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Pleural calcification Unilateral pleural calcification • most commonly occurs as a chronic change secondary to: pleural infection: tuberculous empyema, pyogenic empyema, haemothorax (post-traumatic) Bilateral pleural calcification • Common calcified pleural plaques are usually considered asbestos-related. • Other rarer causes radiation exposure, hyperparathyroidism, pulmonary infarction, pancreatitis.

Solitary pulmonary nodules Definition • A small (≤30 mm), well defined lesion completely surrounded by pulmonary parenchyma detected as an incidental finding, either on chest x-ray or CT scans. • Lesions larger than 3 cm are considered masses and are treated as malignancies until proven otherwise. Causes • benign nodules (The most common) Infectious granulomas and hamartomas are the most common causes of benign nodules. • malignant nodules The most common causes of malignant nodules are primary lung cancer, lung metastases, and carcinoid tumors. Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad

Lung collapse (Atelectasis) • There is increased opacity in the right upper zone, The lateral / inferior border of the shadowing actually represents the horizontal fissure which has been 'dragged' upwards.

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Pulmonology Management • Risk stratification of incidental pulmonary nodules consider the risk factors for lung cancer or metastases, as well as size and character of the nodule. surveillance according to British Thoracic Society Guidelines published in 2005. Nodules < 5 mm require no further surveillance. Nodules 5-6mm require CT at 1 year Nodules 6-8 mm require CT at 3 months Nodules > 8 mm require malignancy risk calculation using the Brock model and should then have CT or PET according to whether this risk is > 10%. To determine risk of malignancy following CT the BTS uses the Brock model The Brock model considers age, gender, family history and features of the nodule Only nodules which are greater than 8mm in diameter and have a greater than 10% risk of malignancy, as assessed by the Brock model (this can be accessed on the BTS website) should undergo PET-CT and then, based on outcomes, be assessed for obtaining a histological sample. • Requesting a previous chest x-ray is the most appropriate first step in management of a patient with a solitary pulmonary nodule, especially when the risk of

malignancy is high (age > 40 years, history of smoking). If there are no new changes, the patient can be followed-up with yearly chest x-rays. However, if there are new changes noted (additional nodules, enlargement), or no previous chest x-ray is available, a CT scan is indicated to assess for nodule size, location, and signs of malignancy, before eventual biopsy.

Alveolar-arterial (A-a) oxygen gradient Definition • The difference between the partial pressure of oxygen in the alveoli (A) and the partial pressure of oxygen in the arteries (a). Indications of uses • Used in diagnosing the source of hypoxemia. For example, in high altitude, the arterial oxygen PaO2 is low but only because the alveolar oxygen (PAO2) is also low. in states of ventilation perfusion mismatch, such as pulmonary embolism or rightto-left shunt, oxygen is not effectively transferred from the alveoli to the blood which results in elevated A-a gradient • in hypoxaemia it can differentiate between extrinsic and intrinsic restrictive lung disease if A-a gradient is normal →the cause is extrinsic, so exclude intrinsic lung disease Normal range • The normal range varies with age, altitude, and the concentration of inhaled oxygen. Normal range for a young person breathing room air at sea level is 5–10 mm Hg and increases with physical exercise. • A–a gradient = alveolar O2 (PAO2) – arterial O2 (PaO2)

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Causes of increased A-a gradient • Age • Higher concentration of inhaled oxygen • Right-to-left shunting (e.g. cyanotic heart disease) • Fluid in alveoli: e.g., CHF, ARDS, pneumonia • Ventilation/perfusion (V/Q) mismatch (due to increased dead space or shunting): e.g., pulmonary embolism, pneumothorax, atelectasis, obstructive lung disease, pneumonia, pulmonary edema • Alveolar hypoventilation: interstitial lung disease, lung fibrosis (usually manifests with ↑ CO2) Causes of hypoxaemia with normal A-a gradient • high altitude (both PAo2 and Pao2 are equally reduced) • hypoventilation (except lung): higher respiratory centre (e.g. drug induced) upper air way (e.g. acute epiglottitis) chest wall (e.g. kyphoscoliosis) respiratory muscles (e.g. myasthenia graves) haemoglobin defect anaemia : normal paO2 , normal SaO2 , low O2 content methemoglobinemia : normal PaO2, low SaO2 , low O2 content An increased A-a gradient may occur in hypoxemia due to shunting, ventilation-perfusion mismatch, or impaired gas diffusion across the alveoli due to fibrosis or edema. The A-a gradient remains normal with hypoventilation due to CNS and neuromuscular disorders (no diffusion defect) and in high altitude (despite a lower fraction of inhaled O2).

Finger clubbing Definition • Loss of the natural angle between the nail and the nail bed. • increased curvature of the nail Causes • Suppurative diseases: long-standing bronchiectasis acute lung abscesses empyema • Malignant disease - especially carcinoma of the bronchus and pleural malignancy • Fibrosing alveolitis • Asbestosis • hypertrophic pulmonary osteoarthropathy, painful osteitis of the distal ends of the long bones of the lower arms and legs. Malignancy is associated in 95% of these cases. Finger clubbing is not seen in uncomplicated bronchitis.

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Pulmonology Respiratory failure Top tips Type 1 respiratory failure • Definition hypoxaemia (PaO2 < 8.0 kPa) without hypercapnia (PaCO2 normal or decreased (<6.0 kPa) Causes: Low ambient oxygen (e.g. at high altitude) Ventilation-perfusion mismatch (parts of the lung receive oxygen but not enough blood to absorb it, e.g. pulmonary embolism) Alveolar hypoventilation (decreased minute volume due to reduced respiratory muscle activity, e.g. in acute neuromuscular disease); this form can also cause type 2 respiratory failure if severe Diffusion problem (oxygen cannot enter the capillaries due to parenchymal disease, e.g. in pneumonia or ARDS) Shunt (oxygenated blood mixes with non-oxygenated blood from the venous system, e.g. right-to-left shunt) Type 2 respiratory failure • Definition: Hypoxemia (PaO2 <8kPa (60 mmHg)) with hypercapnia (PaCO2 >6.0kPa (45 mmHg)). • Causes: Increased airways resistance (COPD, asthma, suffocation) Reduced breathing effort hypoventilation: due to drug overdose and brain stem lesion acutely due to: gross obesity, kyphoscoliosis (and similar chronically musculoskeletal disorders) Hypoventilation, where inadequate alveolar ventilation results in low alveolar PO2, is the only cause of hypoxia that inevitably causes raised PaCO2. A decrease in the area of the lung available for gas exchange (such as in chronic bronchitis) Neuromuscular problems (respiratory muscle weakness) (Guillain-Barre syndrome , motor neuron disease) Deformed (kyphoscoliosis), rigid (ankylosing spondylitis), or flial chest. Respiratory failure • Type 1 hypoxaemia (PaO2 < 8.0 kPa) without hypercapnia (PaCO2 normal or decreased (<6.0 kPa) • Type 2 Hypoxemia (PaO2 <8kPa) with hypercapnia (PaCO2 >6.0kPa). Type 2 respiratory failure with normal CXR neuromuscular weakness

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Bronchial Asthma Immunological response involved in atopic asthma: • Immediate response: type I hypersensitivity immunomodulators involved: mast cells , histamine Result in immediate bronchoconstriction reaction Usually subsides within two hours Reversible with bronchodilators. • Late phase: type IV hypersensitivity response Results in bronchoconstriction, airways inflammation, hyper-responsiveness and oedema. This typically occurs three to12 hours after the immediate response Less susceptible to bronchodilators. Pathogenesis of asthma: • Asthma occurs due to a combination of airway hyper-responsiveness, airflow limitation and airway inflammation • The alveolar functional structure is preserved in asthma. Near fatal asthma • The British Thoracic Society defines near fatal asthma as an attack with raised PaCO2 and/or requiring mechanical ventilation with raised inflation pressures. • A raised PaCO2 is an important sign that intubation may be required if the patient is not responding to maximum medical management. Features • wheeze, cough or breathlessness, and any daily or seasonal variation in these symptoms, any triggers that make symptoms worse • a personal or family history of atopic disorders. Asthma diagnosis (NICE guidelines 2017) NICE guidelines Do not use symptoms alone without an objective test to diagnose asthma. Empirically inhaled corticosteroids may affect the results of spirometry and FeNO tests • Step 1: Exclude occupational asthma by asking if their symptoms are better on days away from work/during holidays. • Step 2: Test for airway inflammation →Fractional exhaled nitric oxide (FeNO) test If ˃ 40 ppb → positive • Step 3: test for obstructive airway disease → Spirometry FEV1/FVC ratio < 70% → positive (obstructive spirometry). • Step 4: test for Bronchodilator reversibility (BDR) → bronchodilators + Spirometry improvement in FEV1 ≥ 12%, + ↑volume ≥ 200 ml → positive

Pulmonology • Step 5: If there is diagnostic uncertainty (e.g. positive BDR with borderline FeNO OR obstructive spirometry + negative BDR) →Peak expiratory flow variability for 2 to 4 weeks value ≥ 20% variability is a positive test. • Step 6: If there is diagnostic uncertainty (positive FeNO ≥ 40 BUT normal spirometry and no variability in peak flow readings OR borderline FeNO with obstructive spirometry BUT negative BDR and no variability in peak flow readings) → Airway hyperreactivity measures → Direct bronchial challenge test with histamine or methacholine PC20 value ≤ 8 mg/ml is a positive test. NICE quality statement : Adults with new onset asthma are assessed for occupational causes. Are you better on days away from work? Are you better on holiday? All patients with suspected B. Asthma should have spirometry with a bronchodilator reversibility (BDR) test and FeNO test Diagnosis of asthma (NICE guidelines 2017)
Patients ≥ 17 years: Exclude occupational asthma (by asked if their symptoms are better on days away from work/during holidays). Do spirometry with a bronchodilator reversibility (BDR) test + Fractional exhaled nitric oxide (FeNO) for all patients obstructive spirometry FEV1/FVC ratio < 70% positive BDR test improvement in FEV1 ≥12%, together with an increase in volume ≥200 positive FeNO test ≥ 40 monitor Peak expiratory flow variability: for 2 to 4 weeks, if there is diagnostic uncertainty: normal spirometry OR obstructive spirometry, positive BDR but a FeNO ≤ 39 Regard a value > 20% variability as a positive test. Patients 5-16 years: Do spirometry with a bronchodilator reversibility (BDR) test Do a FeNO test if there is: normal spirometry OR obstructive spirometry with a negative BDR test Regard a value of FeNO test ≥ 35 as a positive test. In asthma diagnosis: Positive spirometry with a bronchodilator reversibility (BDR) test → improvement in FEV1 of ≥12% Positive peak flow meter → ˃ 20% variation

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Positive tests in Asthma Test Positive result Fractional exhaled nitric oxide (FeNO) 40 ppb or more FeNO 35 ppb or more Obstructive spirometry Forced expiratory volume in 1 second/forced vital capacity (FEV1/FVC) ratio less than 70% (or below the lower limit of normal if this value is available) Bronchodilator reversibility (BDR) test Improvement in FEV1 of 12% or more and increase in volume of 200 ml or more BDR test Improvement in FEV1 of 12% or more Peak flow variability Variability over 20% Direct bronchial challenge test with histamine or methacholine Provocative concentration of methacholine causing a 20% fall in FEV1 (PC20) of 8 mg/ml or less Management of asthma (NICE guidance 2017). One of the key changes is in 'step 3' - patients on a SABA + ICS whose asthma is not well controlled should be offered a leukotriene receptor antagonist, not a LABA Step Notes

Newly-diagnosed asthma Short-acting beta agonist (SABA)

Not controlled on previous step OR Newly-diagnosed asthma with symptoms ≥3 / week or nighttime waking SABA + low-dose inhaled corticosteroid (ICS)

SABA + low-dose ICS + leukotriene receptor antagonist (LTRA)

SABA + low-dose ICS + long-acting beta agonist (LABA) Continue LTRA depending on patient's response to LTRA

SABA +/- LTRA

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Pulmonology Switch ICS/LABA for a maintenance and reliever therapy (MART), that includes a low-dose ICS

SABA +/- LTRA + medium-dose ICS MART OR consider changing back to a fixed-dose of a moderate-dose ICS and a separate LABA

SABA +/- LTRA + one of the following options: • increase ICS to high-dose (only as part of a fixeddose regime, not as a MART) • a trial of an additional drug (for example, a longacting muscarinic receptor antagonist or theophylline) • seeking advice from a healthcare professional with expertise in asthma Drugs used in asthma Drug Mechanism of action Notes Salbutamol Beta receptor agonist • Short-acting inhaled bronchodilator. Relaxes bronchial smooth muscle through effects on beta 2 receptors • Used in asthma and chronic obstructive pulmonary disease (COPD). • Salmeterol has similar effects but is long-acting Corticosteroids Anti-inflammatory • Inhaled corticosteroids are used as maintenance therapy • Oral or intravenous corticosteroids are used following an acute exacerbation of asthma or COPD Ipratropium Blocks the muscarinic acetylcholine receptors • Short-acting inhaled bronchodilator. Relaxes bronchial smooth muscle • Used primarily in COPD • Tiotropium has similar effects but is long-acting Methylxanthines (e.g. theophylline) Non-specific inhibitor of phosphodiesterase resulting in an increase in cAMP • Given orally or intravenously • Has a narrow therapeutic index Montelukast, zafirlukast Blocks leukotriene receptors • Usually taken orally • Useful in aspirin-induced asthma Steroid inhalation • Fluticasone is more lipophilic and has a longer duration of action than beclomethasone • Hydrofluoroalkane is now replacing chlorofluorocarbon as the propellant of choice. Only half the usual dose is needed with hydrofluoroalkane due to the smaller size of the particles • Table showing examples of inhaled corticosteroid doses •

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Dose Example low dose ≤ 400 micrograms budesonide or equivalent moderate dose 400 micrograms - 800 micrograms budesonide or equivalent high dose

> 800 micrograms budesonide or equivalent • Side effects: Inhaled corticosteroids →↓↓ skin collagen synthesis →skin fragility →↑↑ tendency for bruising & vascular changes Cushing's syndrome: Interaction with potent cytochrome P450-3A4 inhibitor → elevations in plasma fluticasone concentrations (even nasal or inhaled preparations) →suppress endogenous

> cortisol levels and produce Cushing's syndrome. eg: a patient with HIV and asthma, C/O tiredness, lethargy and weight gaining → suspect Cushing's syndrome produced by Ritonavir, a protease inhibitor which is an extremely potent cytochrome P450-3A4 inhibitor. Long acting B2-agonists • Action: acts as bronchodilators but also inhibit mediator release from mast cells. The duration of action of salmeterol is around 12 hours • Side effects: Salmeterol →may cause paradoxical bronchospasm Leukotriene receptor antagonists • Action Montelukast, zafirlukast CysLT1 antagonist; it blocks the action of leukotriene on cysteinyl leukotriene receptor CysLT1 by binding to it. Zileuton →blocks leukotriene synthesis by inhibiting 5-lipoxygenase, inhibits 5-lipoxygenase pathway, blocking the conversion of arachidonic acid to leukotrienes. have both anti-inflammatory and bronchodilatory properties • Inductions should be used when patients are poorly controlled on high-dose inhaled corticosteroids and a long-acting b2-agonist have been shown to be as effective as doubling the dose of inhaled steroid. asthma with allergic rhinitis aspirin-induced asthma exercise-induced asthma • Side effects associated with the development of Churg-Strauss syndrome Asthma drugs: leukotriene inhibitor action: Zafirlukast →Inhibitor of LT receptor Zileuton →Antagonist of lipoxygenase

Pulmonology Omalizumab • Action: monoclonal antibody that binds to IgE. • Indications: severe refractory, persistent confirmed allergic IgE-mediated asthma (e.g. positive skin test to a recognised respiratory allergen) • Administration: given subcutaneously every 2 or 4 weeks. • Side effects: injection site pain, swelling, erythema, pruritus, and headaches Non-pharmacological management • Stop smoking. • Weight-loss interventions • Breathing exercise programs (including physiotherapist-taught methods) can be offered to people with asthma as an adjuvant to pharmacological treatment to improve quality of life and reduce symptoms Diaphragmatic breathing, (as opposed to thoracic breathing which is practised by many asthmatics): reduce symptoms Buteyko technique: a breathing technique which can ‘improve asthma symptoms, quality of life and reduce bronchodilator (blue reliever inhaler) requirement Omalizumab anti-IgE monoclonal antibody used for resistant asthma with evidence of raised IgE and allergic symptoms. Mepolizumab anti-IL5 monoclonal antibody used for resistant asthma with raised eosinophils B-blockers, including eye drops, should be avoided in patients with asthma. They are not however absolutely contraindicated.

Acute severe asthma Classification of acute severe asthma • Patients with acute severe asthma are stratified into moderate, severe or lifethreatening. • Note that a patient having any one of the life-threatening features should be treated as having a life-threatening attack. 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 in one breath • RR > 25/min • Pulse > 110 bpm • PEFR < 33% best or predicted • Oxygen sats < 92% • PaO2 < 8 kPa • Normal

PaCO2 (4.6-6.0 kPa) • Silent chest, cyanosis or feeble (Poor) respiratory effort • Bradycardia, dysrhythmia or hypotension • Exhaustion, confusion or coma

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Management of acute severe asthma • β2-agonists should be administered as soon as possible, preferably nebulised driven with high flow oxygen. salbutamol administration can rapidly worsen the V/Q mismatch which is the cause of hypoxia in asthma. They can therefore cause reduction in arterial oxygen tension unless supplemental oxygen is given • Nebulised ipratropium bromide. It's addition produces significantly greater bronchodilation than a β2-agonist alone. • Oxygen: Targeted oxygen in asthma →SpO2 level of 94–98%.
• Steroids: steroids reduce mortality, relapses, subsequent hospital admission and requirement for β2-agonists1. This should be continued for five days, and can then be stopped abruptly. • Magnesium sulphate recommended as next step for patients who are not responding (e.g. 1.2 - 2g IV over 20 mins) . Mechanism: low magnesium levels in bronchial smooth muscle favour bronchoconstriction. reduce rates of admission to intensive therapy units • Intensive care is indicated for patients with severe acute or life threatening asthma who are failing to respond to therapy. Strongest indicator of a need for intubation and ventilation →PH 7.31 Asthmatic patient with + PaCO2 at the upper limit of normal. What would be the most appropriate next step? A normal or elevated pCO2 in an asthmatic indicates impending respiratory failure review by an anaesthetist/intensivist is the next immediate step. Hypercapnia and signs of fatigue are indications for immediate intubation and ventilation. Management of Asthma in pregnancy • In general, the medicines used for asthma are safe during pregnancy. • The British Thoracic Society (BTS) guidelines make it clear that short-acting /long-acting beta 2-agonists, inhaled and oral corticosteroids should all be used as normal during pregnancy. • The BNF advises that 'inhaled drugs, theophylline and prednisolone can be taken as normal during pregnancy and breast-feeding'.

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Pulmonology Chronic Obstructive Pulmonary Disease(COPD) Definition: airflow obstruction that is not fully reversible. Epidemiology: • worldwide prevalence of 10% • COPD is the third leading cause of death worldwide Subtypes of COPD

1. Chronic bronchitis: defined as chronic cough and sputum production for at least three months of two consecutive years in the absence of other disease which could explain these symptoms.
2. Emphysema Pathophysiology • Inflammatory changes → ciliary dysfunction and increased goblet cell size and number, → excessive mucus secretion. • Increased airway resistance is the physiological definition of COPD. • Decreased elastic recoil, fibrotic changes in lung parenchyma, and luminal obstruction of airways by secretions all contribute to increased airways resistance. • Progressive hypoxia →vascular smooth muscle thickening → pulmonary hypertension • Which mechanism is most likely responsible for the increased mean arterial pulmonary pressure in COPD? Hypoxic induced pulmonary vasoconstriction

Causes • Smoking • Alpha-1 antitrypsin deficiency • Using open fires at homes for cooking or heating (patients from the developing world present with a COPD-like history without smoking history) • Occupational exposures, such as harmful dust and chemicals cadmium (used in smelting) (recognised cause of emphysema specifically) coal, cotton, cement, grain Emphysema • Definition emphysema is a term that refers to the actual damage to the air sacs in the lung, called the alveoli. In other words, emphysema is a pathological term. • Pathophysiology destruction of alveolar air sacs due to an imbalance between protease and antiprotease action. loss of elastic recoil, which drives airflow limitation. • Types Panlobular (panacinar) pulmonary emphysema Rare Associated with α1-antitrypsin deficiency Characterized by destruction of the entire acinus Usually affects the lower lobe Centrilobular or proximal acinar pulmonary emphysema Common Classically seen in smokers

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 Characterized by destruction of the respiratory bronchiole (central portion of the acinus) Usually affects the upper lobe most severe at the apex of the lung. Types of emphysema Type Centriacinar (centrilobular) Panacinar Prevalence the most common type Less common destruction focal destruction mainly localized to the proximal respiratory bronchioles destroys the entire alveolus uniformly Location upper lung zones. lower half of the lungs. causes smoking & dust alpha 1-antitrypsin (AAT) deficiency MRCPUK-part-1-septemper-2017: What is the most important factor in airflow limitation in severe emphysema? Loss of elastic recoil Features and complications • Chronic cough , SOB and recurrent infection • extensor plantar response is common in (COPD) due to carbon dioxide retention, which results in carbon dioxide narcosis. • Cor pulmonale features include peripheral oedema, raised jugular venous pressure, systolic parasternal heave, loud P2 use a loop diuretic for oedema, consider long-term oxygen therapy ACE-inhibitors, calcium channel blockers and alpha blockers are not recommended by NICE • Polycythaemia

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Pulmonology COPD - Investigation and diagnosis Who should be suspected of COPD? • 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 exertional breathlessness, chronic cough or regular sputum production. Investigations recommended in patients with suspected COPD: • spirometry 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 • methacholine challenge useful in differentiating between asthma and chronic obstructive pulmonary disease (COPD). methacholine utilizes the M3 receptor for bronchoconstriction. • ABG In long standing COPD the bicarbonate is likely to be normal, or raised if the patient has chronic hypercapnia. (a low pH, low pO2, high pCO2 and a high HCO3) Severity of COPD: categorised by 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

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COPD: causes of acute exacerbations • Infective exacerbation of COPD is the most common cause of haemoptysis in UK patients • bacterial organisms Haemophilus influenzae (most common cause of COPD exacerbation) Streptococcus pneumoniae Moraxella catarrhalis • Respiratory viruses : account for around 30% of exacerbations, with the human rhinovirus being the most important pathogen. COPD: management of acute exacerbations • Bronchodilator • Steroids: prednisolone 30 mg daily for 7-14 days. Prolonged courses offer no additional benefit • Antibiotics: 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' • Oxygen management of COPD patients If the patient have an individual target range: Oxygen should be given to maintain SaO2 within the patient's individual target range, if available If the individual target is not known: prior to availability of blood gases (pCO2 is unknown): saturations should be maintained at 88-92% to avoid risk of hypercapnia after availability of blood gases (pCO2 is normal): adjust target range to 94-98% • Non-invasive ventilation • Respiratory stimulants (e.g. Doxapram) In COPD exacerbations, respiratory stimulants (e.g. Doxapram) should only be used when Non-invasive ventilation is either unavailable or considered inappropriate COPD: stable management

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Pulmonology General management • smoking cessation advice • annual influenza vaccination • one-off pneumococcal vaccination Pharmacological therapy • first-line: short-acting beta2-agonist (SABA) or short-acting muscarinic antagonist (SAMA) • second-line: for patients who remain breathless or have exacerbations despite using short-acting bronchodilators the next step is determined by the FEV1 FEV1 > 50% long-acting beta2-agonist (LABA), for example salmeterol, or: long-acting muscarinic antagonist (LAMA), for example tiotropium FEV1 < 50% LABA + inhaled corticosteroid (ICS) in a combination inhaler, or: LAMA • Third-line: For patients with persistent exacerbations or breathlessness if taking a LABA then switch to a LABA + ICS combination inhaler otherwise give a LAMA and a LABA + ICS combination inhaler Other pharmacological therapies • 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 coprescribed • Mucolytics: should be 'considered' in patients with a chronic productive cough and continued if symptoms improve • LTOT : should be offered to: patients with a pO2 of < 7.3 kPa or patients with a pO2 of 7.3 - 8 kPa and one of the following:

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 secondary polycythaemia nocturnal hypoxaemia peripheral oedema pulmonary hypertension • Roflumilast Indication: recommended by NICE for patients who have suffered two or more exacerbations in a year, despite triple inhaled therapy, where FEV1 is less than 50% of predicted. Mode of action: selective long-acting phosphodiesterase-4 inhibitor. It is orally administered. Management of side effect of steroid inhaler (Oro-pharyngeal and oesophageal candidiasis) • the patient should be taught adequate inhaler technique. Advise him to rinse his mouth each time he uses his inhaler use a spacer device review him in a month. and and • Resistant symptoms can be managed with oral nystatin or a course of fluconazole. Pulmonary rehabilitation • Definition: a programme of aerobic lower-extremity training combined with education. • Indication: Patients with very limited exercise tolerances • Effects It leads to improvements in exercise capacity (walking distance should improve after the rehabilitation programme) The improvement in walking distance would not be a long-lasting improvement Decline in exercise tolerance and health status tends to occur 6–12 months after the completion of a course. The effect of sustained improvement with ongoing rehabilitation has yet to be evaluated. does not improve lung function. does not decrease hospital admissions because of chest problems, but their hospital stays are likely to be shorter. Lung volume reduction surgery • Is a palliative treatment which can be used in advanced COPD to remove the least functional part of the lungs. • there are 3 groups of patients that tend to benefit: Group 1: Upper lobe emphysema and low exercise capacity. These patients show improvement in both functional outcomes and survival after lung volume reduction surgery compared to medical therapy. Group 2: upper lobe emphysema and high exercise capacity. These patients have improved functional outcomes but no difference in survival compared to medical therapy. Group 3: non-upper lobe emphysema and low exercise capacity. These patients have improved survival after surgery but there is no difference in survival compared to medical therapy. • patients with emphysema that are unlikely to do well from lung volume reduction surgery and have a high risk of death includes:

1. non-upper lobe emphysema and high exercise capacity.
2. extremely poor pulmonary function (forced expiratory volume in 1 second (FEV1) 20% or less than predicted) and either homogenous distribution of emphysema on

Pulmonology computed tomography scan or extremely poor carbon monoxide diffusing capacity (20% or less than predicted). • Indications: CO2 retention: The upper cut off for referral for lung reduction surgery for pCO2 is 7.3 Severe limitation of exercise capacity despite maximal therapy predominant upper lobe emphysema, and persistent symptoms despite a period of pulmonary rehabilitation. • selection criteria: used when assessing suitability for treatment: Age <75 years Emphysema by clinical evaluation Ex-smoker of more than 4 months Clinically stable on no more than 20mg prednisolone daily Significant functional limitation after 6-12 weeks of pulmonary rehabilitation on optimal medical therapy Demonstrated compliance with medical regimen FEV-1 >20% predicted Post-bronchodilator FEV-1 >45% predicted and >15% if >70 years Hyperinflation demonstrated by TLC >100% predicted and RV >150% predicted Carbon monoxide lung transfer factor greater than 20% predicted Post rehabilitation 6-minute walk distance >140 m Low post rehabilitation exercise capacity HRCT demonstrating bilateral severe emphysema, ideally with upper-lobe predominance Symptomatic relief of breathlessness in

end-stage COPD (DNR cases) • opioid or benzodiazepine medications for symptomatic relief of breathlessness is appropriate.

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Non-invasive ventilation (NIV) Indications of NIV 1. COPD with respiratory acidosis (pH 7.25-7.35) who have not improved despite immediate maximum standard medical treatment on controlled oxygen for no more than one hour. patients with a pH in the range of 7.25-7.35 achieve the most benefit. If the pH is < 7.25 then invasive ventilation should be considered if appropriate. 2. Type II respiratory failure secondary to chest wall deformity, neuromuscular disease or Obstructive sleep apnoea 3. Cardiogenic pulmonary oedema 4. Weaning from tracheal intubation Advantage of NIV • reduce intubation rates • lower hospital mortality rates and • lead to shorter hospital stays Recommended initial settings for bi-level pressure support in COPD • Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H20 whilst BTS suggest 12-15 cm H2O. • Expiratory Positive Airway Pressure (EPAP): 4-5 cm H2O • back up rate: 15 breaths/min • back up inspiration: expiration ratio: 1:3 Monitoring and setting adjustment • ABGs should be repeated after 1 hour of NIV therapy, and 1 hour after subsequent change in settings or 4 hours in stable patients. If gas exchange is not significantly improved: the IPAP can be gradually increased at a rate of approximately 5 cms (2-5cm) H2O every 10 minutes with a usual target of 20cm H2O or until a response has been achieved or patient tolerability has been reached. Increases in EPAP are not recommended without specialist advice.

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Pulmonology • If the patient struggle to tolerate the NIV mask, what is the most appropriate method to help him settle him? haloperidol or morphine Decreasing the IPAP or stopping NIV would be more comfortable but would be inappropriate as treatment is then likely to fail with greater hypoxia and acidosis. Diazepam is contraindicated Increasing EPAP without increasing IPAP would reduce the amount of ventilatory support and would be inappropriate. Complication of NIV • ventilation associated pneumothorax is (most important complication of NIV →present acutely) • Ventilator associated pneumonia →present in patients who have been ventilated for long period of time and would not present so acutely). Contraindications to NIV • Absolute contraindications: inability to fit the NIV mask appropriately, cardiopulmonary arrest, and need for urgent intubation • Relative contraindications haemodynamically unstable requiring inotropes/pressors (unless in a critical care unit) confusion/agitation NIV modes • Continuous positive airway pressure (CPAP) As this mode only provides a continuous pressure, CPAP is a spontaneous mode of ventilation that requires patient initiation and respiratory muscle effort. Therefore, no respiratory rate or minute ventilation is targeted or guaranteed. The clinical benefit of CPAP is most evident in hypoxemic respiratory failure as the positive airway pressure predominately augments oxygenation with the goal of recruiting alveoli, increasing functional residual capacity, and decreasing shunting. Inspiration during IPPV →↑↑ intrathoracic pressure →↑↑ right atrial pressure →↓↓ venous return →↓↓ cardiac output • Bilevel positive airway pressure (BiPAP) in contrast to CPAP, provides both an expiratory positive airway pressure (EPAP) and inspiratory positive airway pressure (IPAP). BiPAP has utility in both hypoxemic and

hypercarbic respiratory failure. Minute ventilation • Minute ventilation is equal to tidal volume (volume of air moved in normal breathing) multiplied by the respiratory rate. • In metabolic alkalosis, one could increase CO2 content by decreasing the minute ventilation (volume of air moved per minute). • Reducing either one of these variables(↓tidal volume or ↓ respiratory rate) will decrease minute ventilation and lead to increased CO2 retention. •

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Invasive ventilation Indications • unconscious patient • if the pH is below 7.25. Patients with a pH <7.26 should be managed with a low threshold for intubation. give NIV whilst waiting for intensive care. • in Guillain Barre syndrome with respiratory involvement →the parameter used to assess whether a patient needs ventilator support is an FVC <15-20ml/kg. Ethics in decision to ventilate • If the patient had a written advanced directive, properly witnessed, while he was well, then it would not be possible to consider intervention if he wished for it not to happen. • if he has significant hypoxia, he might not be able to give a rational decision with respect to his further treatment. • if significantly hypoxic patient refused intubation during acute exacerbation → Intubate and act on the best interests of the patient, while informing the relatives • The family should not have the final decision with respect to intubation.

Long-term oxygen therapy (LTOT) COPD - LTOT if 2 measurements of pO2 < 7.3 kPa Which patients should be assessed for and offered (LTOT)? • Assess patients if any of the following: Very severe airflow obstruction (FEV1 < 30% predicted). cyanosis polycythaemia peripheral oedema raised jugular venous pressure oxygen saturations less than or equal to 92% on room air How to assess patient for LTOT? Assessment is done by measuring arterial blood gases on 2 occasions at least 3 weeks apart in patients with stable COPD on optimal management. Blood gases should be performed in a stable state, which should be at least four weeks after an exacerbation of the disease. Indications for LTOT in COPD: • patients with pO2 of < 7.3 kPa • patients with pO2 of 7.3 - 8 kPa and one of the following: secondary polycythaemia nocturnal hypoxaemia peripheral oedema pulmonary hypertension Duration of LTOT • Patients who receive LTOT should breathe supplementary oxygen for at least 15 hours a day including at night time.

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Pulmonology Contraindications • Continued cigarette smoking should be a relative contraindication to long-term oxygen therapy. In patients with chronic hypoxaemia, LTOT should be prescribed after assessment, when the PaO2 is consistently at or below 7.3 kPa (55 mm Hg) when breathing air during a period of clinical stability. Clinical stability is defined as the absence of exacerbation of chronic lung disease for the previous five weeks. The level of PaCO2 (which may be normal or elevated) does not influence the need for LTOT prescription.
The only treatment that improves the long-term prognosis in patients with (COPD) is LTOT, given for at least 15 hours per day. Patients who develop a respiratory acidosis and/or a rise in PaCO2 of >1 kPa (7.5 mmHg) during an LTOT assessment on two repeated occasions, while apparently clinically stable, should only have domiciliary oxygen ordered in conjunction with nocturnal

ventilatory support. How will you manage this patient? LTOT with nocturnal BiPAP BiPAP is the modality of choice for treating CO2 retention.

Prognosis of COPD • Once respiratory failure criteria have been met, the 5-year survival rate is only around 25%. • Prognostic indicators in COPD The strongest predictors of survival in patients with (COPD) are FEV1 • 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 in patients who fit criteria lung volume reduction surgery in selected patients

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Pulmonary embolism (PE) Pathophysiology • PE → ↓ lung blood flow → ventilation- perfusion mismatches (Decreased perfusion +
normal ventilation) → ↑ physiologic dead space. Risk factors The Virchow triad pathophysiological components of thrombus formation:

1. Hypercoagulability: thrombophilia (e.g., factor V Leiden mutation), use of oral contraceptives, pregnancy.
2. Endothelial damage: inflammatory or traumatic → activation of clotting factors through contact with exposed subendothelial collagen.
3. Stasis (e.g. varicosis, immobilization)

Major risk factors Minor risk factors • lower limb problems including a fracture or varicose veins • postoperative intensive care • hospitalisation • abdominal/pelvic or advanced malignancy • previous VTE, and • pregnancy. Features Sudden shortness of breath, pleuritic chest pain with haemoptysis and tachypnoea are the commonest features. (triad of pleuritic chest pain, dyspnoea and haemoptysis) • Tachypnea (respiratory rate >16/min) - 96% (Sudden shortness of breath) • Pleuritic chest pain (worse on deep breathing) • haemoptysis • Tachycardia (heart rate >100/min) - 44% • Fever (temperature >37.8 C) - 43%. Diagnosis • If a patient presents with signs or symptoms of pulmonary embolism (PE) performed chest x-ray to exclude other pathology estimate the clinical probability of PE by two-level PE Wells score Clinical feature Points Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins) An alternative diagnosis is less likely than PE

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 Haemoptysis

Malignancy (on treatment, treated in the last 6 months, or palliative)

Clinical probability simplified scores PE likely More than 4 points PE unlikely 4 points or less Notes & Notes for MRCP
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• occult malignancy • long distance travel • hypertension • congestive cardiac failure • thrombotic disorder • use of the oral contraceptive pill

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Pulmonology • PE likely (> 4 points): arrange an immediate computed tomography pulmonary angiogram (CTPA). If there is a delay in getting the CTPA then give low-molecular weight heparin until the scan is performed. If the patient has an allergy to contrast media or renal impairment a Ventilation-perfusion (V/Q) scan should be used instead of a CTPA. • PE unlikely (≤ 4 points): arranged a D-dimer test: If this is positive arrange an immediate (CTPA). If there is a delay in getting the CTPA then give low-molecular weight heparin until the scan is performed. It is interesting to note that the Well's criteria for diagnosing a PE use tachycardia rather than tachypnoea. Pulmonary embolism - normal CXR Investigations • Chest x-ray Should be performed in all patients with symptoms or signs suggestive of PE
 to exclude other pathology usually normal in PE • Computed tomographic pulmonary angiography (CTPA) the first-line diagnostic test If the CTPA is negative then patients do not need further investigations or treatment for PE Disadvantages of CTPA: Contrast induced nephropathy Low sensitivity for detecting pulmonary emboli in sub-segmental pulmonary arteries • Ventilation-perfusion (V/Q) scans Indication? → If CTPA is contra-indicated renal impairment (as the contrast media used during CTPAs is nephrotoxic). allergy to contrast media Sensitivity = 98%; specificity = 40% →high negative predictive value, i.e. if normal virtually excludes PE In pregnancy → Radiation to the fetus is small. • D-dimers Should be performed ONLY when the probability of PE is low, so the normal value would be taken as reassuring and further investigation would not be pursued. High sensitivity (95-98%), but poor specificity

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 A negative d-dimer is useful for excluding PE in patients who are clinically thought to be at low risk, but a 'positive' result does not establish the diagnosis. The negative predictive value is greater than the positive predictive value D-dimers can be positive in: hospitalised patients obstetric patients patients with peripheral vascular disease, cancer and inflammatory conditions increasing age D-Dimer measurements should not be performed if: an alternative diagnosis is likely, the clinical probability is high or there is a probable massive PE. • ECG sinus tachycardia the most common abnormality; seen in 44% of patients. the classic ECG changes S1Q3T3 (seen in no more than 20% of patients) large S wave in lead I large Q wave in lead III inverted T wave in lead III Right bundle branch block seen in 18% of patients. associated with increased mortality; Right axis deviation (seen in 16% of patients). • Elevated cardiac troponin levels also occur in patients with pulmonary embolism because of right ventricular dilation and myocardial injury Management Start low molecular weight heparin and request CT pulmonary angiography if the symptoms and findings clearly point towards pulmonary embolism (PE). Fluid resuscitation is the most appropriate immediate measure before further investigations confirm the presence of a pulmonary embolism (PE).