Respiratoy Review

- ❖ causes of bilateral Hilar lymphadenopathy are :
- ➢ Sarcodosis [Most common cause]
- ➢ Tuberculosis
- ➢ Lymphoma
- ➢ Pneumoconiosis
- ➢ Beryliosis
- ➢ Fungi : Histoplasmosis,cocidiomycosis

- ❖ Transfer factor raised: asthma, haemorrhage, left-to-right shunts, polycythaemia low: everything else
- ❖ Pulmonary function in obesity:
- ➢ Restrictive pattern
- ➢ No effect on KCO⁾
- ❖ Findings in Obstructive pattern of respiratory disease :
- ➢ FEV1 significantly reduced (less 70%)
- ➢ FVC reduced or normal FEV1/FVC = reduced (less than 80% or 0.7)
- ➢ Raised total lung capacity
- ➢ Raised residual Volume
- ❖ Findings in Restrictive pattern of respiratory disease:
- ➢ FEV1 reduced less than 80%
- ➢ FVC =significantly reduced
- ➢ FEV1/FVC =normal or increased more than 80%
- ➢ Lung compliance , TLC RV are decreased
- ➢
- ❖ Causes of Obstructive pattern :
- ➢ COPD
- ➢ Asthma
- ➢ Bronchiectasis
- ➢ Bronchiolitis obliterans
- ❖ Causes of Restrictive pattern
- ➢ Idiopathic pulmonary fibrosis
- ➢ Pulmonary haemorrhages
- ➢ Asbestosis
- ➢ Sarcodosis
- ➢ ARDS
- ➢ Extrinsic allergic alevolitis
- ➢ Histocytosis (diet-2 2017 part-2)
- ➢ Coal worker's
- ➢ pneumococcal
- ➢ Polio
- ➢ Myasthenia Gravis
- ➢ Obesity
- ➢ Scoliosis
- ❖ Causes of increased TLCO are :
- ➢ Most obstructive has low TLCO except Asthma
- ➢ Pulmonary haemorrhages (Wegener granulomatosis, Goodpasture syndrome)
- ➢ Left to right shunt
- ➢ Polycythemia
- ➢ Exercise
- ➢ Hyperkinetic state
- ❖ Causes of Low TLCO are :
- ➢ All restrictive pattern disease gives low TLCO except pulmonary haemorrhages) Pulmonary fibrosis Pulmonary emboli Emphysema Pulmonary oedema Anaemia Pneumonia Sarcodosis

COPD:

- COPD patient presents with worsening of breathlessness. He is hypoxic, hypercapnic and acidotic→ acute exacerbation of COPD with type 2 respiratory failure.
- Acute exacerbated COPD patient with type-2 decompensated respiratory failure→ Arranged for NIPPV
- Most important risk factor for COPD → Smoking
- What is the most important factor in airflow limitation in severe emphysema? → Loss of elastic recoil
- **The most common infective causes of COPD exacerbations → *Haemophilus influenzae***

COPD: investigation and diagnosis:

- A diagnosis of COPD in patients over 35 years of age who are smokers or ex-smokers and have symptoms such as exertional breathlessness, chronic cough or regular sputum production.
- The following investigations are recommended in patients with suspected COPD:
  - post-bronchodilator spirometry to demonstrate airflow obstruction: FEV1/FVC ratio less than 70%
  - chest x-ray: hyperinflation, bullae, flat hemidiaphragm. Also important to exclude lung cancer
  - full blood count: exclude secondary polycythaemia
  - body mass index (BMI) calculation

The severity of COPD is categorised using the FEV1

Post-bronchodilator FEV1/FVC	FEV1 (of predicted)	Severity
< 0.7	> 80%	Stage 1 - Mild - symptoms should be present to diagnose COPD in these patients
< 0.7	50-79%	Stage 2 - Moderate
< 0.7	30-49%	Stage 3 - Severe
< 0.7	< 30%	Stage 4 - Very severe

- Indications of LTOT in COPD patients

patients with a pO2 of < 7.3 kPa or to those with a pO2 of 7.3 - 8 kPa and one of the following:

- secondary **p**olycythaemia
- **p**eripheral oedema
- **p**ulmonary hypertension

- Patients who receive LTOT should breathe supplementary oxygen for at least 15 hours a day.
- Best indicator for prognosis in COPD is determined by FEV1 [FCPS]
- The most common cause of type 2 respiratory failure is Severe COPD. If this develops then **non-invasive ventilation [NIV]** may be used. typically used for COPD with respiratory acidosis pH 7.25-7.35 **[SBA]** [NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used]

❖ Survival benefits in stable COPD patients are :

➢ **Smoking cessation [SBA]**
➢ LTOT
➢ Lung volume reduction surgery
➢ Steriods reduced exacerbation frequency but not the mortality

Asthma:

❖ Think Asthma →
➢ if symptoms worse at night, early morning
➢ if the symptoms to triggers g. pollen, pets, cold air, perfumes) after taking Aspirin).
➢ presence of **Atopy** (e.g. eczema, hay fever).
➢ think of Asthma if (FEVl/FVC) < 70% (<0.7) and **significantly improves** after giving bronchodilators. **(reversible obstruction; improve post bronchodilator)**

❖ whereas in COPD, it remains < 0.7 even post-bronchodilator.
❖ **Exacerbation most commonly precipitated by viral infection [SBA]**

❖ Drugs that aggravates Asthma:
➢ Beta blocker
➢ Betel nut [arecholine]
➢ Aspirin
➢ NSAIDs
➢ OCP

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 Oxygen sats < 92% Silent chest, cyanosis or feeble respiratory effort Bradycardia, dysrhythmia or hypotension Exhaustion, confusion or coma

In addition, a normal pCO_2 in an acute asthma attack indicates exhaustion and should, therefore, be classified as life-threatening.

Diagnostic testing

Patients >= 17 years

➢ **all patients** should have **spirometry with a bronchodilator reversibility (BDR) test [confirmatory test]**
➢ **all patients** should have a **FeNO test**

Children 5-16 years

➢ all children should have spirometry with a bronchodilator reversibility (BDR) test
➢ a FeNO test should be requested if there is normal spirometry or obstructive spirometry with a negative bronchodilator reversibility (BDR) test

Spirometry

➢ **FEV1/FVC ratio** less than 70% (or below the lower limit of normal if this value is available) **is considered obstructive**

Reversibility testing

- in adults, a positive test is indicated by an improvement in FEV1 of 12% or more and increase in volume of 200 ml or more

Management:

Step	Notes
1 Newly-diagnosed asthma	Short-acting beta agonist (SABA)
2 Not controlled on previous step OR Newly-diagnosed asthma with symptoms >= 3 / week or night-time waking	SABA + low-dose inhaled corticosteroid (ICS)
3	SABA + low-dose ICS + leukotriene receptor antagonist (LTRA)
4	SABA + low-dose ICS + long-acting beta agonist (LABA) Continue LTRA depending on patient's response to LTRA
5	SABA +/- LTRA Switch ICS/LABA for a maintenance and reliever therapy (MART), that includes low-dose ICS
6	SABA +/- LTRA + medium-dose ICS MART OR consider changing back to a fixed-dose of a moderate-dose ICS and a sep LABA
7	SABA +/- LTRA + one of the following options: • increase ICS to high-dose (only as part of a fixed-dose regime, not as a M • a trial of an additional drug (for example, a long-acting muscarinic recept antagonist or theophylline) • seeking advice from a healthcare professional with expertise in asthma

- When to give Magnesium sulphate (MgS04) IV?
- Patient not responding to salbutamol, oxygen and steroids
- **Omalizumab→An IgE Antibody** in patients with **Severe allergic/atopic asthma** when other treatments unsuccessful
- Omalizumab (Side effects : abdominal pain, headache, fever)
- **Eosinophilic** asthma treated by = **Mopolizumab [monoclonal antibody blocks the binding of IL-5 on eosinophil]**

- Aspirin induced asthma → Leukotriene receptor antagonist
- Indication of intubation in asthma: pH less than 7. 35, CO2 retention [PaCO2 > 6 kPa [45mmHg]

Oocupational asthma:

- A Spray painter who is asthmatic feels well when he is on holiday for 3 weeks→ Occupational Asthma.
- **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 [SBA]
- ✓ **example occupations include spray painting and foam moulding using adhesives**
- platinum salts
- soldering flux resin
- glutaraldehyde
- flour
- epoxy resins
- proteolytic enzymes

- Serial measurements of peak expiratory flow are recommended at work and away from work [SBA]
- Treatment: Review work environment, change job.

Pneumonia:

- ❖ rusty sputum= strep pneumonia
- ❖ Current jelly sputum = klebsiella pneumonae
- ❖ commonest cause of Atypical pneumonia
- ➔ Mycoplasma
- ❖ Young age + hemolytic anemia + Pneumonia = Mycoplasma
- ❖ Elderly + Lung disease (COPD, Bronchiectasis) = H. influenza [FCPS]
- ❖ Alcoholic,DM = Klebsiella
- ❖ Klebsiella most commonly causes a cavitating pneumonia in the upper lobes.
- ❖ HIV patient CD4 < 200 + pneumonia → Pneumocystis Jiroveci
- ❖ HIV patient CD4 >200 + pneumonia → Step pneumonae
- ❖ Within 1- 6 months of Organ transplant pt [Renal transplant] + infection / pneumonia → CMV
- ❖ Pneumonia following influenza →Staph aureus
- ❖ IV Drug abuser with H/O flu from 1 week now presents with fever and cough, CXR shows bilateral cavitating pneumonia→ staphylococcal pneumonia.
- ❖ Pneumonia followed by pneumothorax . most likely organism ? → Staph aureus
- ❖ Most common cause of CAP = strep. Pneumoniae [FCPS]
- ❖ Most common cause HAP(Hospital Acquired Pneumonia) = pseudomonas aeroginosa [FCPS]
- ❖ COPD respiratory failure patient 2days after admission in ITU developed productive cough and yellow greenish sputum. CXR shows right, middle and lower lobe pneumonia →Pseudomonas pneumonia**(suspected with in 48 hours after admission)**
- ❖ hotel,hospital, industry related,AC pneumonia → Legionella(diarrhoea,**hyponatremia,** confusion, proteinuria, hematuria) [MRCP] [FCPS]
- ❖ Legionella pneumophilia is best diagnosed by the urinary antigen test .
- ❖ Older pt + pneumonia + GBS = Chlamydia pneumonia
- ❖ young pt + pneumonia + GBS+ erythma multiforme + erythema nodusum + hemolytic anemia+ cold agglutinin positive (Coomb's test positive) = Mycoplasma pneumonia [FCPS]
- ❖ most common cause of pneumonia in cystic fibrosis = pseudomonas aeroginosa
- ❖ contact birds + pneumonia = chlamydia psittaci
- ❖ farmer, contact with cattle, sheep, cow + pneumonia = Coxiella burnetti
- ❖ strep pneumoniae causes reactivation of Herpes simplex virus results in herpes labialis(cold sore) pneumonia + cold sore (herpes labialis) →strep pneumoniae
- ❖ Recent foreign travel raises the possibility of infections that may otherwise be unusual in the UK, e.g. MERS-Coronavirus (Middle East) melioidosis caused by Burkholderia pseudomallei

Symptoms:

- A cough with purulent sputum (rust coloured/bloodstained)
- Dyspnoea
- Chest pain (may be pleuritic in nature)
- High Fever
- Malaise

Signs:

- Signs of systemic infection: High temperature, tachycardia, hypotension, confusion
- Tachypnoea
- On auscultation, there may be **reduced breath sounds, bronchial breathing, and crepitations/crackles**
- Dullness on percussion (fluid)

Assessment /prognosis of pneumonia patient: CURB65

Criterion	Marker
C	Confusion (abbreviated mental test score <= 8/10)
U	urea > 7 mmol/L
R	Respiration rate >= 30/min
B	Blood pressure: systolic <= 90 mmHg and/or diastolic <= 60 mmHg
65	Aged >= 65 years

- CURB65 score of 0 or 1 - low risk→ consider home-based care
- CURB65 score of 2 or more - intermediate risk → consider hospital-based care
- CURB65 score of 3 or more - high risk → consider intensive care assessment

Management

- Low severity: first line → Amoxicillin
- Moderate and high severity → dual antibiotic therapy is recommended with amoxicillin and a macrolide
- All cases of pneumonia should have a repeat *chest X-ray at 6 weeks after clinical resolution* to ensure that the consolidation has resolved and there is no underlying secondary abnormalities (e.g. a lung tumour).

Sarcoidosis:

- **Most common cause of bilateral hilar lymphadenopathy [SBA]**
- Afrocarribean + hypercalcaemia /bilateral hilar lymphadenopathy / Erythma nodusum/ Arthralgia→ **think sarcoidosis**
- Young black woman presents with a 3 month history of joint pains, Dyspnea, dry cough, pleuritic chest pain and weight loss. Red nodules over lower legs present → **Sarcoidosis.**
- Erythema nodosum is associated with a good prognosis in sarcodosis [SBA]
- Lupus pernio → bad prognostic [SBA]
- Diagnostic investigation of choice/ confirmtory for sarcodosis is → **Trans bronchial lung biopsy [SBA]**
- **The majority of patents with sarcodosis get better without treatment [SBA]**

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by **non-caseating granulomas.**
It is more common in young adults and in people of African descent

Features

- acute: erythema nodosum, bilateral hilar lymphadenopathy, swinging fever, polyarthralgia
- insidious: dyspnoea, non-productive cough, malaise, weight loss
- skin: lupus pernio
- **Hypercalcaemia:** macrophages inside the granulomas cause an increased conversion of vitamin D to its active form (1,25-dihydroxycholecalciferol) [SBA]

Sarcoidosis: investigation

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

- Diagnostic investigation of choice/ confirmtory for sarcodosis is → **Trans bronchial lung biopsy [SBA]**
- ACE levels not reliable in the diagnosis of sarcoidosis although they may have a role in monitoring disease activity. [SBA]
- Routine bloods may show **hypercalcaemia and a raised ESR**

Other investigations

- spirometry: may show a **restrictive defect**
- tissue biopsy: **non-caseating granulomas**
- **the Kveim test** is no longer performed due to concerns about cross-infection

Sarcoidosis: management

- **The majority of patents with sarcodosis get better without treatment [SBA]**

Indications for steroids [SBA]

- patients with chest x-ray **stage 2 or 3 disease who are symptomatic.**
- **hypercalcaemia**
- **eye, heart or neuro involvement**

Factors associated with poor prognosis:

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

Lung cancer:

- Age > 40 + chronic smoker + chronic cough + weight loss + SOB + **Clubbing** → Suspect Lung cancer

- Patient presented with sudden right sided weakness and CXR shows mass at hilum→most likely brain metastasis → immediately start dexamethasone→ do CT brain contrast .
- **Most common malignant tumor of lung is metastatic carcinoma. Colorectal cancer is the commonest.** Diagnosed by percutaneous CT guided biopsy.
- Patient of bronchogenic carcinoma presents with swelling of face ,chest and upper limbs→SVC obstruction.
- Most common symptoms → cough
- Overall Most common Lung cancer is : Adenocarcinoma **[FCPS]**
- **most common type of lung cancer in non-smokers → adenocarcinoma [SBA]**
- Most important risk factor for Lung cancer → Smoking

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

Factors that are NOT related

- **coal dust**

Features

- Persistent cough
- haemoptysis
- dyspnoea
- chest pain
- weight loss and anorexia
- **hoarseness**

- **seen with Pancoast tumours pressing on the recurrent laryngeal nerve [**CT scan of Chest is diagnostic for Pancoast tumour]

- superior vena cava syndrome

Examination findings

- a fixed, monophonic wheeze may be noted
- supraclavicular lymphadenopathy or persistent cervical lymphadenopathy
- clubbing

Paraneoplastic features:

Small cell	Squamous cell	Adenocarcinoma
• ADH • ACTH - not typical, hypertension, hyperglycaemia, hypokalaemia, alkalosis and muscle weakness are more common than buffalo hump etc • Lambert-Eaton syndrome	• parathyroid hormone-related protein (PTH-rp) secretion causing hypercalcaemia • clubbing • hypertrophic pulmonary osteoarthropathy (HPOA) • hyperthyroidism due to ectopic TSH	• gynaecomastia • hypertrophic pulmonary osteoarthropathy (HPOA)

Lung cancer: investigation

Chest x-ray

- this is often the first investigation done in patients with suspected lung cancer

CT

- is the investigation of choice to investigate suspected lung cancer

Bronchoscopy

- **this allows a biopsy to be taken** to obtain a histological diagnosis sometimes aided by endobronchial ultrasound

NSCLC Management

Surgical resection:

- Lobectomy (with hilar and mediastinal lymph node resection/sampling) is first-line treatment for those with stage I or II cancer who are medically fit for surgery

Radiotherapy:

- **Is first-line for those with stage I-III disease who are not suitable for surgery.**

Chemotherapy:

- Is offered to those with stage III or IV disease to improve survival and quality of life.

SCLC: Chemotherapy is the first line Rx

Idiopathic Pulmonary Fibrosis:

- Male + age 50-70 yo + **Progressively increasing breathlessness, Dry cough, B/L clubbing, inspiratory crackles.** CT chest showing reticular ground glass opacification at basal area. Biopsy shows intraalveolar macrophages and interstitial fibrosis→ **Idiopathic pulmonary fibrosis.** More common in males.
- **Carbon Monoxide transfer factor determine prognosis in Idiopathic pulmonary fibrosis.**

- IPF is a chronic lung condition characterised by progressive fibrosis of the interstitium of the lungs.
- IPF is typically seen in patients aged 50-70 years and is twice as common in men.

Features

- progressive exertional dyspnoea
- bibasal fine end-inspiratory crepitations on auscultation
- dry cough
- clubbing

Diagnosis

- **Spirometry:** classically a **restrictive picture** (FEV1 normal/decreased, FVC decreased, FEV1/FVC increased)
- **Impaired gas exchange**: reduced transfer factor (TLCO)
- imaging: bilateral interstitial shadowing (typically small, irregular, peripheral opacities - 'ground-glass' - later progressing to 'honeycombing') may be seen on a **chest x-ray**
- high-resolution CT scanning is the investigation of choice and required to make a diagnosis of IPF

Management

- pulmonary rehabilitation
- 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

- causes of upper zone fibrosis:

CHARTS

- C - Coal worker's pneumoconiosis
- H - Histiocytosis/ hypersensitivity pneumonitis
- A - Ankylosing spondylitis
- R - Radiation
- T - Tuberculosis
- S - Silicosis/sarcoidosis

- Lower zone fibrosis occur in

- **ACID**
- **A→ Asbestosis**
- **C→ All connective tissue disease except Ankylosing spondiliti, Cryptogenic fibrosing alveolitis.**
- **I→ Idiopathic pulmonary fibrosis**
- **D→ Drugs**

Drugs causing lung fibrosis

- **amiodarone**
- cytotoxic agents: busulphan, **bleomycin**
- anti-rheumatoid drugs: **methotrexate,** sulfasalazine, gold
- **nitrofurantoin**
- **ergot-derived** dopamine receptor agonists **(bromocriptine, cabergoline, pergolide)**

Bronchiectasis:

- Chronic Persistent Cough +Copious Sputum +Recurrent respiratory tract infections+ **Coarse crepitation** on auscultation → Suspect **B**ronchiectasis
- 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 [Most common],** measles, **pertussis/whooping cough, pneumonia**

- **cystic fibrosis**
- bronchial obstruction e.g. lung cancer/foreign body
- immune deficiency: **selective IgA, hypogammaglobulinaemia**
- allergic bronchopulmonary aspergillosis **(ABPA)**
- ciliary dyskinetic syndromes: **Kartagener's syndrome,** Young's syndrome
- yellow nail syndrome

Features

- Persistent productive cough. Large volumes of sputum may be expectorated
- dyspnoea
- haemoptysis

Signs

- abnormal chest auscultation
- ✓ **coarse crackles**
- ✓ wheeze
- clubbing may be present

Investigation:

- CXR : Trameline , Ring shadow…
- Investigation of choice → HRCT [SBA]

Bronchiectasis: management

- physical training (e.g. inspiratory muscle training)
- postural drainage→ is the best techbique to decrease the frequency of bronchiectasis [SBA[
- antibiotics for exacerbations + long-term rotating antibiotics in severe cases
- bronchodilators in selected cases
- immunisations
- surgery in selected cases (e.g. Localised disease)

Most common organisms isolated from patients with bronchiectasis:

- **Haemophilus influenzae (most common) [SBA]**
- Pseudomonas aeruginosa
- Klebsiella spp.
- Streptococcus pneumoniae

❖ Yellow discoloration of nails + lymphedema +pleural effusion + bronchiectasis is → yellow nail syndrome

Kartagener's syndrome

Features

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

Lung abscess:

❖ severe cough + Fever with chills and rigors + sweating + weight loss + Clubbing + chest pain + foul smelling

sputum + haemoptysis is = Lung abscess

- labatatory findings in lung abscess is : High ESR Sputum for gram statin Leucocytosis

Pneumothorax:

- **Sudden history of pleuretic chest pain**, breathlessness and **hemodianamically compromised** and cyanosed patient with reduced breath sounds and deviation of trachea to opposite side→ tension pneumothorax (insert a large bore needle into right second ICS).
- If history and examination suggestive of pneumothorax and stable next step confirm with chest x-ray
- For recurrent pneumothorax next investigation is CT scan if not then video assisted thoracoscopy
- Management of choice for second unilateral Pneumothorax in fit individual is referral for bullectomy and pleuroectomy
- Pentrating injury/surgery + unilateral chest pain + dyspnoea + shock + tachycardia **+ hypotension + shifting of trachea or mediastinum is** → tension Pneumothorax

- Treatment of tension pneumothorax is : **immediate insertion of wide bore needle in 2nd ICS [SBA]** →Intrathoracic tube in 4,5,6ics in mid axillary line Give oxygen Morphine
- If draining Pneumothorax after 3-5 days there is persistent air leaks(bubbling chest drain) or failure of lung to re-expand→ thoracic surgical opinion

Risk factors

- **pre-existing lung disease: COPD [Most common secondary cause]**, asthma, cystic fibrosis, lung cancer, Pneumocystis pneumonia
- connective tissue disease: Marfan's syndrome, rheumatoid arthritis
- ventilation, including non-invasive ventilation
- **catamenial pneumothorax** is the cause of 3-6% of spontaneous pneumothoraces occurring in menstruating women. It is thought to be caused by endometriosis within the thorax

Symptoms tend to come on suddenly.
Features include:

- dyspnoea
- chest pain: often pleuritic
- sweating
- tachypnoea
- tachycardia

Primary pneumothorax

- if the **rim of air is < 2cm** and the patient **is not short of breath** then discharge should be considered
- **otherwise, aspiration** should be attempted
- **if this fails** (defined as > 2 cm or still short of breath) then a chest drain should be inserted

Secondary pneumothorax

- 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. All patients should be admitted for at least 24 hours
- if the pneumothorax is less the 1cm then the BTS guidelines suggest giving oxygen and admitting for 24 hours

Iatrogenic pneumothorax

- 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

Persistentent / recurrent pneumothorax

If a patient has a persistent air leak or insufficient lung reexpansion despite chest drain insertion, or the patient has recurrent pneumothoraces, then video-assisted thoracoscopic surgery (VATS) should be considered to allow for mechanical/chemical pleurodesis +/- bullectomy.

Sleep apnoea:

- Obese man + tired all time + day time somnolence + apnoea at night reduced REM sleep + snoring + Hypertension + retained C02 is → Obstructive sleep apnoea syndrome (sleep apnoea/hypopnoea)
- **OSA is caused by loss of pharyngeal muscle tone during REM sleep**.

Predisposing factors

- Obesity
- Macroglossia: acromegaly, hypothyroidism, amyloidosis
- Large tonsils
- Marfan's syndrome

The partner often complains of excessive snoring and may report periods of apnoea.

Consequence

- daytime somnolence
- compensated respiratory acidosis
- hypertension

- Neck size is the best predictor of OSA.
- **Gold standard diagnostic test for Obstructive sleep apnoea → Polysomnography**
- Mangement of Obstructive sleep apnoea is :
- **Mild OSA → Weight loss**
- CPAP is first line for moderate and severe OSA
- **If CPAP is not tolerated or for patients with mild OSAHS where there is no daytime sleepiness** = intra-oral devices (e.g. mandibular advancement) may be used

Cystic fibrosis:

- Young patient with Diabetes mellitus + **recurrent chest infections** + Diarrhoea + abnormal LFTs + gallstones + steatorrhoea →Cystic fibrosis
- Oragnisms in Cystic fibrosis patients
- Infants and young children = **Staphylococcus Aureus,** Haemophilus
- **Teenagers = Pseudomonas aeruginosa[SBA]**
- **AR disease**
- Occurs due to CFTR gene mutation
- **CFTR gene encode for Chlorides channel**
- CFTR gene present in **chromosome 7**
- Cystic fibrosis associated with development of DM
- Cystic fibrosis→ pancreatic insufficiency → Malabsorption → steatorrhea, diarrhoea

- Features of cystic fibrosis are :
- Recurrent chest infections (40%)
- malabsorption (30%): steatorrhoea, diarrhoea
- Delayed puberty
- Short stature
- Pancreatic polyps
- Diabetes Mellitus
- Rectal prolapse
- **Male infertility (due to Mal development of vas deferens) [SBA]**
- Female sub infertility
- Meconium ileus
- laboratory of Cystic fibrosis are :
- Sweat test : sweat chloride more than 60mmol/L Decreased chloride secretion
- increased sodium absorption CFTR gene
- **Genetic test is confirmatory**
- Treatment of Cystic fibrosis is :
- Chest physiotherapy (postural drainage)
- **High calorie + high fat Vitamin D**
- Pancreatic enzymes
- Supplement of N- Acetylcystein
- Heart lung transplant

Tuberculosis:

- An Asian immigrant complaining of weight loss, tiredness and hemoptysis

especially in the morning → Pulmonary Tuberculosis.

- Patient with fever, night sweats, weight loss and SOB. CXR shows pleural effusion(low glucose, high lymphocytic count) → tuberculosis.
- Patient presented with weight loss, night sweats, hepatosplenomegaly and clubbing. CBC shows lymphocytosis.CXR shows **miliary shadows** both lung fields. Mantoux test negative. No bacteria grown in sputum culture→ Miliary tuberculosis
-
- Pulmonary tuberculosis is an infectious disease caused by *Mycobacterium tuberculosis*.
- **Isonizide, rifampicin and pyrazinamide all are hepatotoxic.**
- **Most hepatotoxic anti TB drug → Pyrizinamide [SBA]**
- ESRD patient develops pulmonary TB→ ethambutol should be used in a reduced dose. [SBA]
- **Slicosis increases risk of TB**

It is important to remember that TB may present as a primary infection, or as a secondary (postprimary/reactivation) infection.

Primary infection

- Primary infection with TB may be asymptomatic
- Symptomatic primary infection

- fever
- pleuritic or retrosternal pain

Secondary infection (postprimary/reactivation)

Features

- cough
  - ✓ gradually becoming productive
  - ✓ haemoptysis is only seen in a minority
- weight loss
- fatigue
- night sweats
- fever: typically low-grade

Investigation:

- First line → Sputum for Add-Fast Bacilli (AFB) [SBA]
- Gold standard test → Sputum Culture [SBA] can take 1-3 weeks (if using liquid media, longer if solid media)
- Gene Xpert is also diagnostic. [allows rapid diagnosis (within 24-48 hours)]

Screening for latent tuberculosis

- The Mantoux test is the main technique used to screen for latent tuberculosis.
- 0.1 ml of 1:1,000 purified protein derivative (PPD) injected intradermally

- **First line test for TB→ MT test**
- **Inv of choice for latent TB → IGRA**

False negative tests may be caused by:

- miliary TB
- sarcoidosis

- HIV
- lymphoma
- very young age (e.g. < 6 months)

Tuberculosis: management

The standard therapy for treating **active tuberculosis** is:

Initial phase - first 2 months (RIPE)

- Rifampicin
- Isoniazid
- Pyrazinamide
- Ethambutol

Continuation phase - next 4 months

- Rifampicin
- Isoniazid

Indication of Steroid in TB patient:

- Tubercular Meningitis
- Tubercular Pericarditis
- Tubercular pleural effusion
- Adrenal TB **[Absolute indication]**
- **IRIS**

Drug adverse effects

- rifampicin
- potent liver **enzyme inducer**
- **hepatitis**, orange secretions
- flu-like symptoms
- isoniazid
- peripheral neuropathy: prevent with pyridoxine (Vitamin B6)
- **hepatitis,** agranulocytosis
- liver enzyme **inhibitor**
- pyrazinamide
- hyperuricaemia causing gout
- arthralgia, myalgia
- **hepatitis**
- ethambutol
- optic neuritis: check visual acuity before and during treatment

Asbestos related lung disease:

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

1. Pleural plaques

- Pleural plaques are **benign and do not undergo malignant change.** They, therefore don't require any follow-up. [SBA]
- The most common form of asbestos-related lung disease. [SBA]

2. Pleural thickening

3. Asbestosis

- **Ship builder/boiler maker/plumbing industry person with SOB.PFTs are mixed obstructive and restrictive type.CXR shows pleural plaques.O/E clubbing and inspiratory crackles noted→ Asbestosis**
- The severity of asbestosis is related to the length of exposure. This is in contrast to mesothelioma where even

very limited exposure can cause disease. The latent period is typically 15-30 years.

- Asbestosis typically causes lower lobe fibrosis.

Features

- dyspnoea and reduced exercise tolerance
- **clubbing**
- bilateral end-**inspiratory crackles**
- lung function tests show a **restrictive pattern with reduced gas transfer**

- Treatment : It is **treated conservatively** - no interventions offer a significant benefit.

4. Mesothelioma

- Mesothelioma is a malignant disease of the pleura.
- Crocidolite (blue) asbestos is the **most dangerous form**. [MRCP]
- **10-50 years exposure to asbestos/ H/O asbestosis+ progressive Shortness of breadth + chest pain + Pleural effusion (mostly right side)+ clubbing + weight loss is → Mesothelioma**
- **Retired builder of railway carriage is at risk of developing asbestosis .**Time duration of 20- 30 years required .Restrictive patters disease.
- **Demolition worker with pleural effusion and clubbing → Suspect Mesothelioma.**

Possible features

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

- Diagnosis confirmed by pleural biopsy . [SBA]
- Patients are usually offered palliative chemotherapy and there is also a limited role for surgery and radiotherapy.
- Unfortunately, the prognosis is very poor, with a median survival from diagnosis of 8-14 months.

5. Lung cancer

- Whilst mesothelioma is in some ways synonymous with asbestos, lung cancer is actually the most common form of cancer associated with asbestos exposure. It also has a synergistic effect with cigarette smoke in terms of the increased risk. Therefore, smoking cessation is very important as the risk of lung cancer in smokers who have a history of asbestos exposure is very high.

Silicosis:

- Person working in Sand blasting industry (quarts exposure) presents with increasing SOB -- ------- Silicosis.(cause

fibrosis of lung and ultimately corpulmonale)

- Stone mason presented with increasing SOB. CXR shows bilateral hilar lymphadenopathy with egg shell calcification.....Silicosis.
- Stonemasons,fettlers and slate-miners are at risk of developing silicosis.
- Silicosis is a fibrosing lung disease caused by the inhalation of fine particles of crystalline silicon dioxide (silica).
- It is a risk factor for developing **tuberculosis (silica is toxic to macrophages). [SBA]**

Occupations at risk of silicosis

- mining
- slate works
- foundries
- potteries

Features

- upper zone fibrosing lung disease
- 'egg-shell' calcification of the hilar lymph nodes

progressive massive fibrosis.

- Coal miner presents with black sputum and dyspnea so severe that he can't move to bathroom. CXR shows upper lobes massive fibrosis. RF and ANA positive, LFTs shows mixed pattern disease → progressive massive fibrosis.
- Coal workers are predisposed to developing progressive massive fibrosis.

Pulmonary embolism:

- Risk factor for VTE [H/O surgery,long air travel, pregnancy , malignancy, OCP....] + sudden SOB + pleuritic chest pain → suspect PE
- Young patient on OCPs presents with pleuretic chest pain and SOB. → Suspect Pulmonary embolism
- Obese man after a long flight collapsed on airport. He is hypoxic, tachycardiac.CXR shows elevation of diaphragm → pulmonary embolism

Features:

triad

1. pleuritic chest pain,
2. sudden shortness of breath and
3. haemoptysis

other features :

- Low grade fever
- tachypnoea

Risk factor :

Major :

- Malignancy
- DVT

- Varicose vein
- Previous VTE
- Pg

Investigation

1. CXR to exclude other pathology (pneumonia, pneumothorax). CXR usually normal in PE.

2. CTPA first line Investigation / Gold standard [FCPS, MRCP]

3. V/Q scan (if renal failure, allergy to contrast, pg present) [FCPS, MRCP]

4. D dimer (positive does not confirm PE but negative result exclude PE)

5. ECG :

- Sinus Tachycardia [most common findings] (FCPS, MRCP)
- S1Q3T3
- Right axis deviation
- Right Bundle Branch Block (RBBB)

Diagnosis :

If a PE is suspected a 2-level PE Wells score should be performed:

Well's Score:

>4 [More than 4] → PE is likely

4 or <4 [4 or less than 4] → PE unlikely

Management:

Sign, symptoms point toward PE → Start anticoagulation [DOACs] and request CTPA [MRCP]

1. Non massive :

DOACs as first line treatment [MRCP] [SBA]

2. Massive PE with Hypotension (PE with hypotension)

→ Thrombolysis (alteplase) [MRCP][SBA]

4. Patients who have **repeat pulmonary embolisms, despite adequate anticoagulation**, may be considered for **inferior vena cava (IVC) filters.**

Pulmonary Hypertension:

- Pulmonary hypertension may be defined as a sustained elevation in mean pulmonary arterial pressure of greater than 25 mmHg at rest. [SBA]
- **Endothelin** thought to play a key role in pathogenesis of PAH. [SBA]
- It is **more common in females** and typically presents between the **ages of 30-50 years.**
- **Around 10% of cases** are inherited in **an autosomal dominant fashion.**

Causes:

1. Pulmonary arterial hypertension (PAH)

- Idiopathic
- familial

- associated conditions: collagen vascular disease, congenital heart disease with systemic to pulmonary shunts, **HIV** , drugs and toxins, **sickle cell disease**
- persistent pulmonary hypertension of the newborn

2. Pulmonary hypertension with left heart disease

- left-sided atrial, ventricular or valvular disease such as left ventricular systolic and diastolic dysfunction, mitral stenosis and mitral regurgitation

3. Pulmonary hypertension secondary to lung disease/hypoxia

- COPD
- interstitial lung disease
- sleep apnoea
- high altitude

4. Pulmonary hypertension due to thromboembolic disease
5. Miscellaneous conditions
   lymphangiomatosis e.g. secondary to carcinomatosis or sarcoidosis

Features

- progressive exertional dyspnoea is the **classical presentation**
- other possible features include exertional syncope, exertional chest pain and peripheral oedema
- **cyanosis**
- **right ventricular heave, loud P2, raised JVP with prominent 'a' waves, tricuspid regurgitation**

Investigation:

- investigation of choice for pulmonary Arterial Hypertension is →Right Heart catheterization [SBA]

Management:

- Management should first involve treating any underlying conditions.
- Following this, it has now been shown that **acute vasodilator testing** is central to deciding on the appropriate management strategy.

If there is a positive response to acute vasodilator testing (a minority of patients)

- **oral calcium channel blockers**

If there is a negative response to acute vasodilator testing (the vast majority of patients)

- **prostacyclin analogues:** treprostinil, iloprost
- **endothelin receptor antagonists:** bosentan, ambrisentan
- **phosphodiesterase inhibitors:** sildenafil

ABPA:

- Allergic bronchopulmonary aspergillosis results from an allergy to ***Aspergillus spores.***
- Asthma + **proximal** bronchiectasis + eosinophilia + raised IgE → ABPA

- **Investigation most specific to ABPA – positive skin prick test for aspergillus fumigatus. [SBA]**
- **Flitting CXR changes, Positive RAAST for aspergillus, Positive IgG precipitants**
- **First line Treatment is prednisolone [SBA]**
- **2nd line → Itracnazole**

Aspergilloma:

- Woman with previous history of pulmonary T.B now complain of hemoptysis off/on from 3 months .CXR shows old T.B cavity containing mass surround by a crescent halo→ Aspergilloma .
- Diagnosed by sputum culture or by serum precipitating antibodies .
- Treated st choice] or long term itraconazole.
- 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:

1. cough
2. haemoptysis (may be severe)

- **Investigations:**

1) chest x-ray containing a rounded opacity
2) **high titers Aspergillus precipitins**

Chrug Strauss syndrome:

- **Asthma + eosinophilia + nerve lesion(ulnar nerve palsy with foot drop)** + high IgE + sinusitis + **PANCA** is = Chrug Strauss syndrome
- **Asthmatic** patient presenting with rash ,abdominal pain ,diarrhea and **foot drop** episodes…… Churg- Strauss syndrome

Eosinophilic granulomatosis with polyangiitis (EGPA) is now the preferred term for **Churg-Strauss syndrome**. It is an ANCA associated small-medium vessel vasculitis.

Features

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

- Leukotriene receptor antagonists [montelukast] may precipitate the disease.
- Treatment of Chrug Strauss syndrome is : Steroids, Immunosuppressant's : cyclophosamide, azithopurine.

Granulomatosis with polyangiitis

Granulomatosis with polyangiitis is now the preferred term for Wegener's granulomatosis. It is an autoimmune condition associated with a necrotizing granulomatous vasculitis, affecting both the upper and lower respiratory tract as well as the kidneys.

Features

- upper respiratory tract: epistaxis, sinusitis, nasal crusting
- lower respiratory tract: dyspnoea, haemoptysis
- rapidly progressive glomerulonephritis ('pauci-immune', 80% of patients)
- saddle-shape nose deformity
- also: vasculitic rash, eye involvement (e.g. proptosis), cranial nerve lesions

Investigations

- cANCA positive in > 90%, pANCA positive in 25%
- chest x-ray: wide variety of presentations, including cavitating lesions
- renal biopsy: epithelial crescents in Bowman's capsule

Management

- steroids
- cyclophosphamide (90% response)
- plasma exchange

Extrinsic allergic alevolitis:

- Pigeons bird fanciers = avian proteins
- Farmers lung = Saccharopolyspora rectivirguls
- Malt workers lung: = Aspergillus clavatus
- Mushroom workers lung: = thermophilic
- actinomycetes byssinosis = textile industrial cotton hemp dust
- bagassosis = sugarcane
- Spirometry in extrinsic allergic alevolitis is mixed
- Cotton worker presents with chest tightness ang gradually worsening SOB……..Byssinosis[due to endotoxin produced by bacteria in cotton].

Extrinsic allergic alveolitis (EAA, **also known as hypersensitivity pneumonitis**) is 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 from bird droppings
- farmers lung: spores of Saccharopolyspora rectivirgula from wet hay (formerly Micropolyspora faeni)
- malt workers' lung: Aspergillus clavatus

- mushroom workers' lung: thermophilic actinomycetes*

Presentation

- ❖ acute (occurs 4-8 hrs after exposure)
- ✓ dyspnoea
- ✓ dry cough
- ✓ fever
- ❖ chronic (occurs weeks-months after exposure)
- ✓ lethargy
- ✓ dyspnoea
- ✓ productive cough
- ✓ anorexia and weight loss

Investigation

- imaging: upper/mid-zone fibrosis
- bronchoalveolar lavage: lymphocytosis
- serologic assays for specific IgG antibodies
- blood: NO eosinophilia

Management

- avoid precipitating factors
- oral glucocorticoids

Rheumatology:

Rheumatoid arthritis: respiratory manifestations A variety of respiratory problems may be seen in patients with rheumatoid arthritis:

- pulmonary fibrosis
- pleural effusion
- pulmonary nodules
- bronchiolitis obliterans
- complications of drug therapy e.g. methotrexate pneumonitis
- pleurisy
- Caplan's syndrome - massive fibrotic nodules with occupational coal dust exposure
- infection (possibly atypical) secondary to immunosuppression

- Rheumatoid arthritis patient on methotrexate presented with mild clubbing and inspiratory crackles → **methotrexate pneumonitis**
- Rheumatoid arthritis patient on methotrexate has patchy consolidation with bilateral small pleural effusions (CXR), patchy ground glass opacities with bronchial dilatation (CT thorax) and PFTs restrictive pattern → **Bronchiolitis obliterance.** Corticosteroids are treatment
- Systemic sclerosis patient presented with increasing SOB.O/E fine crackles are at bases→ **fibrosing alveolitis.**
- Surfactant(80% phosphatidylcholine) is produced by lamellar bodies in **Type-2 Pneumocytes.** Complete alveolization does not occur until 28 weeks gestation.

- ❖ Patient presented with dyspnea, breathlessness 2 weeks after

radiotherapy for lung cancer→Radiation pneumonitis. Treated with corticosteroids.

Psychogenic hyperventilation:

- Multiple presentations of a young woman in ER with acute onset SOB, dizziness and tingling sensations over limbs→ psychogenic breathlessness syndrome(hyperventilation syndrome).
- Young woman with intermittent SOB, saturation 98%, CXR normal. ABGs respiratory alkalosis → hyperventilation syndrome.

Empyema:

- IV drug abuser presents with high fever and cough.CXR shows **pleural effusion with Ph 7.1**→ start iv cefuroxime,oral metronidazole and **insert a chest drain into effusion.**(pt. has pneumonia and empyema).

Berylliosis

H/O working in aerospace industry + lung fibrosis + B/L hilar lymphadenopathy → think berylliosis

Pleural effusion:

causes

Pleural effusions may be classified as being either a transudate or exudate according to the protein concentration.

Transudate (< 30g/L protein)

- heart failure (most common transudate cause)
- hypoalbuminaemia
- ✓ liver disease
- ✓ nephrotic syndrome
- ✓ malabsorption
- hypothyroidism
- Meigs' syndrome

Exudate (> 30g/L protein)

- infection
- ✓ pneumonia (most common exudate cause),
- ✓ tuberculosis
- ✓ subphrenic abscess
- connective tissue disease
- ✓ rheumatoid arthritis
- ✓ systemic lupus erythmatosus
- neoplasia
- ✓ lung cancer
- ✓ mesothelioma
- ✓ metastases
- pancreatitis
- pulmonary embolism
- Dressler's syndrome
- yellow nail syndrome

Features

- dyspnoea, non-productive cough or chest pain are possible presenting symptoms
- classic examination findings include dullness to percussion, reduced breath sounds and reduced chest expansion

Pleural effusion: investigation and management

Imaging

- posterioranterior (PA) chest x-rays should be performed in all patients
- ultrasound is recommended: it increases the likelihood of successful pleural aspiration and is sensitive for detecting pleural fluid septations
- contrast CT is now increasingly performed to investigate the underlying cause, particularly for exudative effusions

Other characteristic pleural fluid findings:

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

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**

Chest x-ray: Cavitating lung lesion

Causes:

1. abscess (Staph aureus, Klebsiella and Pseudomonas) [FCPS]
2. **squamous** cell lung cancer [MRCP]
3. tuberculosis
4. Wegener's granulomatosis
5. pulmonary embolism
6. rheumatoid arthritis
7. aspergillosis, histoplasmosis, coccidioidomycosis

Pulmonary eosinophilia

Causes of pulmonary eosinophilia

1. Churg-Strauss syndrome
2. allergic bronchopulmonary aspergillosis (ABPA)
3. Loffler's syndrome
4. eosinophilic pneumonia
5. hypereosinophilic syndrome
6. tropical pulmonary eosinophilia
7. drugs: **nitrofurantoin, sulphonamides**
8. less common: Wegener's granulomatosis

Loffler's syndrome

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

Tropical pulmonary eosinophilia
associated with Wuchereria bancrofti infection

Q fever:

- Shepherd presents with high fever, pharyngitis, blood stained sputum, headache and systolic murmurr →Q fever(Doxycycline is treatment).

- Normal anatomical dead space is 150ml. Pneumonia increases anatomical dead space.
- Unilateral diaphragm paralysis(hemidiaphragm on X-Ray)→ Fluoroscopy for confirmation of diagnosis

- Flow volume loop is the investigation of choice for upper airway compression [MRCP]

- Most useful investigation to assess severity of airway obstruction in tracheal compression by retrosternal goiter(extra thoracic) → flow/volume loop.

- causes of nocturnal cough are:
  - Asthma
  - Reflux disease
  - Postural drip

- Causes of high KCO with normal or low TLCO are:
  - Lobectomy / pneumonectomy
  - Neuromuscular weakness
  - Scoliosis/kyphosis
  - Ankylosing spondylitis