvbmZpc
VEFbXV0+PC9vC	JHF1VmlldyRidG5			

The patient presents with symptoms and signs that are consistent with pulmonary fibrosis.

This is supported by his CXR which shows bilateral lower zone shadowing - a reticulo-rnodular pattern is seen - and his spirometry which demonstrates a restrictive defect.

His blood gases show type 1 respiratory failure, again which is consistent with the diagnosis.

High-resolution CT of the chest is the investigation of choice, often preventing the need for lung biopsy. It will confirm the diagnosis of interstitial fibrosis and can be diagnostic in a number of diseases such as:

Usual interstitial pneumonia (CFA)
Asbestosis, and
Sarcoidosis.

[C]

99/A 19-year-old man with a 10 year history of asthma presents with a six week history of worsening symptoms of exertional breathlessness and wheeze.

He has a history of eczema and hayfever as a child. He smokes 10 cigarettes a day and works as an electrician. In addition he has a cough which wakes him up most nights. The cough is unproductive.

Systems review was negative apart from some occasional diarrhoea. His current treatment is inhaled beclomethasone 800 ug per day and inhaled salbutamol 200 ug when required via a metered dose inhaler.

Which one of the following is the next most appropriate step in management?

(Please select 1 option)

○	Add aminophylline
○	Add montelukast
○	Add salmeterol
○	Change to a dry powder inhaler
○	Double the dose of beclomethasone

This patient has poorly controlled asthma.

His current regime places him at step 2 of BTS guidelines but there is a clear need for an increase in therapy to control symptoms.

Many randomised controlled trials (RCTs) and meta-analysis of RCTs (for example, Pauwels RA et al. *N Eng J Med*1997;337:1405-1411. Shrewsbury et al

*BMJ*200;320:1368-1373) have shown that the addition of a long acting b agonist (LABA) such as salmeterol or eformoterol improves symptom control, lung function, and reduces exacerbations over increasing the dose of inhaled corticosteroids.

Other studies show clear benefits of LABA over leucotriene receptor antagonists such as montelukasts and theophyllines. See BTS/Sign asthma guidelines in *Thorax* 2003 (58) Suppl 1. p1-94.

[C]

100/A 57-year-old man presented with deteriorating breathlessness over the last one year.

He had received inhalers for the last two years prescribed by his GP which he has, until the last three months, used intermittently. He was also taking ramipril 10 mg od and bendroflumethiazide 2.5 mg daily for a six year history of hypertension. He stopped smoking two years previously and consumed approximately 14 units of alcohol weekly.

On examination he was cyanosed and had a swollen face and dilated superficial veins over the anterior chest wall with fixed dilated neck veins. His blood pressure was 154/88 mmHg, pulse is 88 beats per minute. Heart sounds were normal. There was pitting oedema of the ankles. Respiratory examination revealed a hyperexpanded chest with scattered expiratory wheeze. Abdominal examination was normal.

Investigations revealed:

Haemoglobin	14.8 g/dL (13.0-18.0)
White cell count	12.91 $x10^9$/L (4-11 $x10^9$)
Platelets	488 $x10^9$/L (150-400 $x10^9$)
Serum sodium	130 mmol/L (137-144)
Serum urea	10.8 mmol/L (2.5-7.5)
Serum corrected calcium	2.81 mmol/L (2.2-2.6)

The ECG was normal. Chest x ray showed hyperexpanded lung fields with left paratracheal shadowing. A CT scan of the thorax showed an anterior mediastinal mass.

Which single investigation would be most helpful in making a diagnosis?

(Please select 1 option)

	Biopsy of the mediastinal mass
	Bone marrow trephine biopsy
	Bronchoscopy
	CT scan of the abdomen
	Tuberculin skin test

The patient presents with symptoms and signs of superior vena caval obstruction (SVCO).

This is due to the anterior mediastinal mass and the single most useful diagnostic investigation is histological confirmation of the lesion from a biopsy. This can usually be done by a percutaneous CT guided biopsy.

The most common cause of SVCO is primary lung cancer. Other causes include lymphoma.

Treatment is of the underlying condition although in some cases of non-small cell lung cancer stenting of the SVCO may be required for relief of symptoms prior to chemotherapy/palliative radiotherapy.

[A]

101/A 45-year-old retired coal miner with simple silicosis presented with shortness of breath.

He retired early and received a coal workers' pension. He had been short of breath for three months. He has kept budgerigars as pets for the last three months. On auscultation he had basal crepitations but CXR showed fine nodular shadowing in the apices.

What is the most likely diagnosis?

(Please select 1 option)

	Allergic bronchopulmonary aspergillosis
	Extrinsic allergic alveolitis
	Progressive silicosis
	Psittacosis
	Tuberculosis

Silicosis usually results in small nodular opacities in the mid and upper zones. There may be associated hilar gland enlargement which may calcify producing

characteristic 'eggshell' calcification. These radiological shadows are not usually associated with symptoms or loss of lung function.

Extrinsic allergic alveolitis (EAA) is an inflammatory reaction to inhaled organic dusts of which there are many causes such as*M. faeni*, *T. vulgaris* (farmer's lung),*Thermoactinomyces sacchari* (bagassosis), *A. clavatus* (malt worker's lung), etc.

Bird fancier's lung is EAA caused by the inhalation of avian serum proteins present particularly in the bloom of birds' feathers. It occurs most commonly among pigeon fanciers and budgerigar owners the latter usually presenting with a chronic EAA. Patients present with a progressive dyspnoea on exertion. Inspiratory crackles are heard on auscultation of the lungs. CXR shows fine linear opacities in the upper lobes which may progress to honeycombing.

The diagnosis of EAA is based on typical clinical, radiological and lung function changes in the presence of an identified source of antigen with positive precipitating antibodies in the patient's serum to the causal antigen.

Improvement of clinical abnormalities following avoidance of the causal antigen helps confirm the diagnosis.

[B]

102/A 58-year-old man presents with shortness of breath. His respiratory function tests are shown below:

Lung function test	Actual	Predicted
FEV_1(l)	2.4	3.0
FVC (l)	2.8	3.8
RV (l)	1.4	2.0
Tlco (mmol/min/kPa)	6.2	7.4
KCO (mmol/min/kPa/l)	1.7	1.4

Which of the following is the most likely diagnosis?

(Please select 1 option)

○	Anaemia
○	Cryptogenic fibrosing alveolitis (CFA)
○	Emphysema
○	Extrinsic allergic alveolitis
○	Pleural thickening

This patient's FEV_1 and FVC are both reduced producing a restrictive picture on spirometry. This is confirmed by the small RV.

Tlco is also reduced in restrictive defects but the elevated Kco is in keeping with extrapulmonary restriction. This can be caused by pleural disease such as pleural thickening, respiratory muscle weakness and chest wall disease such as thoracoplasty or scoliosis.

Emphysema produces an obstructive spirometry with increased RV but decreased Tlco.

CFA produces a restrictive picture but Kco is normally reduced although it can be normal but is never elevated.

Anaemia does not affect spirometry or lung volumes but will result in a decreased Tlco and Kco although there are equations to correct for this

[E]

103/A 32-year-old female floor manager for a paint warehouse presented with shortness of breath.

Recently, whilst on holiday in the Lake District her symptoms had completely resolved.

She presented in the early hours of the morning having used her salbutamol inhaler 12 times. She was treated with a salbutamol nebuliser and steroids and completely recovered.

Which is the best investigation to determine the diagnosis?

(Please select 1 option)

○	Bronchial provocation testing
○	CXR
○	PEFRs measure at home and work
○	RAST IgE
○	Skin prick testing

This patient has occupational asthma. She has symptoms of asthma that improve when she is on holiday from her job.

A good history is essential to diagnosing occupational asthma. All the jobs a patient has had need to be noted as well as materials to which they have been exposed, when they were exposed and the interval between exposures and development of symptoms.

Objective measurements are the key to confirming the diagnosis and are best performed using serial peak expiratory flow rate (PEFR) measured two hourly from waking to sleeping at least over a four week period which should include at least three periods away from work for at least two consecutive days, although patients with more severe disease may require more than 10 days away from the work environment before improvements are noted.

Whilst serial PEFR measurement is the diagnositic test of choice bronchial provocation tests and specific IgE radioallergosorbent tests (RAST) are required to identify the specific agent at work causing the asthma.

[C]

104/A 24-year-old male who works in a plastic factory presented with shortness of breath.

He went for two weeks to Spain and was completely well.

He presented at 0200 hours to theemergency unit after having used his salbutamol inhaler eight times. He was treated with a salbutamol nebuliser and steroids and completely recovered.

What is the most likely diagnosis?

(Please select 1 option)

○	Bronchopulmonary aspergillosis
○	Chemical pneumonitis
○	Extrinsic allergic alveolitis
○	Late onset asthma
○	Occupational asthma

Occupational asthma is the commonest industrial lung disease in the western world with more than 500 causes. It accounts for up to 10% of adult onset asthma. It is caused by agents that are encountered at work.

The commonest occupations affected (with causes) in the United Kingdom are

Paint sprayers (isocyanates)
Bakers (flour mainly but also enzymes such as amylase used in the baking process)
Chemical processors (acids, detergents, bleaches)
Plastics workers (polyethylene, polyvinyl chloride)
Solderers (colophony)
Laboratory technicians (rats, mice, rabbits, locusts).

The typical history is usually one of breathlessness, wheeze and cough which occur during the working week and remit during absences from work such as holidays.

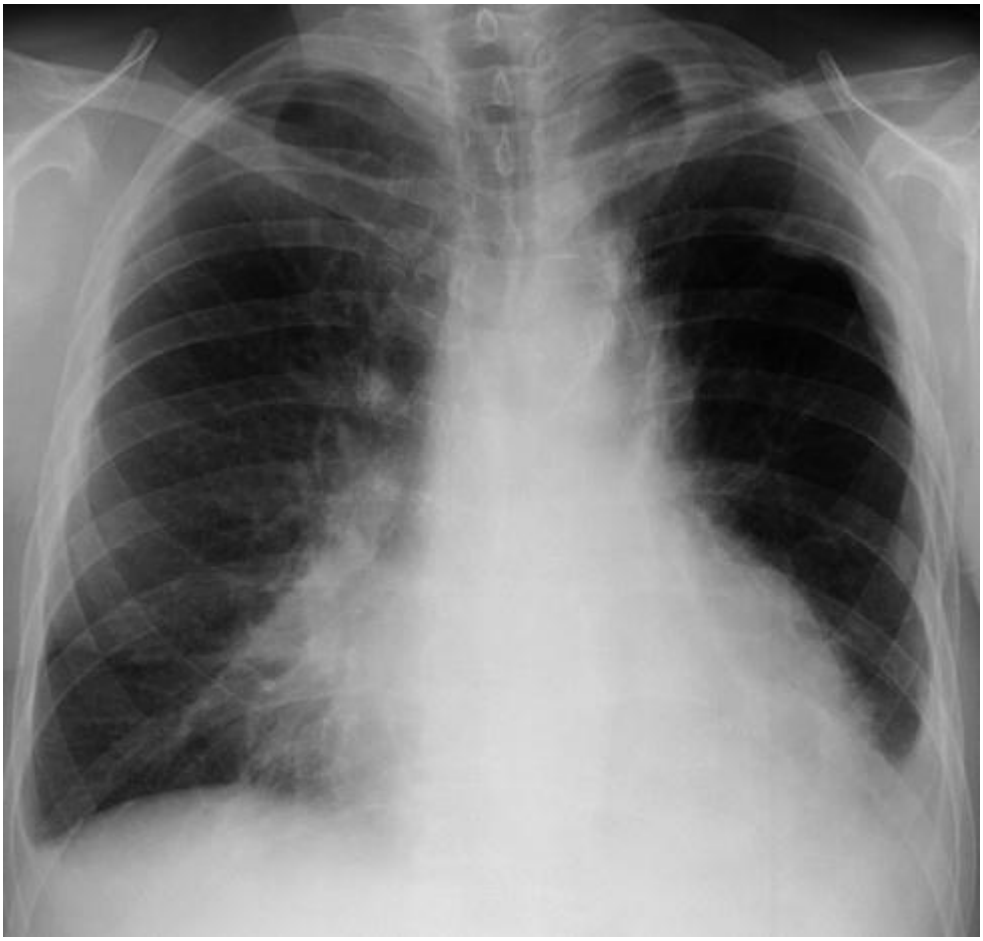
The symptoms do not usually develop immediately on first exposure but begin days, months or even years later.

Removal from exposure to the sensitising agent at an early stage can lead to remission of asthma although sensitisation to the agent is usually permananent.[E]

105/A 56-year-old female presented with a four day history of breathlessness, cough and a high fever.

She gave a long history of indigestion and intermittent dysphagia to solids and liquids. She would often wake at night with episodes of coughing and spluttering. There was no history of weight loss. On examination she had a temperature of 39°C, resp rate of 30/min and a HR of 102. On auscultation of the lung fields there were coarse basal crackles.

Her chest x ray on admission to hospital is shown.



What is the cause of her illness?

(Please select 1 option)

	Achalasia
	Benign oesophageal stricture
	Bronchiectasis
	Hiatus hernia
	Pharyngeal pouch

Achalasia results in megaoesophagus which is seen on plain CXR as smooth convex opacity widening the mediastinum to the right (as here). There is usually an absent gastric bubble (shown here). A fluid level is often present. If the oesophagus is full of air (for example, after oesophagoscopy) the thickened wall of the oesophagus can be clearly deliniated running parallel to the right heart border.

Achalasia results in dysphagia for both solids and liquids and retention of contents in the body of the oesophagus with regurgitation and resultant aspiration.

Aspiration can result in

1. A chemical pneumonitis from gastric acid aspiration
2. Mechanical obstruction from the aspiration of particulate matter and
3. Bacterial infection from the aspiration of oropharyngeal bacteria.

[A]

106/A 51-year-old man presents with a dry irritating unproductive cough. It often keeps him awake at night. He is a heavy snorer.

He admits to breathlessness on exertion and feels tired during the day. He has smoked 20 cigarettes a day since the age of 20 years and has recently been diagnosed as having hypothyroidism. He is overweight (BMI = 34).

His lung function shows:

	Observed	**Normal Range**
FEV1	2.8 L	(3.3 - 4.8)
VC	2.9 L	(3.7 - 5.6)
TLC	3.4 L	(6.0 - 8.2)
RV	0.9 L	(1.3 - 2.9)
TLCO	4.3 mmol/min/kPa	(8.2 - 12.8)

KCO	1.1 mmol/min/kPa/L	(1.2 - 2.0)

What is the most likely diagnosis?

(Please select 1 option)

	Asthma
	Bronchiolitis obliterans and organising pneumonia
	Chronic obstructive pulmonary disease
	Cryptogenic fibrosing alveolitis
	Sleep apnoea

This man's lung function demonstrates a restrictive defect with an increased ratio of forced expiratory volume in one second (FEV1)/forced vital capacity (FVC) and decreased lung volumes.

Both Tlco and Kco are reduced.

The Tlco is a sensitive (but not specific) indicator of the integrity of the alveolar capillary membrane and gas exchange function of the lung.

The Kco corrects for any reduction in lung volume (for example, after pneumonectomy).

[D]

107/A 66-year-old retired plumber presents with a two year history of progressive breathlessness.

He has no cough nor chest pain. He has a 50 pack/year smoking history and drinks 40 units of alcohol a week. His appetite is good and his weight is unchanged (BMI = 24).

His spirometry shows:

	Actual	**Predicted**
FEV1	2.7 L	3.3 L
FVC	2.9 L	4.2 L
PEF (l/min)	500	550
TLC	4.1 L	7.2 L

TLco (mmol/min/kPa)	7.8	8.0
Kco (mmol/min/kPa/l)	2.0	1.4

What is the diagnosis?

(Please select 1 option)

	Asbestosis
	Chronic obstructive airways disease
	Cryptogenic fibrosing alveolitis
	Extensive bilateral pleural thickening
	Extrinsic allergic alveolitis

This man's lung function shows a restrictive pattern with increased ratio of forced expiratory volume in one second (FEV1)/forced vital capacity (FVC) and decreased total lung capacity (TLC).

Extra-pulmonary restriction produces a characteristic pattern where Kco is greater than normal with a normal or slight reduction in Tlco because of the patient's inability to achieve a full inspiration.

Causes of extrapulmonary restriction include:

Pleural disease
Skeletal deformities, or
Respiratory muscle weakness.

Diffuse pleural thickening is usually as a result of asbestos exposure although the relationship to exposure dose is not as clearly defined as in asbestosis or mesothelioma. The Kco correlates with the degree of pleural thickening.

The degree of breathlessness and disability increases with increasing severity of pleural thickening. The condition tends to progress although in itself is not an additional risk factor for the development of malignancy

[D]

108/A 65-year-old retired miner attends the outpatient department with a six month history of breathlessness on exertion which has got worse following a recent chest infection.

He has a cough productive of mucoid sputum. He smoked 20 cigarettes a day until five years ago. He keeps pigeons at home.

Lung function tests before and after bronchodilators show:

	Before	After	Normal range
FEV1	0.9	1.0	(2.2 - 4.4)
FVC	2.1	2.3	(3.0 - 4.8)
TLC	7.2	-	(2.4 - 4.6)
Kco	0.6	-	(1.2 - 2.1)

What is the diagnosis?

(Please select 1 option)

○	Asthma
○	Chronic obstructive pulmonary disease (COPD)
○	Coal workers pneumoconiosis
○	Extrinsic allergic alveolitis
○	Pigeon fancier's lung

This man's lung function demonstrates an obstructive pattern.

The forced expiratory volume in one second (FEV1)/forced vital capacity (FVC) is usually greater than 75% in normal young people but falls to 70-75% in normal elderly subjects. Below this is an obstructive pattern.

As airway obstruction progresses both the FEV1 and FVC decrease. There is an increased total lung capacity (TLC).

The results also show that there is no significant reversibility to bronchodilators (that is, less than 15% improvement in FEV1) and the transfer co-efficient is reduced which is consistent with a diagnosis of COPD rather than asthma.

COPD is divided into

Mild (FEV= >80%)
Moderate (50-79%)
Severe (30-49%)
Very severe (less than 30% predicted).

[B]

109/A 72-year-old female was admitted with a deteriorating dyspnoea and fever which had deteriorated over the preceding three days.

Prior to this admission she had been well having returned one week ago from a Spanish holiday with her husband. The only other history of note was that three years

ago she was diagnosed with type 2 diabetes for which she was treated with diet alone. She is a smoker of five cigarettes per day.

On examination she was suntanned, slightly confused with saturations of 92% on air. She had a pyrexia of 40°C, a pulse of 118 bpm and a blood pressure of 118/90 mmHg. Auscultation of the chest revealed left basal crackles only.

Investigations showed:

Haemoglobin	14.3 g/dL (11.5-16.5)
White cell count	8.2 $x10^9$/L (4-11 $x10^9$)
Platelets	320 $x10^9$/L (150-400 $x10^9$)
Serum sodium	123 mmol/L (137-144)
Serum potassium	3.6 mmol/L (3.5-4.9)
Urea	4.2 mmol/L (2.5-7.5)
Plasma glucose	10.9 mmol/L (3.0-6.0)
Urine sodium concentration	35 mmol/L

Arterial blood gas analysis:

pH	7.36 (7.36-7.44)
pCO_2	5.1 kPa/38 mmHg (4.7-6.0 kPa)
pO_2	10.7 kPa/80 mmHg (11.3-12.6 kPa)
Standard bicarbonate	30 mmol/L (20-28)

Which of the following tests would be most useful in providing diagnostic information?

(Please select 1 option)

○	Blood culture
○	Serum antibody tests
○	Short Synacthen test
○	Sputum culture
○	Urine antigen test

This patient is likely to have legionnaire's disease.

This is due to *Legionella pneumophila*a Gram negative rod which is frequently found in heating systems and air conditioner units.

The urinary antigen test for *Legionella* species is the most useful test being rapidly available and accurate (70% specificity and 100% sensitivity).

Blood cultures are frequently negative and sputum culture may not be positive and take approximately three days to process.

Again serologic tests for *Legionella* antibodies may be negative for up to three months after infection and require acute and convalescent samples.

It is associated with a 5-15% mortality associated with respiratory and renal failure. The low serum sodium and urine sodium excretion are mainly related to SIADH.

It is treated with macrolides and/or ofloxacins

[E]

110/A 68-year-old man was admitted with nausea and general malaise.

He was a hill farmer and continued to work his farm, on which he kept mostly sheep, with his sons. Over the last two weeks, since returning from holiday in Spain, he had become increasingly fatigued.

The only other symptoms of note were a three month history of poor appetite and a 8 kg weight loss. He was receiving thyroxine 100 mcgm daily having been diagnosed with hypothyroidism by his GP nine years previously. He was a smoker of five cigarettes per day and had drunk more alcohol than usual whilst on holiday but usually drank about 12 units of alcohol daily.

On examination, he was sun-tanned, slightly confused, appeared dehydrated and had a pulse of 92 bpm regular, a temperature of 37.2°C with a blood pressure of 100/80 mmHg. Cardiovascular and respiratory examination were unremarkable. He had a slight liver edge on palpation and neurological examination was normal.

Investigations revealed:

Serum sodium	125 mmol/L (137-144)
Serum potassium	5.6 mmol/L (3.5-4.9)
Serum corrected calcium	2.73 mmol/L (2.2-2.6)
Serum standard bicarbonate	15 mmol/L (20-28)
Serum urea	22 mmol/L (2.5-7.5)
Plasma TSH	6 mU/L (0.4 - 4.0)

Which of the following is the most likely diagnosis?

(Please select 1 option)

	Bronchogenic carcinoma with syndrome of inappropriate ADH (SIADH) secretion.
	Hypoadrenalism
	Hypothyroidism
	Primary hyperparathyroidism
	Sarcoidosis

This farmer has a three month history of weight loss, anorexia and fatigue. He has recently returned from Spain but this is a distractor as is his occupation.

The investigations show hyponatraemia, hyperkalaemia, uraemia, hypercalcaemia and slightly elevated thyroid-stimulating hormone (TSH) which suggest the diagnosis of hypoadrenalism.

He is known to have an autoimmune disease - hypothyroidism and a diagnosis of Addison's is suggested.

Slight elevation of TSH and a mild hypercalcaemia are typical of hypoadrenalism.

Bronchogenic carcinoma with SIADH would be expected to produce a hyponatraemia but with normal potassium and urea.

The patient's symptoms do not fit with hypothyroidism (weight loss) and the uraemia and hyperglycaemia would not be expected.

Although sarcoidosis may produce the hypercalcaemia, the hyponatraemia would not be a typical finding and respiratory signs may be expected.

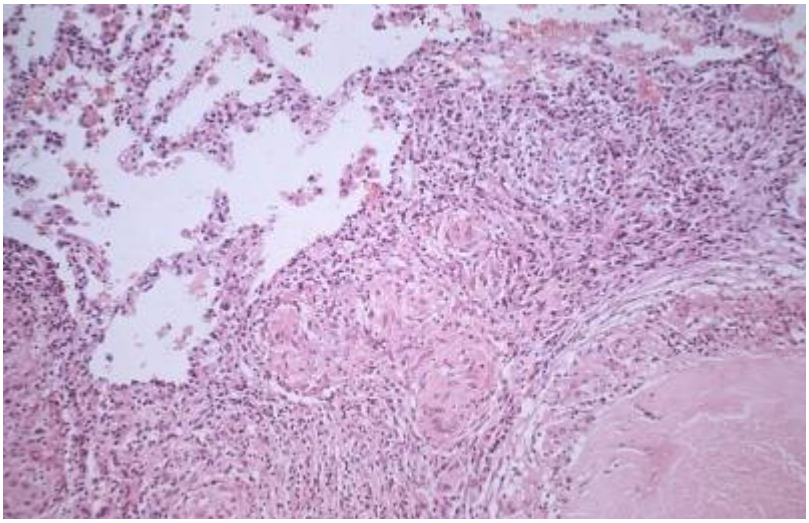
Primary hyperparathyroidism would not produce such problems with such a calcium concentration.

[B]

111/A 65-year-old man presented with an upper respiratory tract infection.

He had a longstanding history of occupational lung disease. Despite maximal therapy he developed respiratory failure and died.

A post-mortem examination was requested as he had been an inpatient for less than 24 hours. Lung histology is shown.



On the basis of the lung histology, which of the following agents was most likely responsible for his occupational lung disease?

(Please select 1 option)

	Asbestos
	Beryllium
	Coal dust
	Magnesium silicate
	Silicon dioxide

The lung histology shows non-caseating granulomata, a characteristic feature of berylliosis.

Asbestos exposure can lead to pulmonary fibrosis, pleural plaques and mesothelioma.

Occupational exposure to the other options listed has the potential to induce pulmonary fibrosis.

[B]

112/A 72-year-old female was admitted with a deteriorating dyspnoea and fever which had deteriorated over the preceding three days.

Prior to this admission she had been well having returned one week ago from a Spanish holiday with her husband. The only other history of note was that three years ago she was diagnosed with type 2 diabetes for which she was treated with diet alone. She is a smoker of five cigarettes per day.

On examination she was suntanned, slightly confused with saturations of 92% on air. She had a pyrexia of 40C, a pulse of 118 bpm and a blood pressure of 118/90 mmHg. Auscultation of the chest revealed left basal crackles.

Investigations revealed:

Haemoglobin	14.3 g/dL (11.5-16.5)
White cell count	8.2 $x10^9$/L (4-11 $x10^9$)
Platelets	320 $x10^9$/L (150-400 $x10^9$)
Serum sodium	128 mmol/L (137-144)
Serum potassium	3.6 mmol/L (3.5-4.9)
Serum urea	8.2 mmol/L (2.5-7.5)
Serum glucose	10.9 mmol/L (3.0-6.0)
Urine sodium	15 mmol/L

Arterial blood gases showed:

pH	7.36 (7.36-7.44)
pCO_2	5.1 kPa (4.7-6.0)
pO_2	10.7 kPa (11.3-12.6)
Standard HCO_3	30 mmol/L (20-28)

How would you treat this patient's hyponatraemia?

(Please select 1 option)

○	Demeclocycline therapy
○	Fluid restriction
○	Furosemide therapy
○	Hypertonic saline
○	Normal saline

This patient presents with high temperature, dyspnoea and mild hyponatraemia following a Spanish holiday.

The likely diagnosis is legionnaire's disease.*Legionella* infection is treated with antimicrobials that achieve high concentrations intracellularly (macrolides, quinolones, rifampicin, tetracycline).

She has a moderate hyponatraemia which is likely multifactorial. In view of the high fever, lowish BP, tachycardia, elevated BUN, and urine Na <30, IV normal saline is indicated.

Demeclocycline should be reserved for chronic syndrome of inappropriate antidiuretic hormone (SIADH) and is not an appropriate therapy for legionnaire's disease.

[E]

113/A 28-year-old Afro-Caribbean woman presents with a three month history of progressively worsening dyspnoea. She usually went jogging every morning but found it increasingly difficult to maintain her usual circuit. Over the past three days she has become breathless walking upstairs at home. She also complains of a dry cough and a low-grade fever. There is no history of weight loss.

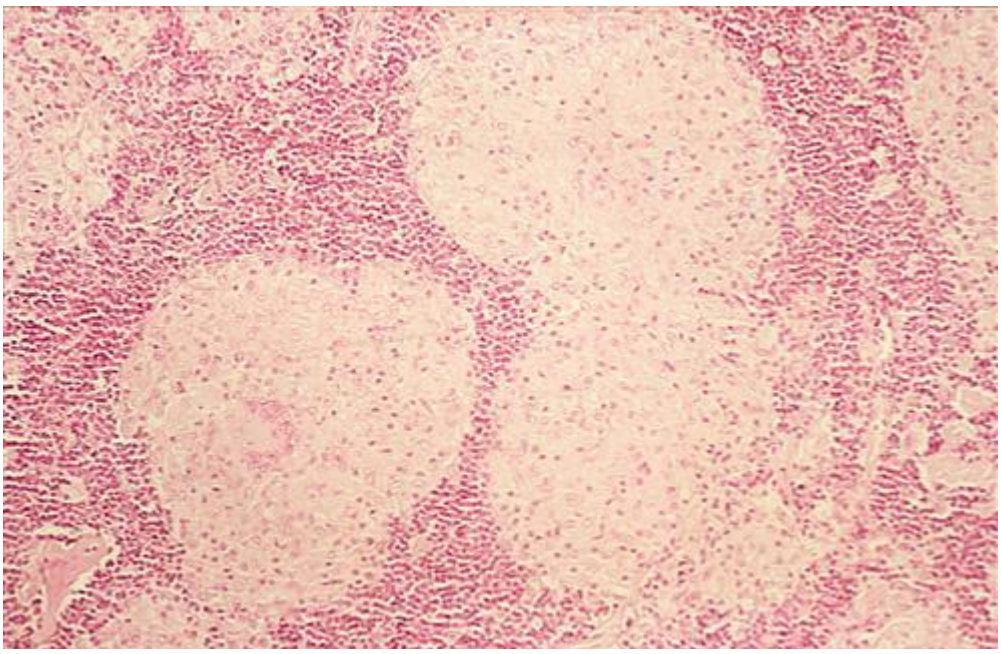
There is no past history of note. She has lived in the United Kingdom for five years and is married with two children. There is no history of recent travel and there have been no sick contacts. She was immunised with BCG as a child.

On examination she is febrile (37.5°C) and multiple bilateral cervical lymph nodes are palpable. Fine inspiratory crackles are heard in both mid- and lower-zones of her chest.

Investigations show:

Haemoglobin	14.8 g/dL	(11.5-16.5)
WBC	9.8 $\times 10^9$/L	(4-11)
Platelets	355 $\times 10^9$/L	(150-400)
Sodium	137 mmol/L	(137-144)
Potassium	4.2 mmol/L	(3.5-4.9)
Urea	15.1 mmol/L	(2.5-7.5)
Creatinine	224 µmol/L	(60-110)

Chest x ray shows diffuse bilateral interstitial shadowing, most marked in the middle zones. A Mantoux test was positive at a low level. A lymph node biopsy was performed; histology of the excised node is shown.



What is the most likely diagnosis?

(Please select 1 option)

○	Brucellosis
○	HIV infection with generalised lymphadenopathy
○	Hodgkin's disease (nodular sclerosing type)
○	Sarcoidosis
○	Tuberculous lymphadenitis

Sarcoidosis is a disease of unknown cause characterised by the presence histologically of non-caseating granulomata.

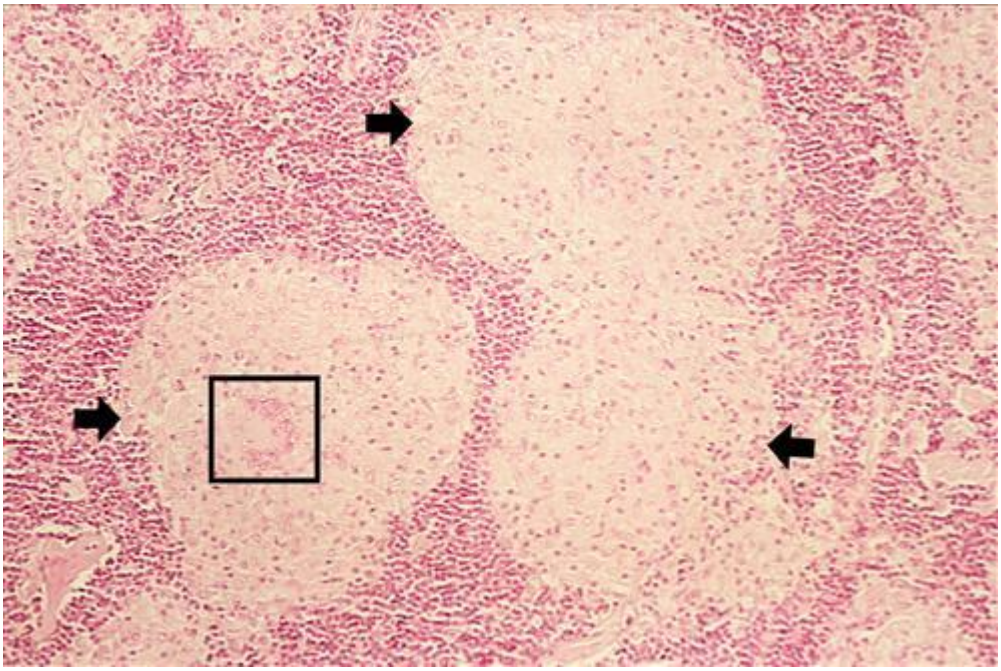
The disease often affects young adults and is commoner in Afro-Caribbeans.

Although any organ may be affected, patients typically present with pulmonary and/or extrapulmonary disease. Symptoms include dyspnoea, fever and cough. Lymphadenopathy may be present either clinically or on the chest x ray.

Other features include erythema nodosum, arthralgia, cranial nerve palsies and hepatosplenomegaly.

Hypercalcaemia is due to increased production of vitamin D precursors by macrophages within sarcoid granulomata and may induce hypercalcaemic nephropathy.

The lymph node biopsy specimen shown contains three large granulomata (arrowed, below). In addition, the granuloma at the bottom left of the picture also contains a multinucleate Langhans giant cell (in box). There is no evidence of caseation.



[D]

114/A 66-year-old retired foundry worker was referred to the medical admissions unit after developing sudden onset of right sided chest pain. For the past 24 hours he has felt unwell, with malaise, headache and myalgias. The GP's letter states that the patient has become mildly confused over the past three to four hours.

On examination he is febrile (39°C) and confused. Pulse 62 per minute, blood pressure 110/75.

Investigations showed:

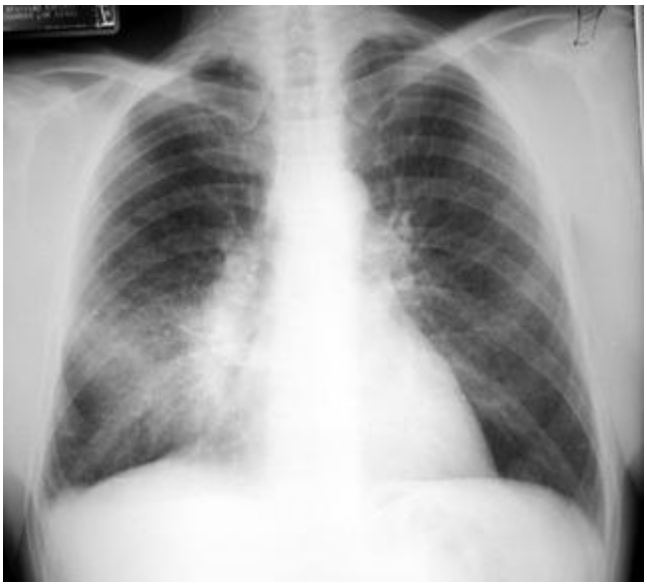
Haemoglobin	16.5 g/dL	(13.0-18.0)
White cell count	20.1 $\times 10^9$/L	(4-11)
Neutrophils	18.5 $\times 10^9$/L	(1.5-7)
Lymphocytes	0.8 $\times 10^9$/L	(1.5-4)
Monocytes	0.8 $\times 10^9$/L	(0-0.8)
Platelets	390 $\times 10^9$/L	(150-400)
Serum sodium	121 mmol/L	(137-144)
Serum potassium	4.3 mmol/L	(3.5-4.9)
Serum urea	6.2 mmol/L	(2.5-7.5)
Serum creatinine	99 µmol/L	(60-110)

Serum bilirubin	7 µmol/L	(1-22)
Serum AST	63 U/L	(1-31)
Serum ALP	100 U/L	(45-105)
Serum albumin	39 g/L	(37-49)

ABGs on air:

pH	7.42	(7.36-7.44)
pO_2	9.9 kPa	(11.3-12.6)
pCO_2	3.9 kPa	(4.7-6.0)
Bicarbonate	22 mmol/L	(20-28)
Urinalysis:	Protein +	

His chest x ray is shown below.



Which of the following investigations is most likely to establish the identity of the causative organism?

(Please select 1 option)

○	Blood cultures
○	*Chlamydia pneumoniae* serology
○	Immunocytological staining of bronchoalveolar lavage fluid
○	*Legionella* urinary antigen

	PCR

This patient's symptoms, signs and investigations are highly suggestive of *Legionella* pneumonia.

The illness may start abruptly with a brief prodrome of malaise, myalgia and headache. High fever and non-productive cough are common and may be accompanied by pleuritic chest pain. Confusion may represent toxic encephalopathy.

A marked neutrophil leukocytosis may be associated with concomitant lymphopenia.

Hyponatraemia occurs more commonly than with other pneumonias.

Liver function abnormalities are common but non-specific.

Proteinuria (sometimes myoglobinuria) is common.

Chest x ray usually shows lobar consolidation and progresses to bilateral involvement in 50% of cases.

Although the diagnosis may be made by culturing the organism from sputum, tests for *Legionella* antigens in urine offer a rapid test.

[D]

115/A 66-year-old retired foundry worker was referred to the medical admissions unit after developing sudden onset of right sided chest pain. For the past 24 hours he has felt unwell, with malaise, headache and myalgias.

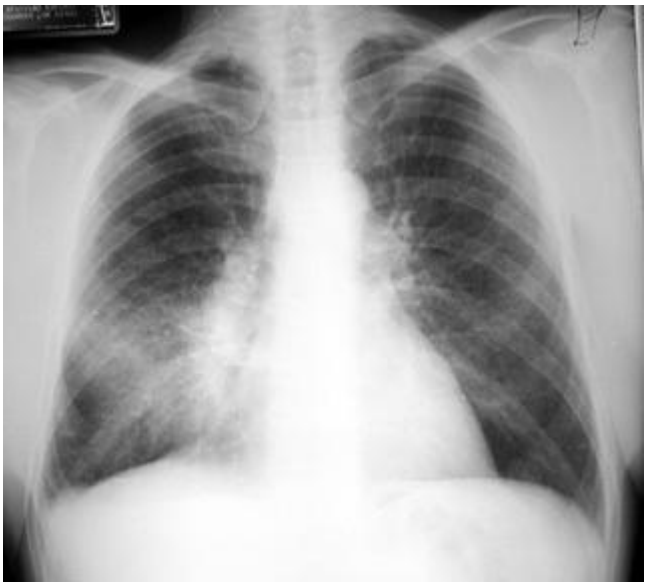
The GP's letter states that the patient has become mildly confused over the past three to four hours. On examination he is febrile (39°C) and confused. Pulse 62 per minute, blood pressure 110/75 mmHg.

Investigations show:

Haemoglobin	16.5 g/dL	(13.0-18.0)
WBC	20.1 $\times 10^9$/L	(4-11)
Neutrophils	18.5 $\times 10^9$/L	(1.5-7)
Lymphocytes	0.8 $\times 10^9$/L	(1.5-4)
Monocytes	0.8 $\times 10^9$/L	(0-0.8)
Platelets	390 $\times 10^9$/L	(150-400)
Serum sodium	121 mmol/L	(137-144)
Serum potassium	4.3 mmol/L	(3.5-4.9)
Serum urea	6.2 mmol/L	(2.5-7.5)

Serum creatinine	99 µmol/L	(60-110)
Serum bilirubin	7 µmol/L	(1-22)
Serum aspartate transaminase	63 U/L	(1-31)
Serum alkaline phosphatase	100 U/L	(45-105)
Serum albumin	39 g/L	(37-49)
pH	7.42	(7.36-7.44)
pO_2	9.9 kPa/75 mmHg	(11.3-12.6)
pCO_2	3.9 kPa/29 mmHg	(4.7-6.0)
HCO_3	22 mmol/L	(20-28)
Urinalysis:	Protein (+)	

His chest x ray is shown below.



Which of the following antibiotic regimes would be most effective in treating this specific infection?

(Please select 1 option)

○	Amoxicillin
○	Doxycycline
○	Amoxicllin and Erythromycin
○	Erythromycin and rifampicin

	Levofloxacin

This patient's symptoms, signs and investigations are highly suggestive of *Legionella* pneumonia.

The illness may start abruptly with a brief prodrome of malaise, myalgia and headache. High fever and non-productive cough are common and may be accompanied by pleuritic chest pain. Confusion may represent toxic encephalopathy.

A marked neutrophil leukocytosis may be associated with concomitant lymphopenia.

Hyponatraemia occurs more commonly than with other pneumonias.

Liver function abnormalities are common but non-specific.

Proteinuria (sometimes myoglobinuria) is common.

Chest x ray usually shows lobar consolidation and progresses to bilateral involvement in 50% of cases.

The newer macrolides (especially azithromycin) and the newer quinolones (especially levofloxacin) are effective for treating legionellosis. In comparison with erythromycin, these agents are more potent, have better tissue penetration and significantly less gastrointestinal toxicity.

With these more potent antibiotics, legionnaires' disease can be treated with monotherapy.

There are some reports that patients who receive levofloxacin defervesce more quickly than those who receive azithromycin, but the difference is not statistically significant.

Rifampin combined with erythromycin was previously recommended based on reports that these antibiotics in combination were more effective than erythromycin alone. However, combination therapy is now only recommended in patients who are failing standard therapy.

Other drugs that can be used include tetracycline, doxycycline, and co-trimoxazole.

[E]

116/A 53-year-old man is admitted with a history of intermittent haemoptysis, cough and breathlessness over the last several weeks.

On examination, he is apyrexial and is not clubbed. There are scattered crackles in his chest.

Investigations show:

Haemoglobin	9.8 g/dL (13.0-18.0)
MCV	76 fL (80-96)

White cell count	15.0 $x10^9$/L (4-11 $x10^9$)
Platelets	246 $x10^9$/L (150-400 $x10^9$)
ESR	89 mm/1st hour (0-20)
Serum sodium	134 mmol/L (137-144)
Serum potassium	4.9 mmol/L (3.5-4.9)
Serum urea	14 mmol/L (2.5-7.5)
Serum creatinine	189 µmol/L (60-110)
D-dimers	390

The chest x ray showed diffuse alveolar infiltrates.

Sputum cytology was negative for malignant cells.

What is the most likely diagnosis?

(Please select 1 option)

○	Churg-Strauss syndrome
○	Goodpasture’s syndrome
○	Polyarteritis nodosa (PAN)
○	Pulmonary embolism
○	Tuberculosis

Goodpasture's syndrome is characterised by pulmonary haemorrhage and crescentic glomerulonephritis.

Circulating antibodies against antigens in the glomerular and alveolar basement membranes are present. It is almost always associated with a microcytic anaemia.

Initially there may be mild proteinuria and microscopic haematuria, but acute renal failure may progress rapidly.

The carbon monoxide transfer factor is markedly elevated because the blood in the alveoli combined with the carbon monoxide.

PAN rarely involves the lungs.

Churg-Strauss syndrome is associated with asthma, eosinophilia and vasculitis with granuloma in various organs.

Neither TB nor PE are consistent with the clinical findings.[B]

117/An 82-year-old man complains of a six month history of feeling generally unwell, tired and lethargic.

He complains of aches and pains, especially of the left chest wall and shoulder. He has also lost 10 lbs in weight over the last three months because of a loss of appetite. There is longstanding prostatism, which has got gradually worse. His past medical history includes previous cholecystectomy and pancreatitis.

Examination reveals a mildly tender left chest wall, and discomfort when moving his left shoulder. A left partial ptosis and miosis are noted. There is no evidence of clubbing or lymphadenopathy. Percussion note over his chest is normal, as are his breath sounds.

CXR is normal.

What is the next investigation of choice?

(Please select 1 option)

○	Bronchoscopy
○	Computed tomography of chest
○	Isotope bone scan
○	Prostatic specific antigen level
○	Ultrasound abdomen

This patient has a left Horner's syndrome from a Pancoast's tumour which compresses the sympathetic fibres (from T1 and T2) as they travel upwards to superior cervical ganglion and then to the dilator pupillae as the long ciliary nerve.

Carcinoma at the apex of the lung can erode the ribs and the lower part of the brachial plexus (C8, T1, T2) causing pain in the shoulder and the medial surface of the arm. The diagnosis is often delayed as the most common feature of Pancoast's tumour is pain, extending down the arm to the elbow.

Patients may initially present to rheumatologists or orthopaedic surgeons.

CT is essential to locate the tumour and the extent of rib, vertebral and muscle involvement.

[B]

118/A 56-year-old woman is admitted with one stone weight loss over the last six months and worsening breathlessness. Her exercise tolerance is now limited to walking 20 yards on the flat.

She also complains of orthopnoea, requiring four pillows at night. She has a history of mild ischaemic heart disease and asthma. She has smoked 15 cigarettes perday for many years and admits to drinking 30 units of alcohol per week.

On examination, there is no evidence of clubbing or lymphadenopathy. Examination confirms a right pleural effusion, extending up to the upper zone. Her jugular venous pressure is not elevated.

Investigations show:

Chest x ray	Right pleural effusion
Isotope bone scan	Increased uptake in vertebrae, pelvis and right femur
Bronchoscopy	No endobronchial lesion is seen, but there is some distortion of right bronchial anatomy due to extrinsic compression. Bronchial trap cytology negative for malignant cells

What is the most appropriate diagnostic procedure?

(Please select 1 option)

○	CT scan thorax
○	Echocardiogram
○	Pleural fluid analysis
○	Serum tumour markers
○	Video assisted thoracoscopy (VATS)

There is likely to be a central tumour accounting for the external compression seen on bronchoscopy. This may well be amenable to CT guided biopsy.

[A]

119/A 48-year-old lady presents with increasing breathlessness and cough. This has been getting worse over the last year and she has had repeated chest infections over the last six months. She smoked 10 cigarettes a day until eight years ago. She has no known allergies. She works as a hairdresser.

A chest x ray was reported as being normal.

Pulmonary function testing demonstrated:

FEV_1	1.60 l	(53% predicted)

FVC	2.86 l	(78% predicted)
Total lung capacity	4.83 l	(110% predicted)
TLCO	6.63%	(93% predicted)
KCO	1.36	(120% predicted)

What is the most likely diagnosis?

(Please select 1 option)

○	Asthma
○	Chronic bronchitis
○	Emphysema
○	Obesity
○	Pulmonary embolism

This lady has moderate airways obstruction: FEV_1/FVC = 56% predicted.

Transfer factor and transfer co-efficient can be normal or elevated in patients with asthma but are always reduced in emphysema.

Patients with extra-pulmonary restrictive defects such as morbidity show an elevated KCO with normal TLCO, but the restrictive defect produces a normal or elevated FEV_1/FVC and reduced lung volumes.

Although in chronic bronchitis the KCO may be relatively well preserved (in the absence of emphysema) it would not be raised. Elevated KCO is more typical of asthma, possibly occurring due to increased density of pulmonary capillaries secondary to active inflammation.

There is also an occupational link between hair bleach/spray and asthma.

[A]

121/ A 48-year-old teacher is admitted with a two day history of increasing breathlessness and cough productive of purulent sputum.

He has smoked 20 cigarettes a day since the age of 18. He has not been in hospital before but was recently diagnosed by his general practitioner as having chronic obstructive pulmonary disease. He is taking an inhaled agonist on an as required basis.

On examination he is breathless at rest, alert and orientated. He is cyanosed and has a respiratory rate of 26 breaths per minute. His temperature is 37.8°C. His pulse is

100/minute and blood pressure is 150/100 mmHg. Auscultation of his chest reveals bilaterally reduced air entry.

His chest radiograph demonstrates a normal heart size but the lung fields are hyperinflated. There is no pneumonic consolidation.

Arterial blood gases (ABGs) on admission on 24% oxygen by nasal cannulae show:

pH	7.34 (7.36-7.44)
pO_2	6.5 kPa (11.3-12.6)
pCO_2	6.8 kPa (4.7-6.0)
Standard bicarbonate	27 mmol/L (20-28)

He is treated with nebulised bronchodilators and his FIO_2 is increased to 28%. The results of arterial blood gases repeated after 30 minutes are:

pH	7.30 (7.36-7.44)
pO_2	7.0 kPa (11.3-12.6)
pCO_2	8.5 kPa (4.7-6.0)
Standard bicarbonate	28 mmol/L (20-28)

What further management is required now?

(Please select 1 option)

○	Continuous positive pressure ventilation (CPAP)
○	Give oxygen by face mask
○	Intubation and mechanical ventilation
○	Non-invasive positive pressure ventilation (NIPPV/NIV)
○	Reduce FiO_2 to 24%

This patient's ABGs are deteriorating and he is developing an increasingly severe respiratory acidosis.

He is still alert and is haemodynamically stable and therefore NIV (such as BiPAP) is the treatment of choice and should be instigated without delay

[D]

122/ A 26-year-old diabetic patient presented to the emergency department at 2 am.

He had been to a party but complained of sudden worsening of a three day history of increasing breathlessness.

The following results were obtained from an arterial blood gas sample.

pH	7.66 (7.36 - 7.44)
pO_2	7.4 kPa (11.3 - 12.6)
pCO_2	4.7 kPa (4.7 - 6.0)
Serum bicarbonate	28 (20 - 28)
H^+	21 nmol/L (35 - 45)

What is your interpretation of these blood gas results?

(Please select 1 option)

○	Laboratory error
○	Metabolic acidosis and respiratory alkalosis
○	Metabolic alkalosis
○	Mixed metabolic and respiratory alkalosis
○	Respiratory alkalosis

The patient is severely alkalotic and has a normal bicarbonate. He has a normal pCO_2 and is hypoxic. The only explanation must be a laboratory error.

The Henderson-Hasselbalch equation might be of use here. In short the pH is proportional to the ratio of bicarbonate to pCO_2. The ratio here is normal so why is the pH so alkalotic? It must be an error.

Also why has the H^+ concentration been quoted? You can derive the pH from the H^+ concentration and the pH level is about right for that H^+ concentration but who on earth would be expected to calculate that in an exam?! I'll tell you who ... one of the editors of the question.

Without the H^+ ion concentration they may have complained saying that it was not obvious enough and could perhaps be explained by something else.

Bizarre things in tests should raise your suspicions of corrective editorial forces.

[A]

123/ An 18-year-old student presented with sudden onset of left sided chest pain and breathlessness.

He had a history of cardiac surgery as a child. He smoked ten cigarettes a day. He denied any alcohol or illicit drug use. He had returned from a holiday in Thailand ten days ago.

On examination he was tall and thin. There was no clubbing or lymphadenopathy. He was not cyanosed. He had a resting tachycardia and there was an audible click with expiration.

What is the likely diagnosis?

(Please select 1 option)

	Marfan's syndrome
	Mitral valve prolapse
	Pulmonary embolism
	Tietze's syndrome
	Viral pericarditis

Sudden onset chest pain and breathlessness are most likely due to a PE or pneumothorax.

Hamman's sign (or 'crunch') is a crunching systolic sound heard over the sternal edge in mediastinal emphysema or left apical pneumothoraces.

It can be dependent on the patient's position when auscultating.

[A]

124/ A 54-year-old sales representative was referred by his general practitioner complaining of feeling tired all the time.

He had a history of depression for which he was taking anti-depressant tablets prescribed by the GP. He had recently resigned from his job as he was too tired to do the large amount of driving required and had nearly been involved in a car accident when his car had swerved across the road for no apparent reason.

He was overweight and admitted to 3 stone increase in weight over the last three years. His blood pressure was elevated at 170/100 mmHg.

What is the most likely diagnosis?

(Please select 1 option)

	Absence seizures

	Chronic hyperventilation syndrome
	Hypothyroidism
	Infectious mononucleosis
	Obstructive sleep apnoea syndrome

Obstructive sleep apnoea (or sleep apnoea/hypopnoea) syndrome occurs when episodes of partial or complete obstruction of the pharyngeal airway occur during sleep.

This causes

A. Repetative apnoeas (cessation of airflow for more than 10 seconds) and hypopnoeas (50% reduction in airflow for greater than 10 seconds)

B. Loud snoring and

C. Excessive daytime somnolence as a result of repeated arousals.

The gold standard diagnostic test is overnight polysomnography.

Increasingly though simpler sleep monitoring systems or simple overnight oximetry are being used often with the studies undertaken in the patient's home.

The treatment of choice is weight loss, avoid sedatives drugs/excess alcohol and nasal continuous positive airway pressure (CPAP).

[E]

125/ A 54-year-old sales representative was referred by his general practitioner complaining of feeling tired all the time. He had a history of depression for which he was taking anti-depressant tablets prescribed by the GP.

He had recently resigned from his job as he was too tired to do the large amount of driving required and had nearly been involved in a car accident when his car had swerved across the road for no apparent reason. He was overweight and admitted to 3 stone increase in weight over the last three years, which he had put down to depression. His blood pressure was elevated at 170/100 mmHg.

Which investigation is most likely to explain the reason for his presentation?

(Please select 1 option)

	24 hour blood monitoring
	EEG
	Overnight oximetry

	Paul Bunnell test
	TSH level

Obstructive sleep apnoea (or sleep apnoea/hypopnoea) syndrome occurs when episodes of partial or complete obstruction of the pharyngeal airway occur during sleep.

This causes

A. Repetative apnoeas (cessation of airflow for more than 10seconds) and hypopnoeas (50% reduction in airflow for greater than 10 seconds)

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C. Excessive daytime somnolence as a result of repeated arousals.

The gold standard diagnostic test is overnight polysomnography.

Increasingly though simpler sleep monitoring systems or simple overnight oximetry are being used often with the studies undertaken in the patient's home. The treatment of choice is weight loss, avoid sedatives drugs/excess alcohol and nasal continuous positive airway pressure (CPAP).

[C]

126/ A 56-year-old builder presents with cough and breathlessness.

He is known to have chronic obstructive pulmonary disease and ulcerative colitis. He smokes up to 20 cigarettes a day.

The cough is productive of clear sputum up to 500 ml a day. He has had no haemoptysis. The breathlessness now restricts his exercise tolerance to 50 metres. He has lost over 2 stone in two months.

Examination revealed dullness to percussion at the right lung base. His abdomen was generally tender but there was no guarding.

What is the most likely diagnosis?

(Please select 1 option)

	Adenocarcinoma of the lung
	Alveolar proteinosis
	Bronchiolitis obliterans with organising pneumonia
	Bronchioloalveolar cell carcinoma
	Bronchopleural fistula

Bronchioloalveolar cell carcinoma of the lung accounts for around 5% of all primary lung carcinomas.

The classic massive clear frothy sputum that is produced by patients with this cancer is a late manifestation but can be up to one litre a day. Other symptoms are dyspnoea, weight loss and chest pain.

Almost a half of patients are diagnosed on routine CXR, usually demonstrating a peripheral lesion.

Its name arises from its pattern of growth along the alveolar walls without actually destroying them.

It is an adenocarcinoma.

In those whose tumour is not resectable, prognosis is poor.

[D]

127/ A 26-year-old man presents with haemoptysis.

He has had a productive cough since childhood and suffered from recurrent sinusitis. He was known to be infertile.

Investigations revealed normal immunoglobulins, normal sweat sodium and negative skin prick tests for grass pollen, house dust mite and aspergillus.

What is the most likely diagnosis?

(Please select 1 option)

○	Bronchiectasis
○	Bronchiolitis
○	Cystic fibrosis
○	Primary ciliary dyskinesia
○	Situs inversus

Primary ciliary dyskinesia is a hereditary condition in which there is partial or complete deficiency of outer or inner dynein arms of cilia causing slow and poorly co-ordinated ciliary beating throughout the body.

Patients suffer from bronchiectasis and sinusitis. They are infertile because of the reduced motility of the sperm. It is associated in 50% of cases with dextrocardia and situs inversus when it is called Kartagener's syndrome.

Patients with cystic fibrosis also have bronchiectasis and sinusitis and are infertile as the vas deferens fails to develop. Patients with cystic fibrosis have an abnormal sweat test with high levels of sodium and chloride although care needs to be taken

with interpreting the test in adults. The diagnosis is usually confirmed by determining the patient's genotype.

[D]

128/ The following blood gases were obtained on a 58-year-old confused female:

pH	7.66	(7.36-7.44)
pO_2	7.4 kPa	(11.3-12.6)
pCO_2	5.9 kPa	(4.7-6.0)
HCO_3	34 mmol/L	(20-28)

What is your interpretation of these blood gas results?

(Please select 1 option)

○	Laboratory error
○	Metabolic alkalosis
○	Metabolic alkalosis with respiratory acidosis
○	Mixed metabolic and respiratory alkalosis
○	Respiratory alkalosis

The pH is high indicating an alkalosis and the high bicarbonate suggests a metabolic alkalosis.

Therefore there should be respiratory compensation with reduced respiratory rate giving rise to a high pCO_2 and low pO_2.

However, this is not the picture and we see a type 1 respiratory failure with low pO_2 and low pCO_2.

The results therefore suggest a mixed respiratory and metabolic alkalosis.

This picture could be found in association with lung disease and prolonged vomiting

[D]

129/ A 22-year-old man presented to the Emergency Department with dyspnoea.

He had no past history of note and had previously been fit and well.

He reported that he had experienced some upper anterior chest discomfort after climbing the four flights of stairs to his apartment and that this was accompanied by

an unusual degree of breathlessness. The symptoms subsided once he had rested, but occurred again later in the day when he climbed the stairs again.

On examination he appeared well and was not dyspnoeic at rest. Heart sounds were normal. An additional clicking noise was heard in the left fourth intercostal space; this sound occurred with each heart beat.

What is the most likely cause for his pain?

(Please select 1 option)

○	Acute pericarditis
○	Aortic dissection
○	Pulmonary embolism
○	Spontaneous pneumothorax
○	Unstable angina

Young adult males, often tall and slim, are frequently affected by spontaneous pneumothorax.

Clinical features include

Sudden onset of chest pain, sometimes radiating to the shoulder
Dyspnoea (may not be a dominant feature)
Dry cough.

Left-sided pneumothoraces may be associated with a clicking sound synchronous with the heart-beat and may occasionally be audible to the patient

[D]

130/ A 15-year-old girl presented to the Emergency department with difficulty breathing. She was a known asthmatic who had seen her general practitioner the previous day with a sore throat.

He had diagnosed tonsillitis and had prescribed a five day course of oral amoxicillin. The patient had been diagnosed with ulcerative colitis three years previously. Her regular medication included inhaled salbutamol and beclomethasone and mesalazine 400 mg tds.

On examination she was alert and oriented but was in considerable distress with laboured breathing. Inspiratory wheeze was noted. She was pale, sweaty and cyanosed. Her temperature was 36.5ºC, pulse 120/minute and regular, blood pressure 90/35 mmHg. The remainder of the examination was normal.

She was given high-flow oxygen through a face mask but despite this her breathing became increasingly difficult.

What antibiotic treatment should be given?

(Please select 1 option)

○	Amoxicillin + clarithromycin
○	Amoxicillin + clavulanic acid
○	Benzyl penicillin
○	Cefotaxime
○	Flucloxacillin

The most likely diagnosis is acute epiglottitis.

Sudden airway obstruction may occur and it is vital to obtain the assistance of an anaesthetist urgently. No attempt should be made to visualise the epiglottis until an anaesthetist is present as there is a high risk of causing acute airway obstruction by touching the inflammed tissue.

The diagnosis may be confirmed on direct visualisation of a cherry-red epiglottis. Early intubation is essential, especially in cases where there is respiratory distress. Adult epiglottitis is much less common but has a higher mortality.

A significant number of strains are resistant to ampicillin and a third generation cephalosporin is the treatment of choice

[D]

131/ A 15-year-old girl presented to the Emergency department with difficulty breathing.

She was a known asthmatic who had seen her general practitioner the previous day with a sore throat. He had diagnosed tonsillitis and had prescribed a 5 day course of oral amoxicillin. The patient had been diagnosed with ulcerative colitis three years previously. Her regular medication included inhaled salbutamol and beclomethasone and mesalazine 400 mg tds.

On examination she was alert and oriented but was in considerable distress with laboured breathing. Inspiratory wheeze was noted. She was pale, sweaty and cyanosed. Her temperature was 36.5°C, pulse 120/minute and regular, blood pressure 90/35 mmHg. The lungs were clear and the remainder of the examination was normal.

She was given high-flow oxygen through a face mask but despite this her breathing became increasingly difficult.

What is the most likely pathogen?

(Please select 1 option)

	Corynebacterium diphtheriae
	Haemophilus influenzae
	Peptostreptococcus
	Staphylococcus aureus
	Streptococcus pneumoniae

The most likely diagnosis is acute epiglottitis.

Sudden airway obstruction may occur and it is vital to obtain the assistance of an anaesthetist urgently. No attempt should be made to visualise the epiglottis until an anaesthetist is present as there is a high risk of causing acute airway obstruction by touching the inflamed tissue.

The diagnosis may be confirmed on direct visualisation of a cherry-red epiglottis.

Early intubation is essential, especially in cases where there is respiratory distress. Adult epiglottitis is much less common but has a higher mortality.

The usual causative organism is *Haemophilus influenzae*type b.

[B]

132/ A 15-year-old girl presented to the Emergency department with difficulty breathing.

She was a known asthmatic who had seen her general practitioner the previous day with a sore throat. He had diagnosed tonsillitis and had prescribed a five day course of oral amoxicillin. The patient had been diagnosed with ulcerative colitis three years previously. Her regular medication included inhaled salbutamol and beclomethasone and mesalazine 400 mg tds.

On examination she was alert and oriented but was in considerable distress with laboured breathing. Inspiratory wheeze was noted. She was pale, sweaty and cyanosed. Her temperature was 39°C, pulse 120/minute and regular, blood pressure 90/35 mmHg. The lungs were clear and the remainder of the examination was normal.

She was given high-flow oxygen through a face mask but despite this her breathing became increasingly difficult.

What immediate treatment is required?

(Please select 1 option)

○	Endotracheal intubation
○	Hydrocortisone
○	Increased concentration of inspired oxygen
○	Intravenous benzyl penicillin
○	Nebulised bronchodilators

The most likely diagnosis is acute epiglottitis.

Sudden airway obstruction may occur and it is vital to obtain the assistance of an anaesthetist urgently. No attempt should be made to visualise the epiglottis until an anaesthetist is present, as there is a high risk of causing acute airway obstruction by touching the inflamed tissue.

The diagnosis may be confirmed on direct visualisation of a cherry-red epiglottis.

Early intubation is essential, especially in cases where there is respiratory distress.

Adult epiglottitis is much less common but has a higher mortality.

[A]

133/ A 39-year-old woman presented to the casualty department giving a two week history of palpitations and breathlessness. She had a past history of diabetes mellitus, well controlled on metformin 850 mg bd. She was also being treated for longstanding hypertension for which she had been on therapy for several years.

Her current medications comprised: captopril 50 mg bd; furosemide 40 mg od; and nifedipine 20 mg bd. She had recently consulted her GP with the symptoms of breathlessness and he had increased the dose of furosemide to 80 mg od.

On examination she was overweight and appeared distressed. She was afebrile. Her pulse 120, regular; BP 145/95 mmHg. Heart sounds 1 and 2 normal with no added sounds or murmurs. Respiratory rate 28/minute; chest clear to auscultation. The rest of the examination was normal.

Investigations revealed:

Hb	13.4 g/dL	(11.5-16.5)

WBC	8.9 $\times 10^9$/L	(4-11)
Platelets	199 $\times 10^9$/L	(150-400)
Sodium	139 mmol/L	(137-144)
Potassium	4.4 mmol/L	(3.5-4.9)
Urea	5.8 mmol/L	(2.5-7.5)
Creatinine	110 µmol/L	(60-110)
Glucose	5.9 mmol/L	(3.0-6.0)

Arterial blood gases on air:

pH	7.6	(7.36-7.44)
O_2 Saturation	99%	
PaO_2	112 mmHg/15 kPa	(75-100)
$PaCO_2$	13.7 mmHg/1.8 kPa	(35-45)
Standard bicarbonate	20 mmol/L	(20-28)
Base excess	-7.0 mmol/L	(±2)

Chest x ray: Normal

What treatment should be given?

(Please select 2 options)

☐	Aminophylline infusion
☐	Breathe into a paper bag
☐	Inspired oxygen (24%)
☐	Intravenous hydrocortisone
☐	Intravenous normal saline
☐	Intravenous salbutamol
☐	Nebulised bronchodilators
☐	Oral amoxicillin + clarithromycin

☐	Reassurance
☐	Stop metformin

The patient has a respiratory alkalosis as a result of hyperventilation.

[H and H]

134/ A 28-year-old plumber was referred to hospital by his general practitioner.

He had initially presented seven days previously, giving a three day history of malaise, headache, and myalgia and subsequently developed a dry cough and fever. His GP had started a course of amoxicillin/clavulanic acid, but the symptoms failed to resolve. On the day of referral, the patient continued to complain of cough and had become mildly dyspnoic; he also complained of a global headache, myalgia and arthralgia.

On examination, he appeared unwell and was febrile (39°C). A maculopapular rash was evident over his upper body. Heart sounds were normal; BP 120/70 mmHg. On auscultation of his chest, fine crackles were audible in the left mid-zone. Mild neck stiffness was noted.

Investigations showed:

Hb	8.4 g/dL (13.0-18.0)
WBC	8 $x10^9$/L (4-11 $x10^9$)
Platelets	210 $x10^9$/L (150-400 $x10^9$)
Reticulocytes	8% (0.5-2.4)
Na	129 mmol/L (137-144)
K	4.2 mmol/L (3.5-4.9)
Urea	5.0 mmol/L (2.5-7.5)
Creatinine	110 µmol/L (60-110)
Bilirubin	19 µmol/L (1-22)
Alk phos	130 U/L (45-105)
AST	54 U/L (1-31)
GGT	48 U/L (<50)

Chest x ray shows patchy consolidation in both mid-zones.

What is the most appropriate course of treatment?

(Please select 1 option)

	Cefotaxime
	Cefuroxime
	Ciprofloxacin
	Clarithromycin
	Co-trimoxazole

Mycoplasma pneumoniae most commonly causes disease in individuals aged 15-30 years.

This patient has a pneumonia associated with mild hepatitis and possible haemolytic anaemia.

Mycoplasma pneumonia presents with systemic upset, dry cough and fever. Myalgia and arthralgia are common. The WBC is often within the normal range.

Extrapulmonary manifestations of *Mycoplasma* occur in up to 10% of cases of *Mycoplasma* pneumonia. These include

Haemolytic anaemia
Renal failure
Hepatitis
Myocarditis
Meningism and meningitis
Transverse myelitis
Cerebellar ataxia.

Cutaneous manifestations include erythema multiforme.

Haemolysis is associated with the presence of cold agglutinins, found in up to 50% of cases of *Mycoplasma* pneumonia. Diagnosis is based on demonstration of anti-*Mycoplasma*antibodies in paired sera.

First choice treatment is with macrolide antibiotic(clarithromycin or erythromycin); alternatives include tetracycline or doxycycline

[D]

135/ A 28-year-old plumber was referred to hospital by his general practitioner.

He had initially presented seven days previously, giving a three day history of malaise, headache, and myalgia and subsequently developed a dry cough and fever.

His GP had started a course of amoxicillin/clavulinic acid, but the symptoms failed to resolve. On the day of referral, the patient continued to complain of cough and had become mildly dyspnoic; he also complained of a global headache, myalgia and arthralgia.

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Alk phos	130 U/L	(45-105)
AST	54 U/L	(1-31)
GGT	48 U/L	(<50)

Chest x ray shows patchy consolidation in both mid-zones.

What two investigations should be performed to confirm the cause of his abnormal blood count?

(Please select 2 options)

☐	Blood cultures

☐	Blood film
☐	Bone marrow aspirate
☐	Direct Coombs' test
☐	Hepatitis serology
☐	Lewis blood group
☐	Liver biopsy
☐	Paul-Bunnell test
☐	Serum electrophoresis
☐	Unconjugated bilirubin

The presentation is typical of *Mycoplasma* pneumonia: pneumonia associated with mild hepatitis and haemolytic anaemia.

Extrapulmonary manifestations occur in ~10% of cases:

Haemolytic anaemia
Renal failure
Hepatitis
Myocarditis
Meningism
Meningitis
Transverse myelitis
Cerebellar ataxia.

Cutaneous manifestations include erythema multiforme.

Haemolysis is associated with the presence of IgM antibodies (cold agglutinins) directed against the I antigen of the erythrocyte membrane. Haemolysis would be confirmed on a blood film and a direct Coombs' test.

Unconjugated bilirubin would support haemolysis, but is not a diagnostic test.

Infectious mononucleosis may also present with haemolytic anaemia.

However, the presentation is not consistent with acute Epstein-Barr virus

[B and D]

136/ A 28-year-old plumber was referred to hospital by his general practitioner.

He had initially presented seven days previously, giving a three day history of malaise, headache, and myalgia and subsequently developed a dry cough and fever.

His GP had started a course of amoxicillin/clavulanic acid, but the symptoms failed to resolve. On the day of referral, the patient continued to complain of cough and had become mildly dyspnoic; he also complained of a global headache, myalgia and arthralgia.

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Alk phos	130 U/L	(45-105)
AST	54 U/L	(1-31)
GGT	48 U/L	(<50)

Chest x ray shows patchy consolidation in both mid-zones

What is the most likely cause of his abnormal blood count?

(Please select 1 option)

○	Clavulanic acid
○	Glucose-6- phosphate dehydrogenase deficiency
○	IgG directed against P antigen complex

	IgM anti-i antibodies
	Sepsis syndrome

This patient has a pneumonia associated with mild hepatitis and haemolytic anaemia.

Mycoplasma pneumonia presents with systemic upset, dry cough, fever, myalgia and arthralgia. Extrapulmonary manifestations occur in ~10% of cases of *Mycoplasma* pneumonia.

These include:

Haemolytic anaemia
Renal failure
Hepatitis
Myocarditis
Meningism and meningitis
Transverse myelitis, and
Cerebellar ataxia.

Cutaneous manifestations include erythema multiforme.

Haemolysis is associated with the presence of IgM antibodies (cold agglutinins) directed against the I antigen of the erythrocyte membrane.

Sepsis may be associated with a microangiopathic haemolytic anaemia.

IgG antibodies directed against the P antigen complex are the cause of paroxysmal nocturnal haemoglobinuria.

Clavulanic acid causes hepatitis.

Haemolysis can be induced by some drugs in patients with G6PD deficiency (primaquine, dapsone, sulfamethoxazole, nitrofurantoin, doxorubicin).

[D]

137/ 28-year-old plumber was referred to hospital by his general practitioner.

He had initially presented seven days previously giving a three day history of malaise, headache, and myalgia and subsequently developed a dry cough and fever. His GP had started a course of amoxicillin/clavulinic acid, but the symptoms failed to resolve. On the day of referral, the patient continued to complain of cough and had become mildly dyspnoic; he also complained of a global headache, myalgia and arthralgia.

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Bilirubin	19 µmol/L	(1-22)
Alk phos	130 U/L	(45-105)
AST	54 U/L	(1-31)
GGT	48 IU/L	(<50)

Chest x ray shows patchy consolidation in both mid-zones.

What is the most likely cause of his symptoms?

(Please select 1 option)

○	Epstein-Barr virus
○	*Haemophilus influenzae*
○	*Klebsiella pneumoniae*
○	*Mycoplasma pneumoniae*
○	*Pneumocystis carinii*

Mycoplasma pneumoniae most commonly causes disease in individuals aged 15-30 years.

This patient has a pneumonia associated with mild hepatitis and possible haemolytic anaemia. *Mycoplasma* pneumonia presents with systemic upset, dry cough and

fever. Myalgia and arthralgia are common. The WBC is often within the normal range.

Extrapulmonary manifestations of *Mycoplasma* occur in up to 10% of cases of *Mycoplasma* pneumonia.

These include:

Haemolytic anaemia
Renal failure
Hepatitis
Myocarditis
Meningism and meningitis
Transverse myelitis, and
Cerebellar ataxia.

Cutaneous manifestations include erythema multiforme.

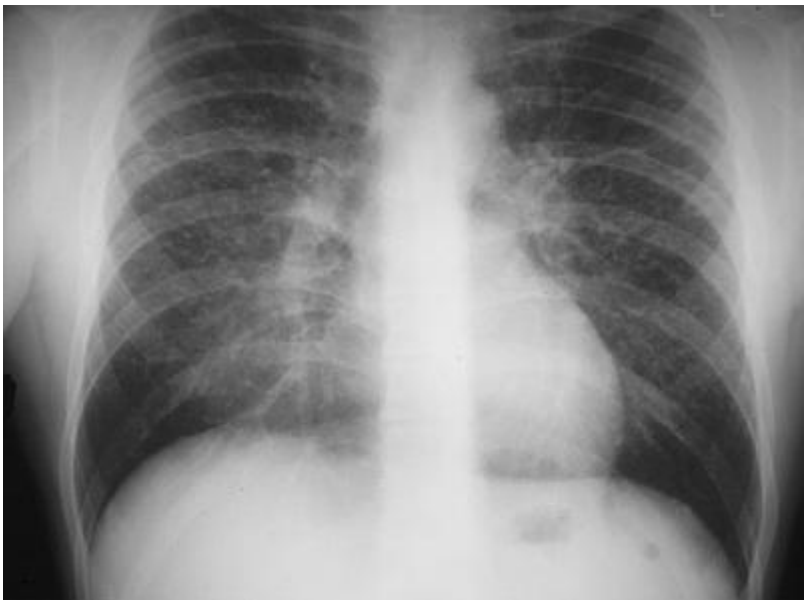
Haemolysis is associated with the presence of cold agglutinins, found in up to 50% of cases of *Mycoplasma* pneumonia. Diagnosis is based on demonstration of anti-mycoplasma antibodies in paired sera.

[D]

138/ A 24-year-old Irishman presented with a ten week history of progressively worsening exertional dyspnoea and a dry cough. He was otherwise well in himself.

Auscultation of his chest revealed fine inspiratory crackles to the mid-zones. He was afebrile.

His chest radiograph is shown below. A Heaf test was negative.



Which of the following investigations will be most helpful in establishing a diagnosis?

(Please select 3 options)

☐	Abdominal ultrasound scan
☐	Atypical serology
☐	Bone marrow aspiration
☐	Bronchoalveolar lavage
☐	CD4 T lymphocyte count
☐	C reactive protein (CRP)
☐	Cytoplasmic antineutrophil cytoplasmic antibody (cANCA)
☐	Erythrocyte sedimentation rate (ESR)
☐	HIV antibody test
☐	Peak flow rate
☐	Plasma lactate dehydrogenase (LDH)
☐	Sputum culture
☐	Transbronchial lung biopsy
☐	Trial of steroids
☐	24 hour urinary calcium excretion

The chest x ray shows bilateral reticulonodular shadowing and bilateral hilar lymphadenopathy. The most likely diagnosis is sarcoidosis.

Most cases of sarcoid present between 20 and 40 years of age with a slightly increased incidence in women.

Prevalence varies amongst different ethnic populations; in Europe, sarcoid is commonest amongst Caucasians - and has a significantly higher incidence in the Irish.

A definitive diagnosis can only be made with biopsy evidence of a non-caseating granulomatous inflammatory process. Supportive evidence of sarcoid is provided by:

Elevated serum angiotensin converting enzyme (ACE) levels
Elevated 24 hour urine calcium excretion
Increased uptake on gallium 67 scanning.

Elevated inflammatory markers (ESR, CRP) are non-specific. Elevated serum calcium, though classical of sarcoid, is actually relatively rare.

The main differential diagnosis in this case is HIV disease. Hilar adenopathy is common in HIV disease and CXR abnormalities are common.

Pneumocystis carinii pneumonia (PCP) frequently presents with a protracted history of worsening dyspnoea and dry cough with reticular shadowing on the CXR.

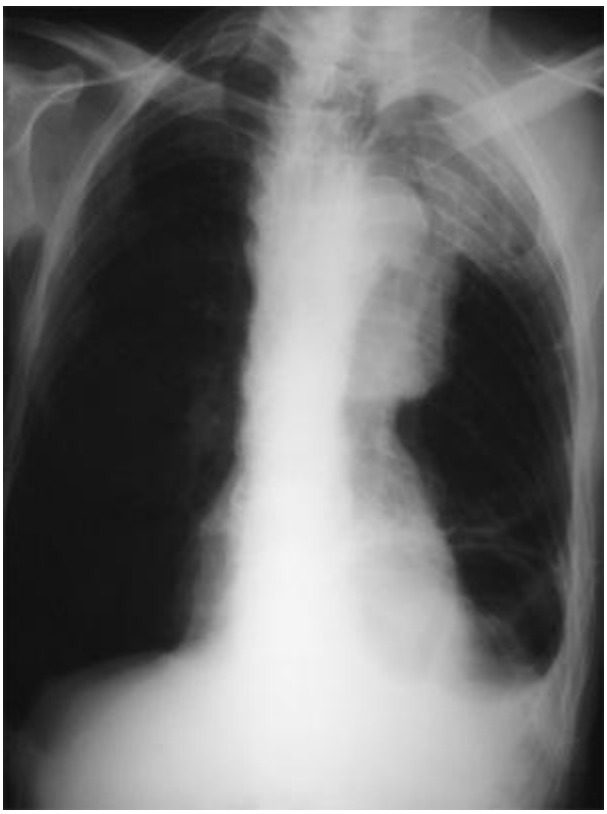
Lymphopenia is a common feature in both sarcoidosis and HIV. In this setting an HIV test is essential.

CD4 counts cannot be used as a surrogate test of HIV infection since CD4 counts can be within the normal range in HIV seropositive individuals and is an ethically questionable means of trying to establish a diagnosis for which there is a definitive test.

[I,M and O]

139/ An 82-year-old man presents with weight loss (5 kg) and a hoarse voice of two months duration.

His chest radiograph is shown below.



What clinical signs are likely to be found?

(Please select 1 option)

○	Dilated left pupil
○	Inability to blink left eyelid

	Inability to sweat on the right upper body
	Left ptosis
	Right-sided exophthalmos

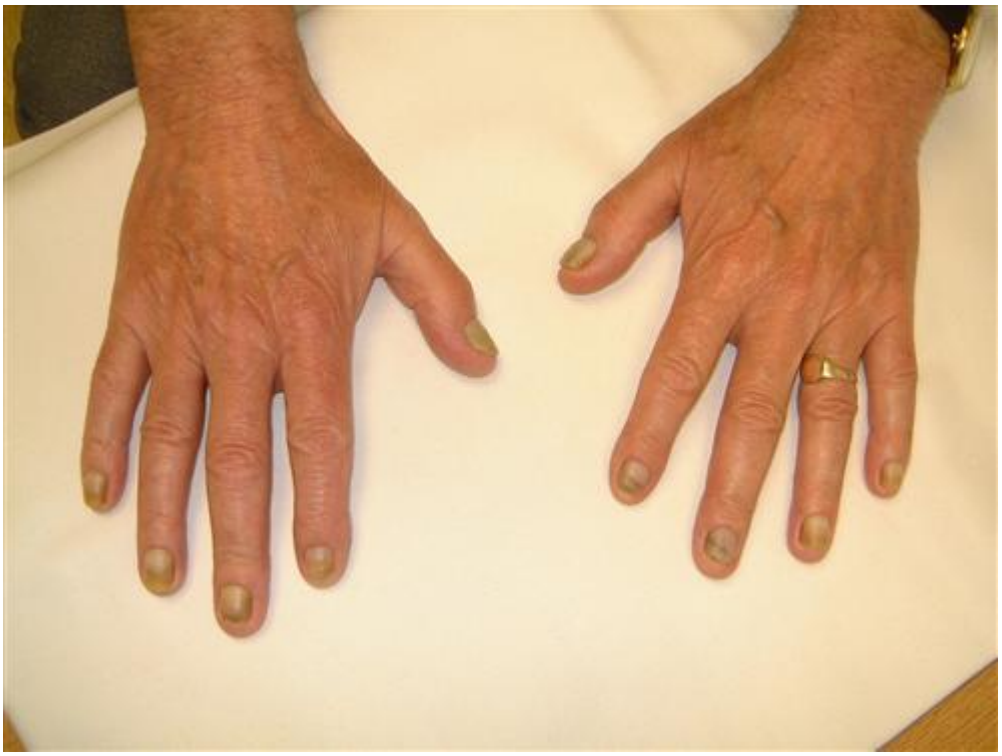
The chest x ray shows left upper lobar consolidation; the history of hoarseness implies involvement of the recurrent laryngeal nerve - most likely due to invasion by tumour.

Upper lobar malignancies involving the superior pulmonary sulcus can destroy surrounding structures leading to a characteristic clinical pattern - Pancoast's syndrome. The syndrome consists of pain in a C8-T2 distribution (caused by infiltration of these nerves) often accompanied by radiological evidence of destruction of the first and second ribs.

Horner's syndrome frequently co-exists due to infiltration of the sympathetic trunk. Horner's syndrome consists of enophthalmos, ptosis, miosis and ipsilateral loss of the ability to sweat

[D]

139/ This patient presented with a six month history of increasing dyspnoea and swollen legs.



What is the diagnosis?

(Please select 1 option)

	Bacterial endocarditis

	Chronic renal failure
	Congestive cardiac failure
	Hypoalbuminaemia
	Yellow nail syndrome

Yellow nail syndrome is caused by hypoplastic lymphatics and is characterised by the triad of lymphoedema, pleural effusions and yellow discolouration of the nails.

Approximately 40% of patients also have bronchiectasis.

Hypoalbuminaemia is associated with white nails (leukonychia)
Chronic renal failure may be associated with brown discolouration of the nails
Bacterial endocarditis is associated with finger clubbing, splinter haemorrhages and nail-fold infarcts

[E]

142/ A 21-year-old woman presented with a four week history of increasing exertional dyspnoea and a dry cough.

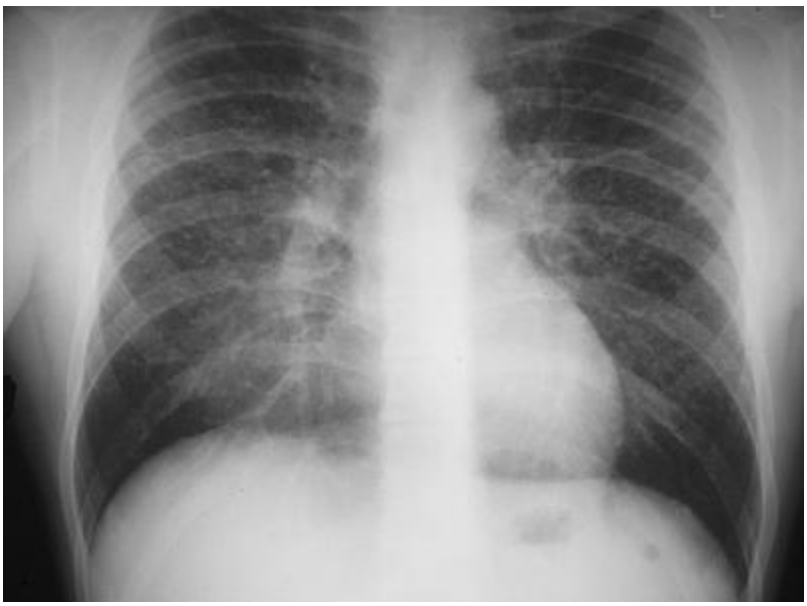
Her chest was clear to auscultation and she was afebrile.

Her chest radiograph is shown below.

Investigations show:

Hb	13.2 g/dL	(11.5-16.5)
WBC	3.9 $\times 10^9$/L	(4-11)
Neutrophils	2.5 $\times 10^9$/L	(1.5-7)
Lymphocytes	1.0 $\times 10^9$/L	(1.5-4)
Monocytes	0.4 $\times 10^9$/L	(0-0.8)
Platelets	390 $\times 10^9$/L	(150-400)
Sodium	141 mmol/L	(137-144)
Potassium	4.7 mmol/L	(3.5-4.9)
Urea	5.5 mmol/L	(2.5-7.5)
Creatinine	102 µmol/L	(60-110)
Calcium	2.6 mmol/L	(2.2-2.6)
Bilirubin	8 µmol/L	(1-22)

Alkaline phosphatase	110 U/L	(45-105)
AST	22 U/L	(1-31)
Total protein	55 g/L	(61-76)
Globulin	31 g/L	



What is the most likely diagnosis?

(Please select 1 option)

	Chlamydia pneumoniae pneumonia
	Lymphoma
	Pneumocystis carinii pneumonia
	Sarcoidosis
	Wegener's granulomatosis

The chest x ray shows bilateral reticulonodular shadowing and bilateral hilar lymphadenopathy. There is lymphopenia, hyperglobulinaemia and hypercalcaemia.

Calculation of corrected calcium:

Add 0.1 mmol/l of calcium for every 4 g/dl that the albumin level is below 40 g/dl.

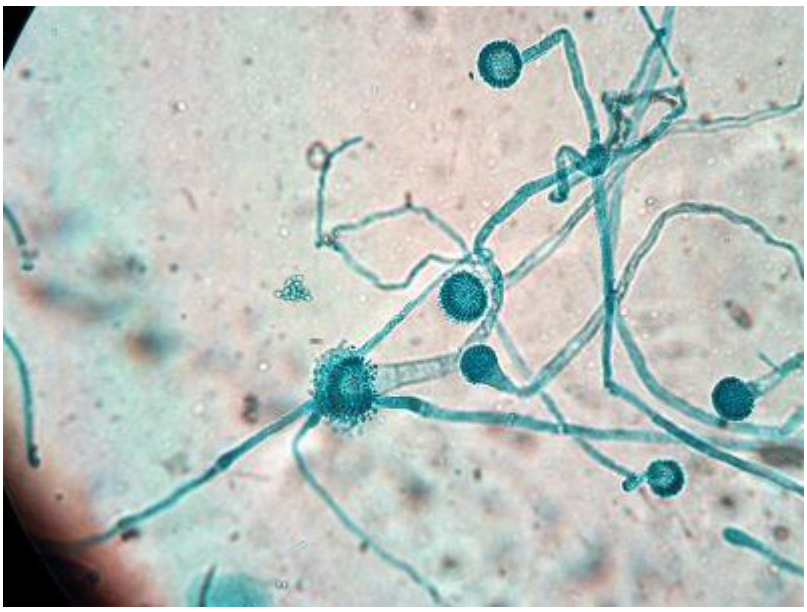
Albumin + globulin	= Total protein
Albumin	= Total protein globulin
	= 55 - 31

	= 24
Corrected calcium	= 2.6 + (((40-24)/4) x 0.1)
	= 2.6 + ((16/4) x 0.1)
	= 2.6 + (4 x 0.1)
	= 2.6 + 0.4
	= 3.0 mmol/l

[D]

143/ The structure shown below was identified on microscopy of a sputum sample from a patient who presented with haemoptysis. He has an abnormal chest radiograph.

What treatment should be started?



(Please select 1 option)

○	Intravenous amoxicillin/clavulanic acid + clarithromycin
○	Intravenous amphotericin B
○	Intravenous cefotaxime
○	Intravenous vancomycin
○	Isoniazid + rifampicin + ethambutol + pyrazinamide

The slide shows the typical morphology of *Aspergillus fumigatus*.

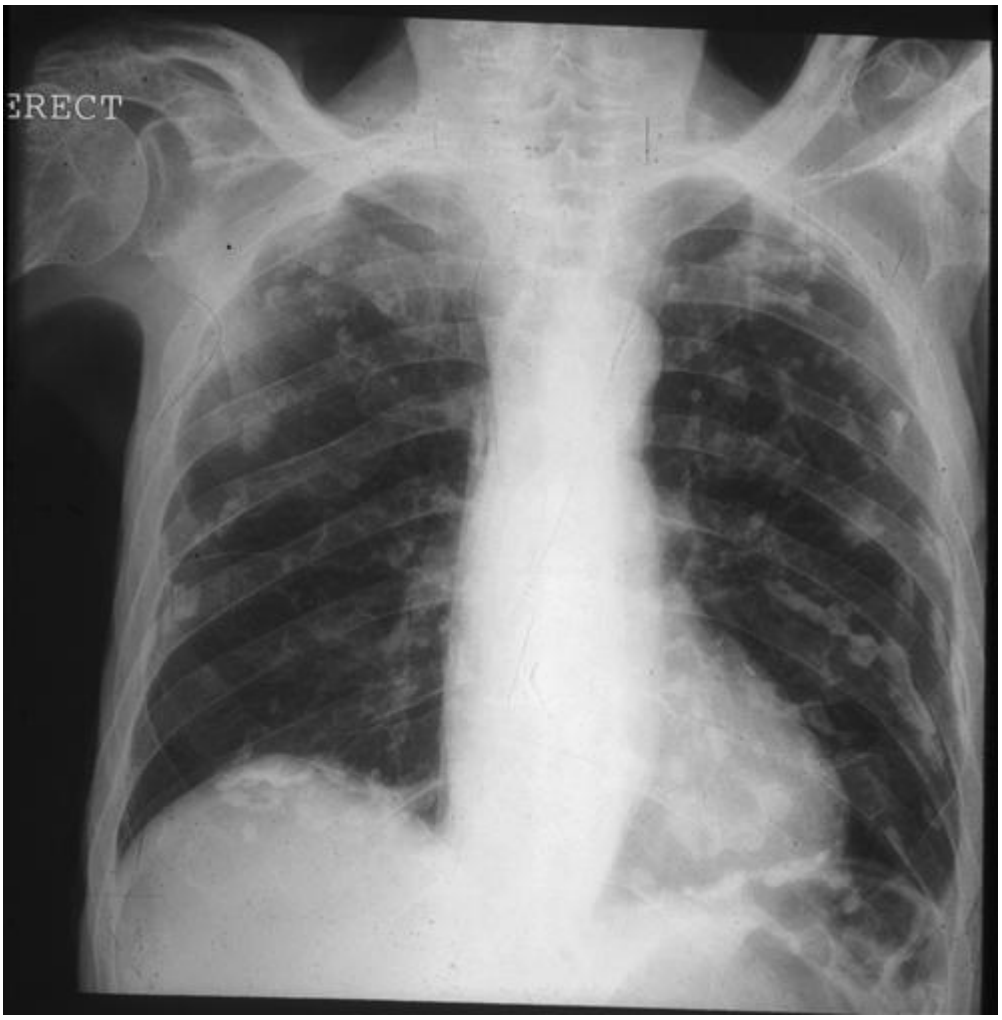
The patient will need treatment with amphotericin.

[B]

144/ A 53-year-old engineer presents with increasing shortness of breath on exertion.

Physical examination is normal apart from some inspiratory crackles at both lung bases.

His chest x ray is shown.



What is the diagnosis?

(Please select 1 option)

○	Asbestosis
○	Byssinosis

	Siderosis
	Silicosis
	Talcosis

The x ray shows extensive pleural plaques and the clinical history would suggest previous exposure to asbestos.

Disease	Agent	Effects
Aluminosis	Alum, and al. oxide	Fibrosis, bullae, pneumothorax
Asbestosis	Asbestos	Pleural plaques, lung cancer, mesothelioma
Byssinosis	Cotton, flax, hemp	Airway obstruction, loss of elasticity
Metal fume fever	Cadmium, cobalt, nickel, zinc and others	Chemical pneumonitis
Occupational asthma	Western Red Cedar and others	Reversible airway obstruction
Siderosis	Iron oxide	Dust deposits
Silicosis	Silica	Dust deposits and fibrosis
Talcosis	Talc, hydrated Mg. silicates	Perivascular fibrosis

[A]

146/ A 48-year-old male accountant is referred from his general practitioner with a three month history of dry, nocturnal cough.

He is an ex-smoker having given up five years ago. He does not produce any sputum, has not suffered with any haemoptysis and despite his steady weight has an exercise tolerance similar to his work colleagues.

He denies any other symptoms of note. Examination reveals he is 5' 10" (1.77m) tall and weighs 98kg (BMI = 31 kg/m^2). Chest is clear to auscultation.

Results of spirometry are shown below:

FEV_1	3.0 L	(Predicted 3.38 L)
FVC	4.4 L	(Predicted 4.40 L)
FEV_1/FVC	0.68	(Predicted 0.77)

PEFR	540 L/min	(Predicted 559 L/min)

What would be the most appropriate first line investigation?

(Please select 1 option)

	24 Hour oesophageal pH and manometry
	Bronchoscopy
	Flexible nasendoscopy
	Peak flow chart
	Sleep studies

Three common causes to consider with nocturnal dry cough are: asthma, reflux and post nasal drip.

The clue here is the obstructive picture on spirometry (FEV1/FVC ratio <70%) - which immediately excludes reflux and post nasal drip (there is no reason for these conditions to have abnormal presentation on spirometry). Effectively excluding oesophageal manometry and nasendoscopy from the options available.

If this were a case of obstructive sleep apnoea, one would expect a restrictive defect secondary to obesity, hence excluding sleep studies as a useful entity.

Bronchoscopy looking for a bronchial carcinoma for instance which may also present with an obstructive defect, is a viable option but there is nothing in the history which points to a diagnosis of malignancy in this man and as a first line investigation bronchoscopy compared to maintianing a peak flow chart is an highly invasive investigation.

A variation of greater than 25% on a peak flow chart (pre and post bronchodilator) would support an initial diagnosis of reversible small airways disease, such as asthma.

[D]

147/ The following arterial blood gases (ABGs) were taken from an unconscious 45-year-old man in the Emergency department:

pH	7.36	(7.36-7.44)
pO_2	13.0 kPa	(11.3-12.6)
pCO_2	3.7 kPa	(4.7-6.0)
HCO_3^-	15 mmol/l	(20-28)

Which is the correct interpretation of the ABG result?

(Please select 1 option)

○	Compensated metabolic acidosis
○	Compensated metabolic alkalosis
○	Compensated respiratory acidosis
○	Compensated respiratory alkalosis
○	Delayed analysis of ABG sample

The pH and bicarbonate results suggest that there is an acidosis. In a respiratory acidosis one would expect the carbon dioxide to be high. In this case the CO_2 is low, favouring metabolic acidosis.

Some degree of respiratory compensation of the acidosis is seen. Typically, a patient's respiratory rate increases with increased respiratory effort. This allows some of the excess H^+ to be excreted as CO_2 helping to prevent large variations in pH.

$H^+ + HCO_3^- \rightarrow H_2O + CO_2$

This deep and rapid breathing can lead to oxygen levels which are high or high-normal. An alternative explanation for the high oxygen levels here is that the patient may be receiving oxygen therapy in the Emergency department.

Delayed analysis of an ABG sample can result in a spuriously low pO_2. There is no reason to suspect this has occurred in this case.

[A]

148/ A 62-year-old man attends the Emergency department because of progressively worsening dyspnoea. He also gives a history of dry cough and a low-grade fever.

He has a past history of hypertension, and was hospitalised six months previously when he suffered an acute inferior myocardial infarction that was complicated by left ventricular failure and arrythmia. His chest x ray shows a diffuse interstitial pneumonia.

Other investigations are shown below.

ESR	110 mm/h
FEV1	90%
FVC	70%
KCO	60%

Which of the following agents is most likely to have caused these findings?

(Please select 1 option)

	Amiodarone
	Captopril
	Procainamide
	Propranolol
	Verapamil

The side effects of amiodarone are well recognised, and include pneumonitis and pulmonary fibrosis.

Pneumonitis and lung fibrosis present with

A progressively-worsening dry cough
Pleuritic chest pain
Dyspnoea
Malaise.

Other side-effects of amiodarone include

Neutropenia
Hepatitis
Phototoxicity and slate-grey skin discolouration
Hypothyroidism
Hyperthyroidism
Arrhythmias
Corneal deposits
Peripheral neuropathy
Myopathy

[A]

150/ What organism has been cultured from a sputum sample from a 15-year-old girl with a chronic cough and diarrhoea?

(Please select 1 option)

	Haemophilus influenzae
	Klebsiella pneumoniae
	Mycobacterium tuberculosis
	Pseudomonas aeruginosa
	Staphylococcus aureus

The culture plate shows a growth of *Pseudomonas aeruginosa*, characterised by the green colouration of the colonies due to production of the pigment pyocyanin.

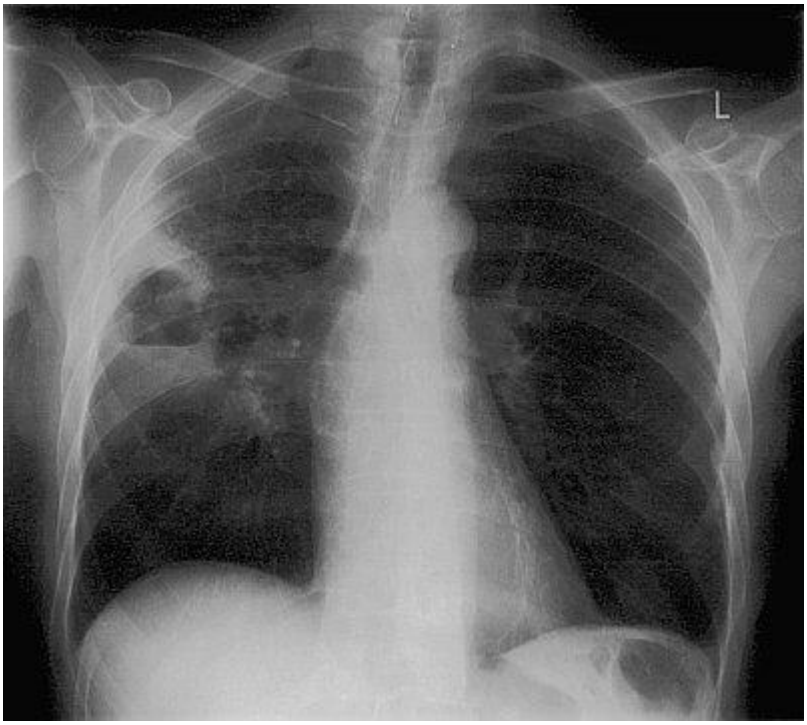
The history is consistent with a diagnosis of cystic fibrosis: bronchiectasis associated with CF frequently results in recurrent infections with *Pseudomonas*

[D]

151/ A 27-year-old man was referred to hospital with fevers and haemoptysis.

Two weeks earlier he had presented to casualty following a grand mal seizure.

Pending the results of sputum cultures, what is the most appropriate combination of antibiotics that should be used to treat this patient initially?



(Please select 1 option)

○	Amoxicillin/clavulanate + clarithromycin
○	Azithromycin
○	Cefuroxime + metronidazole
○	Flucloxacillin
○	Vancomycin + ceftazidime

The slide shows an abscess in the right mid-zone.

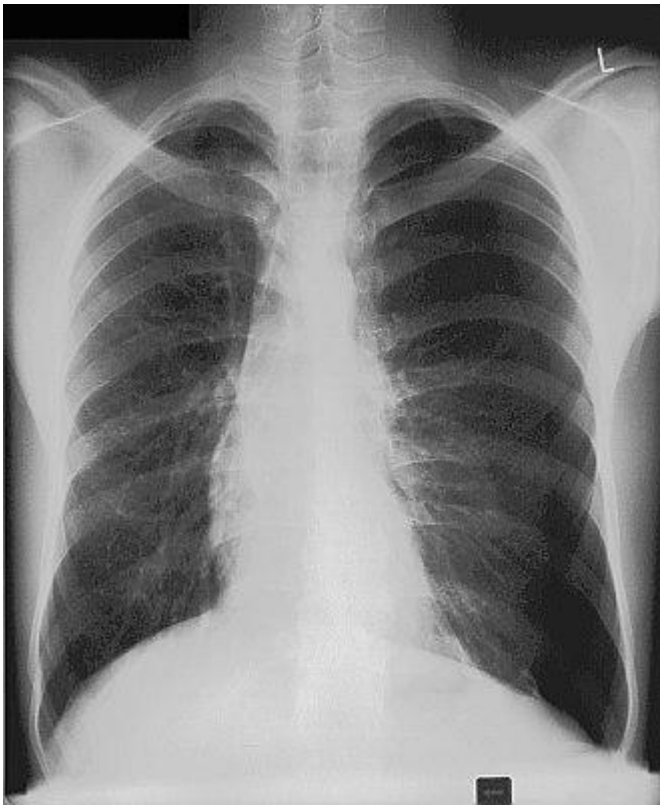
The lung abscess is likely the result of aspiration during the grand mal seizure that occurred several weeks earlier. Due to the angle of the bronchi, the right lung is more commonly affected by aspiration than the left lung.

Classically, when the subject is lying down, aspirated oral or gastric contents enter the apical segment of the right lower lobe.

Anaerobes and Gram negative organisms are the usual organisms in abscesses following aspiration

[C]

152/ A 19-year-old man presented with pleuritic chest pain which occurred suddenly while playing football. He presented to the Emergency department complaining of dyspnoea. His chest x ray is shown.



Which of the following would be the definitive treatment for this condition?

(Please select 1 option)

○	High-flow inspired oxygen
○	Intercostal chest drain insertion
○	Intravenous amoxicillin + clarithromycin
○	Low molecular weight heparin
○	Nebulised salbutamol

The slide shows a large left sided tension pneumothorax.

The left hemithorax is hyperinflated with loss of lung markings peripherally. This is particularly noticeable in the left lower zone. There is also mediastinal shift away from the midline towards the right.

This is a classical presentation of pneumothorax: young fit male (often tall) who develops chest pain and shortness of breath while excercising.

What are the alternatives presented here?

This would be an unusual presentation of PE
There is no history of wheeze to suggest bronchoconstriction
There is no consolidation on the CXR
Nor any suggestion in the history that suggests infection.

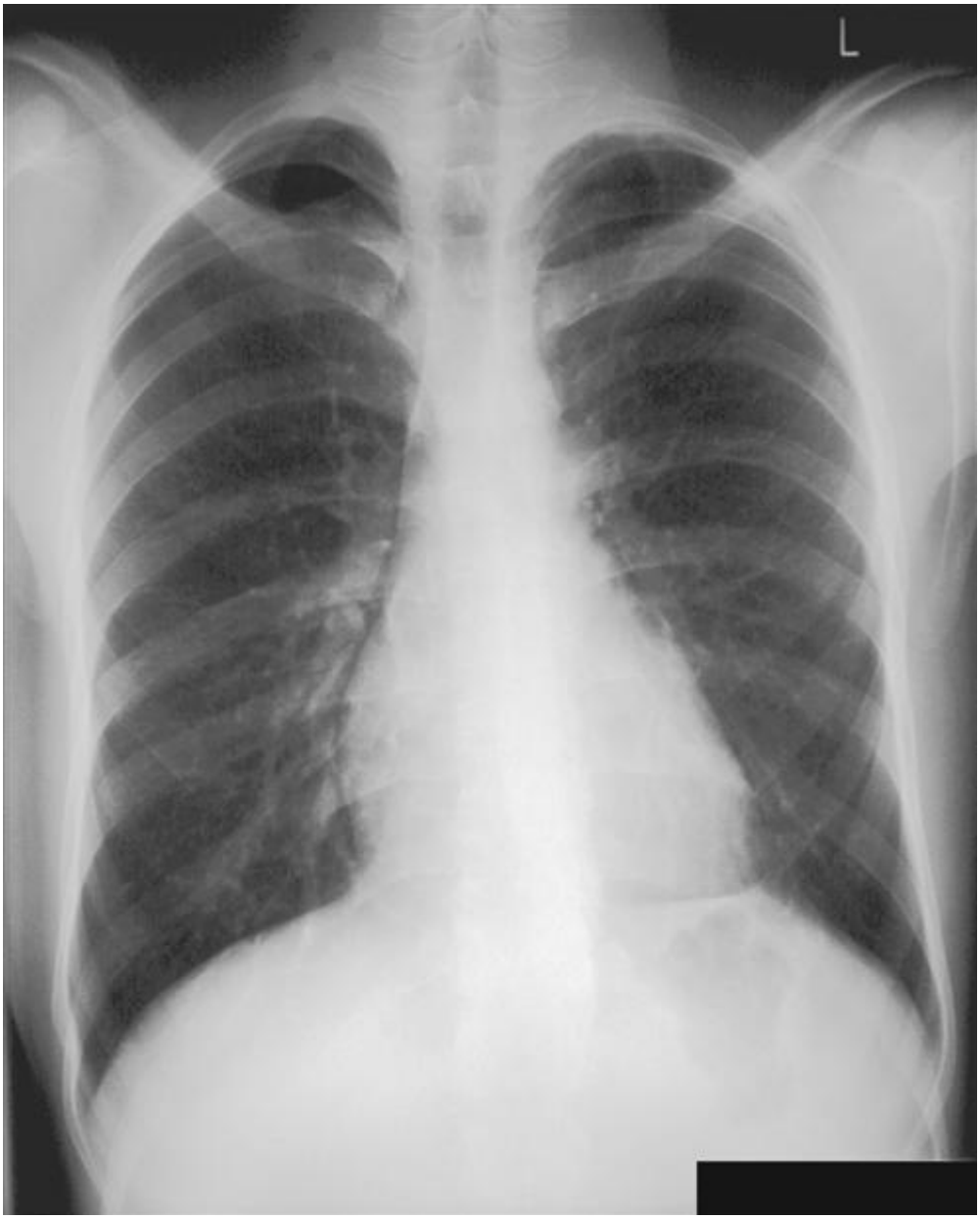
Having ruled these alternatives out, you have two options that might be used to treat pneumothorax (oxygen or chest tube). If you realise that this is a pneumothorax, the question tries to establish whether you are aware of the circumstances in which oxygen alone is adequate versus those in which chest tube insertion is necessary.

Where there is a tension pneumothorax, neither oxygen alone nor needle aspiration would be appropriate and a chest tube must be inserted.

[B]

153/ A 25-year-old man presents with vague chest pain and cough.

His chest x ray, taken in the Emergency department, is shown.



What is the most appropriate treatment for this condition?

(Please select 1 option)

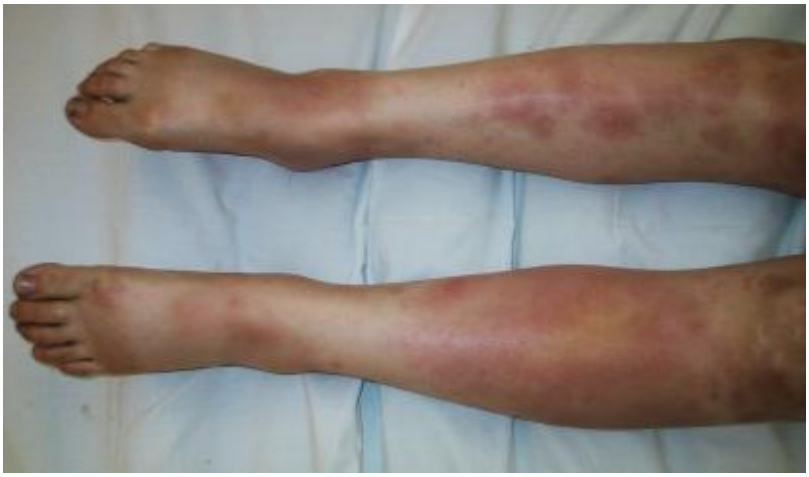
	Amoxicillin/clavulanate and clarithromycin
	High-flow inspired oxygen
	Intercostal chest drain insertion
	Low molecular weight heparin
	Nebulised salbutamol

The slide shows a small right apical pneumothorax.

High-flow inspired oxygen is sufficient treatment; neither aspiration nor chest drain insertion is required.

[B]

154/ Which of the following is the most important in establishing the cause of these lesions?



(Please select 1 option)

	Anti-streptolysin O titre
	Chest x ray
	Erythrocyte sedimentation rate
	Serum angiotensin converting enzyme (ACE)
	Skin biopsy

Erythema nodosum is shown in the slide.

The commonest precipitant is a streptococcal infection.

However, the commonest potentially serious causes (and therefore those that should be excluded first) include sarcoidosis and tuberculosis.

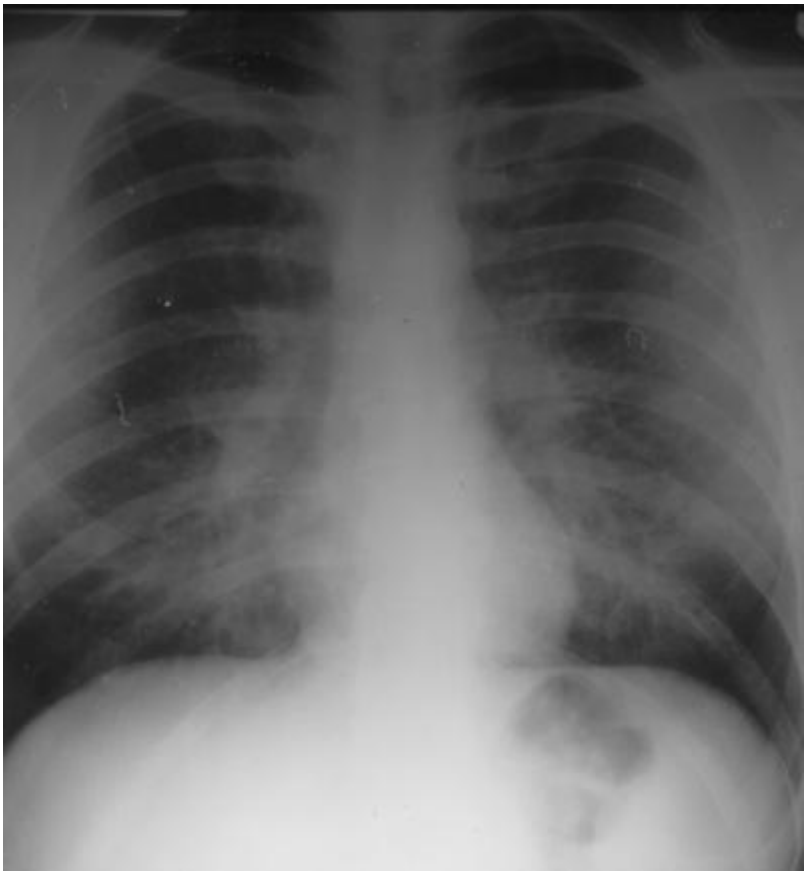
A chest x ray is an important investigation to exclude both of these causes.

[B]

155/ A 32-year-old man presented to hospital with a four week history of progressively worsening dyspnoea on exertion. He also complained of a non-productive cough. Over the two days preceding admission the patient had become breathless at rest and was started on oral co-amoxiclav by his general practitioner.

On examination he was febrile 38°C and looked unwell.*Candida* was noted on the tonsilar pillars. No wheeze or crackles were heard in his chest. His chest radiograph is shown.

Which of the following is most likely to assist in making the diagnosis?



(Please select 1 option)

○	Blood pressure measured in inspiration and expiration
○	*Legionella* urinary antigen
○	Oxygen saturations pre- and post-exercise
○	Peak expiratory flow rate

Sputum culture

The history is characteristic of *Pneumocystis carinii* pneumonia (PCP).

The presence of oropharyngeal candidiasis in a patient without a known immunosuppressive illness is highly suggestive of HIV/AIDS.

Salient features are:

Several days/weeks of increasing dyspnoea
Dry cough
Marked oxygen desaturation with exercise.

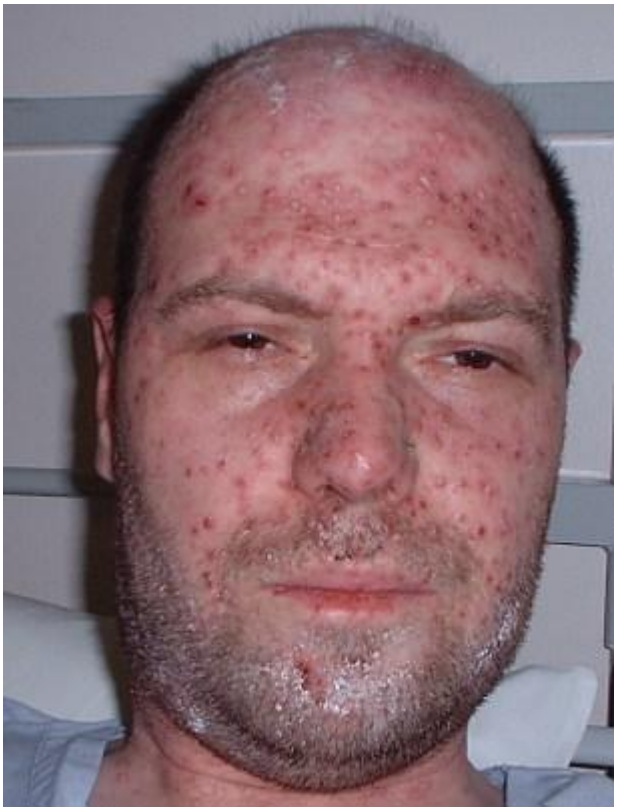
This can be done on an exercise bike, or by asking the patient to do some simple exercise on the ward (sit back and forward in bed several times; squat and stand several times; walk a flight of stairs, etc).

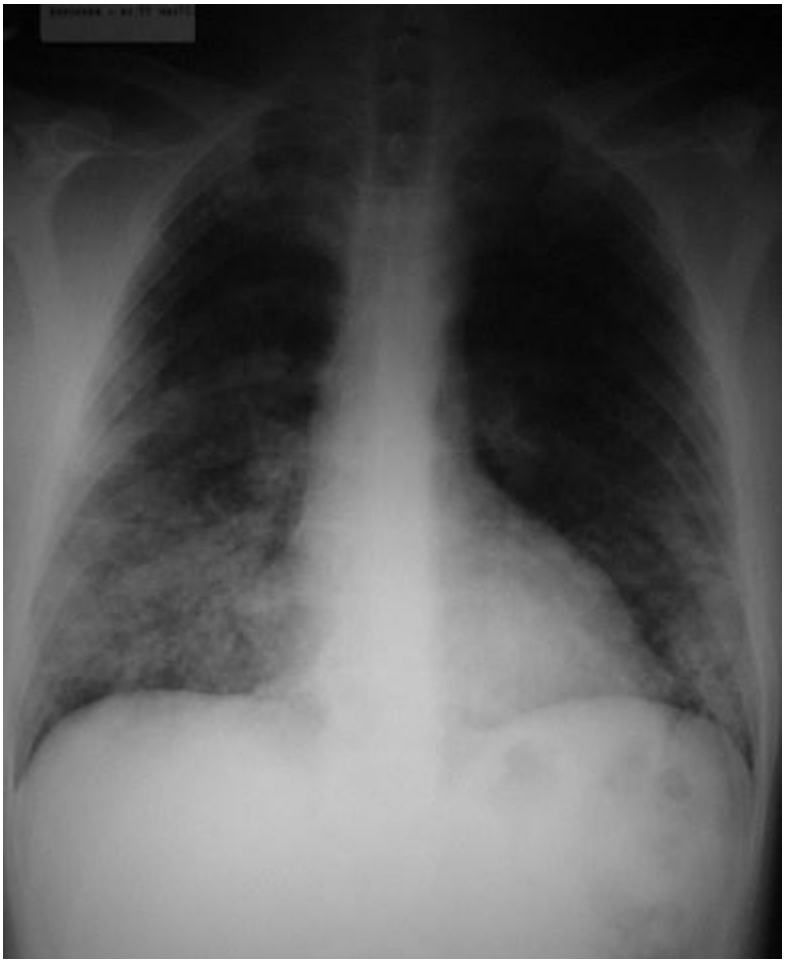
[C]

156/ This 28-year-old man presented to hospital after becoming progressively more breathless over the preceding day.

He had developed a dry cough and reported expectoration of bright red blood. He gave a history of malaise and low-grade fever for five days.

The rash (pictured) had appeared three days before presentation.





What is the most likely diagnosis?

(Please select 1 option)

○	Goodpasture's syndrome
○	Meningococcal septicaemia
○	Tuberculosis
○	Varicella pneumonia
○	Wegener's granulomatosis

The slide shows the typical rash of chickenpox.

Varicella pneumonia occurs in up to 20% of adults with chickenpox, appearing three to five days into the course of the illness.

Symptoms include

Tachypnoea
Cough
Dyspnoea

Fever.

Cyanosis, pleuritic chest pain and haemoptysis are common.

Chest x ray shows patchy shadowing.

About 30 young adults die each year from varicella pneumonia. They are best managed on a high-dependency unit

[D]

157/ A 19-year-old male is brought to casualty after having a generalised seizure in a nightclub.

Some friends, who are accompanying him, state that he has a known history of epilepsy. They also report that he has consumed approximately 10 pints of lager during the evening and that he vomited while having the seizure.

On examination the patient is drowsy but responsive to verbal commands. His clothing is covered in vomitus. He is febrile, 37.5°C. On auscultation of his chest, there are coarse crackles in the right upper and mid zones. His chest radiograph shows diffuse right upper lobar airway shadowing.

Which of the following combinations of antibiotics should be started?

(Please select 1 option)

○	Amoxicillin + metronidazole
○	Ceftazidime + erythromycin
○	Co-trimoxazole
○	Erythromycin + rifampicin
○	Penicillin + gentamicin

This is the only appropriate combination for aspiration pneumonia, which this patient clearly has.

[A]

158/ You are asked advice by a young professional couple, Mr and Mrs X. Mrs X is nine weeks pregnant.

Mr X's brother and his partner had a child with cystic fibrosis. As a result, Mr X was screened and found to carry the DF508 mutation for cystic fibrosis. Mrs X declines to be tested.

What are the chances of Mr and Mrs X's child having cystic fibrosis, given that the gene frequency for this mutation in the general population is 1/20?

(Please select 1 option)

	1/4
	1/20
	1/40
	1/80
	1/160

The chance of Mrs X being a carrier of the gene is 1/20.

The chances of two carriers of a recessive gene having a child that is homozygous for that disease (that is both genes are transmitted to the child) is ¼.

Therefore, the chances of this couple having a child with CF are ¼ × 1/20 = 1/80.

[D]

158/ A 63-year-old man is brought to hospital after being found unconscious in his caravan. He is drowsy but rousable and complains of a severe headache and nausea.

On examination his temperature is 36.5°C but appears flushed. Neck is supple and there is no palpable lymphadenopathy. His BP is 110/65 mmHg. Heart sounds normal with no murmurs or added sounds, and his chest is clear to auscultation. The remainder of the examination is unremarkable.

His son reported that his father, usually a skilled model-maker, had appeared clumsy lately and had been confused at times when talking on the telephone.

Investigations show

Haemoglobin	15.8 g/dL	(13.0-18.0)
White cell count	10.1 $\times 10^9$/L	(4-11)
Platelets	401 $\times 10^9$/L	(150-400)
Serum sodium	140 mmol/L	(137-144)
Serum potassium	4.4 mmol/L	(3.5-4.9)
Serum urea	5.8 mmol/L	(2.5-7.5)

Serum creatinine	110 µmol/L	(60-110)
Serum glucose	4.5 mmol/L	(3.0-6.0)
CSF opening pressure	150 mm H_2O	(50-180)
CSF cell count	<3 mL^{-1}	(≤5)
CSF protein	0.4 g/L	(0.15-0.45)
CSF glucose	3.3 mmol/L	(3.3-4.4)

Arterial blood gases breathing air:

PaO_2	11.6 kPa	(11.3-12.6)
$PaCO_2$	4.3 kPa	(4.7-6.0)
HCO_3	20 mmol/L	(20-28)
pH	7.33	(7.36-7.44)

Based on the information available to you, which investigation would you like to do?

(Please select 1 option)

○	Carboxyhaemoglobin level
○	CT scan head
○	Estimation of carbon monoxide diffusion factor (KCO)
○	Methaemoglobin level
○	Mini mental state examination

The history is suggestive of carbon monoxide (CO) poisoning due to poor ventilation in his caravan.

CO binds with high affinity to haemoglobin, forming carboxyhaemoglobin. CO also binds myoglobin and mitochondrial cytochrome oxidase.

CO poisoning causes tissue hypoxia, anaerobic metabolism and lactic acidosis.

Elevated carboxyhaemoglobin levels document exposure, but do not correlate with severity

[A]

159/ A breathless patient undergoes pulmonary function testing.

The following results are obtained:

FEV1	74% predicted
FVC	68% predicted
TLC	77% predicted
TL_{co}	46% predicted
K_{co}	53% predicted

Which of the following is the most likely cause ?

(Please select 1 option)

	Asthma
	Chronic obstructive pulmonary disease
	Cryptogenic fibrosing alveolitis
	Kyphoscoliosis
	Morbid obesity

The restrictive lung pattern together with the reduced TLCO and KCO suggest lung fibrosis

[C]