Short cases in medicine

Cardiovascular system

Examination routine

Outline of CVS Examination

General examination
Pulse examination
Blood pressure
Precordial examination
Inspection
Palpation
Auscultation
Lung bases
Liver

- Introduce yourself and take consent
- Ensure adequate exposure and request for a chaperone if neccessary
- Proper positioning of the patient at 45 degrees
- Comment on the general appearance of the patient especially whether he or she is dyspnoeic or ill looking
- **Start with the general examination**
  Febrile/not
  Marfanoid features
  Conjunctivae for pallor
  Central and peripheral cyanosis
  Dental hygiene
  Finger and toe clubbing
  Peripheral stigmata of infective endocarditis
  Ankle edema/ sacral edema

- **Then move on to the examination of the pulses. This is divided into arterial pulse examination and examination of the jugular venous pulse**
- **Arterial pulse examination**
  Start with the radial pulse and assess the rate and the rhythm
  Look for the character of the pulse and examine for collapsing pulse
  Examine the peripheral pulses quickly and then go on to look for radio-radial or radio-femoral delay
  Look at the neck for visible carotid pulses
  Feel the carotid pulse and comment on the volume of the pulse
- **Examine the jugular venous pulse**
  Try to look for the waveform of the JVP and measure the height of the venous pressure in centimeters
- **Offer to measure the blood pressure**
- **Examine the precordium**
  Inspection
  Look for any chest deformities
  Surgical scars- especially midline sternotomy scars and left thoracotomy scars
  Look for visible pulsations over the chest wall
  Palpation

Palpate for the apex beat and comment on the position of the apex beat and whether it is displaced or not. Also comment on the character of the apex beat

Palpate for thrills over the precordium

Palpate for a left parasternal heave which is indicative of right ventricular hypertrophy

Look for a palpable 2nd heart sound in the pulmonary area which is suggestive of pulmonary hypertension

Auscultation

Listen to the heart sounds first and comment on any abnormality

Auscultate in all 4 areas

Remember to demonstrate auscultation in the mitral area with the bell and the patient in the left lateral position for the murmur of mitral stenosis.

Examine the patient seated and leaning forward with the breath held in expiration for the murmur of aortic regurgitation

Listen for any murmur and describe the murmur based on the following points. Always remember to show the examiner that you are timing the murmur by palpating the carotid pulse (R side)

Systolic or diastolic

Further as pan systolic, mid systolic, early diastolic etc.

The location where the murmur is best heard

The grade of the murmur

Radiation of the murmur

The effect of respiration on the murmur (Remember that the intensity of all right sided murmurs is increased with inspiration and the intensity of all left sided murmurs are increased with expiration)

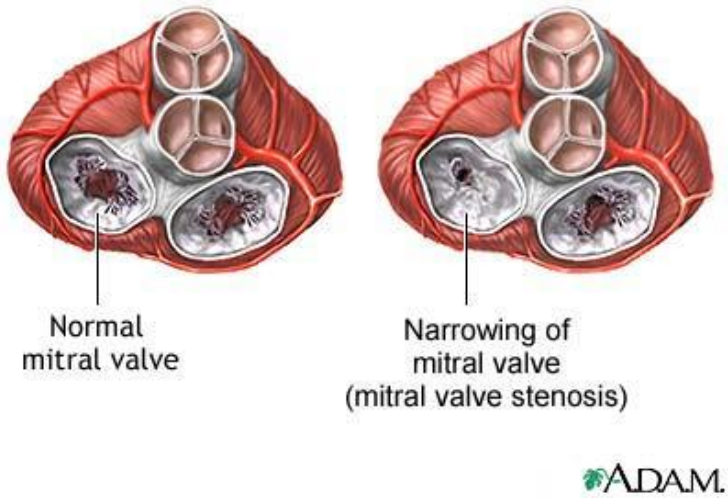
- **Examine the liver and lung bases for evidence of cardiac failure**

Try to reach a diagnosis before auscultation. This is possible and is extremely helpful for those who are not that good in auscultation. Study the following table.

Mitral stenosis

What is the diagnosis?

Why do you say so? What are the other lesions which could present with similar clinical findings?



Remember that the lesions given below are extremely rare and are never given for the exam. But the above question is a very popular one.

- Carey Coombs murmur in acute rheumatic carditis
- Austin flint murmur in severe aortic regurgitation
- Atrial myxoma

What is the most probable aetiology?

- Mitral stenosis is almost always rheumatic in origin

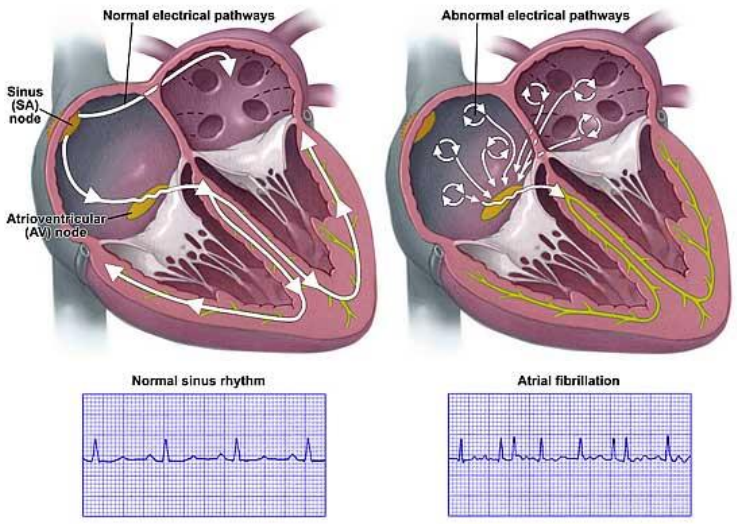
How would you clinically determine the severity of the lesion from the findings of the physical examination?

The following are some features which would indicate a severe lesion

- Longer the murmur the more severe the stenosis
- The gap between the second sound and the opening snap (a narrow gap would indicate severe stenosis but the opposite is not true)
- Evidence of complications
  Atrial fibrillation
  Pulmonary hypertension

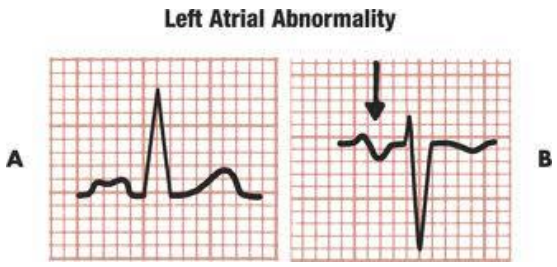
What are the complications associated with mitral stenosis?

- Left atrial dilatation and atrial fibrillation
- Left atrial thrombus +/- systemic embolization
- Pulmonary hypertension
- Tricuspid regurgitation
- Right heart failure



What are the investigations you would like to perform on this patient?

- ECG – P mitrale
- CXR – Enlarged left atrial appendage and pulmonary congestion
- Echo – This is the most important investigation. It confirms the diagnosis and looks for other associated valvular lesions. It also estimates the severity of the lesion based on the valve surface area and pressure gradient across the mitral valve. Echo is also useful to decide the mode of intervention



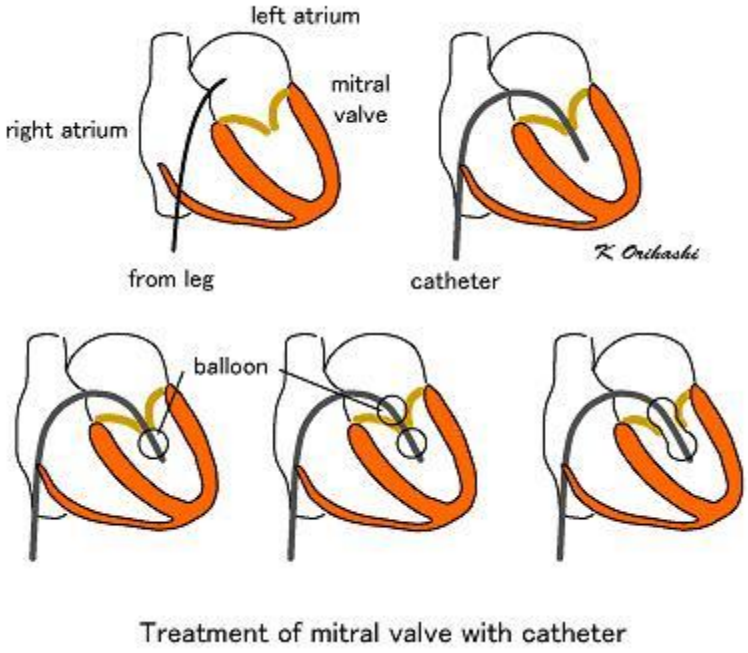
How would you manage this patient?

Medical management

- Management of atrial fibrillation

Interventional management

- Consider the following indications for intervention in a patient with mitral stenosis
  Symptomatic patients with significant mitral valve stenosis
  Patients with pulmonary hypertension even if minimally symptomatic
- The options available for management are PTMC (percutaneous trans-septal mitral valve comissurectomy) and valve replacement
- PTMC is a less invasive method but requires the following
  Isolated MS (no evidence of valvular regurgitation)
  Mitral valve should be mobile and pliable
  Left atrium free of thrombus



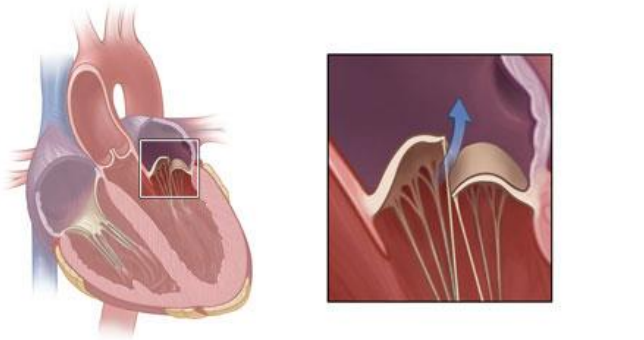
Treatment of mitral valve with catheter

Mitral regurgitation

What is the diagnosis?

What are the possible causes you would consider for this condition?

- The causes for mitral regurgitation can be classified as acute and chronic



- **Acute**
  Acute myocardial infarction
  Infective endocarditis
  Acute rheumatic carditis

  Chronic
  Mitral valve prolapse
  Rheumatic heart disease
  Cardiomyopathy

How would you clinically assess the severity of the lesion?

- Degree of left ventricular dilation
- Evidence of complications – heart failure

What are the investigations you would like to perform in this patient?

- ECG – P mitrale left ventricular hypertrophy, atrial fibrillation. Remember that if myocardial infarction is the aetiology there will be evidence of an old infarct
- CXR – Enlarged left atrium and ventricle with evidence of pulmonary congestion
- Echocardiogram confirms the diagnosis, establishes the anatomy and function of the mitral valve and assesses the severity of the lesion

How would you manage this patient?

Medical management

- Management of atrial fibrillation
- Management of heart failure

Surgical intervention

- The following are general indications for surgical intervention
  Severe acute mitral regurgitation
  Severe symptomatic chronic MR (symptoms are described based on the NYHA classification)

Asymptomatic chronic MR with evidence of progressive dilation of the left ventricle and deterioration of the ejection fraction

- Options available are mitral valve repair and mitral valve replacement

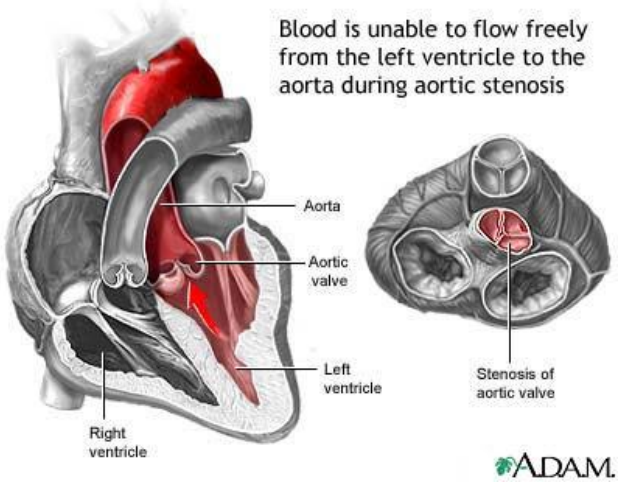
Aortic stenosis

What is the diagnosis?

What are the other lesions which can present with similar physical signs?

- Aortic sclerosis may be confused with aortic stenosis
  Aortic sclerosis affects the elderly. On examination the pulse volume is normal and the murmur is more localized

What is the probable aetiology you would consider?



- Remember that the aetiology of aortic stenosis varies according to the age of the patient
- In adolescents the lesion could be congenital
- Young adults and middle aged individuals
  Calcification of a bicuspid aortic valve
  Rheumatic heart disease
- Middle aged to elderly
  Senile degenerative aortic stenosis
  Rheumatic heart disease
  Calcification of a bicuspid aortic valve

How would you clinically determine the severity of the lesion?

- Narrow pulse pressure
- Narrow or reverse split second heart sound
- Clinical evidence of heart failure

What are the investigations you would like to perform on this patient?

- ECG – Left ventricular hypertrophy, ST segment depression and T wave inversion in advanced cases
- CXR
- Echocardiogram
  This investigation confirms the diagnosis and assesses the severity of the lesion based on valve surface area and pressure gradient across the aortic valve

How would you manage this patient?

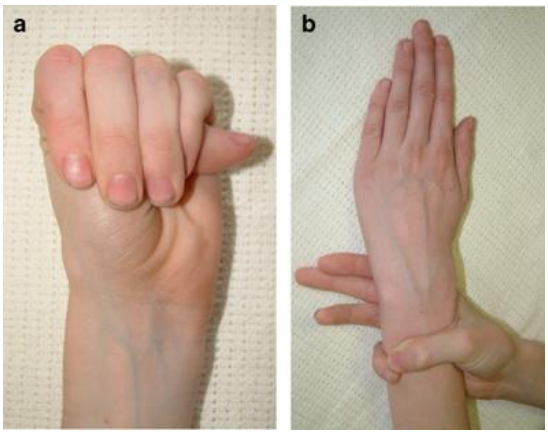
- Immediate intervention is recommended in patients with AS who develop symptoms – angina, shortness of breath, syncopal attacks as the lesion progresses rapidly to cause cardiac failure
- Asymptomatic patients with severe aortic stenosis
- Valve replacement is the treatment of choice

Aortic regurgitation

What is the diagnosis?

What are the signs you would look for in the general examination if you are suspecting the above diagnosis?

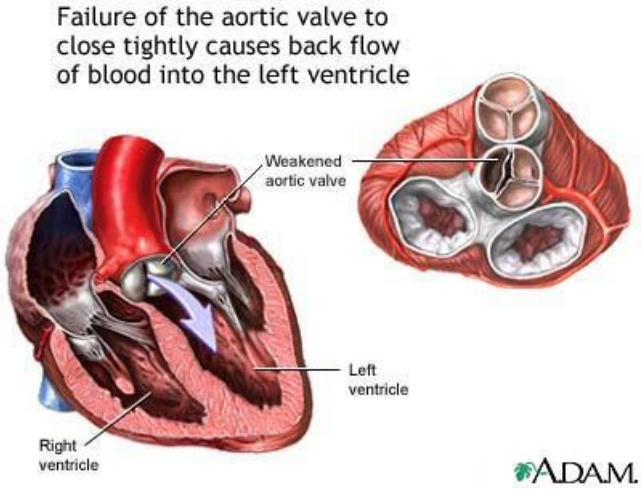
- The general examination will reveal special signs associated with AR and also give clues to the aetiology of the condition
- Head nodding (de Musset's sign)
- Visible carotid pulsations (Corrigan's sign)
- Capillary pulsations (Quincke's sign)
- Pistol shot femorals
- Look for features of Marfan's syndrome – high arched palate, arachnodactyly, arm span greater than height
- Examination of the pupils for Argyll Robertson pupil which is seen in syphilis
- Examine the joints and back for rheumatoid arthritis and ankylosing spondylitis



The diagram demonstrates the signs associated with Marfan's syndrome

What is the probable aetiology you would consider for this lesion?

- **Congenital** abnormality of the aortic valve cusps
- **Acquired**
  Rheumatic heart disease
  Infective endocarditis
  Trauma
  Aortic root dilation in – Marfan's syndrome, aortic dissection, syphilis, seronegative arthritis (ankylosing spondylitis), rheumatoid arthritis



How would you clinically assess the severity of the lesion?

- The duration and intensity of the murmur – a longer and louder murmur indicates a severe lesion
- Presence of the Austin Flint murmur (MDM heard at the apex)
- Wide pulse pressure
- Features of left ventricular failure

What are the investigations you would like to perform in this patient?

- ECG – May show left ventricular hypertrophy in advanced cases
- CXR – left ventricular dilation and aortic root dilation in certain cases
- Echocardiogram
  This is the investigation of choice for the confirmation of the diagnosis. It also assesses the severity of the lesion and may indicate an aetiology

How would you manage this patient?

- Medical management
  Manage heart failure and any associated co morbidities
- Surgical management with valve replacement should be considered in the following circumstances
  Acute AR
  Symptomatic patients
  Asymptomatic patients with progressively increasing ventricular dilation and declining left ventricular function

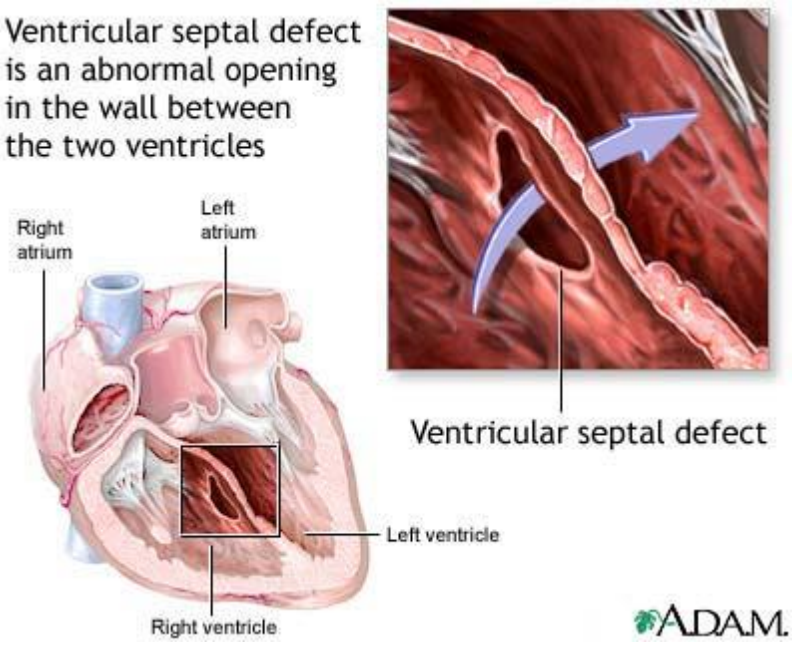
Mixed valve disease

- The most common combination given for the exam is mixed mitral valve disease. If such a case is encountered in the exam it is important to state which lesion is prominent

Ventricular septal defect

What is the diagnosis?

What are the other lesions which can present with similar clinical signs?



- The murmur of tricuspid regurgitation may mimic that of a VSD. To distinguish the two the following clinical signs are useful
- **TR**
  Large v waves (also known as cv waves) on the JVP
  Murmur increases in intensity during inspiration
  Pulsatile liver

What are the causes of a VSD?

- The commonest cause is a congenital VSD usually located in the membranous part of the interventricular septum
- But in adults VSD's are known to occur as a complication of myocardial infarction

What are the complications of a VSD?

- Cardiac failure
- Pulmonary hypertension
- Reversal of the shunt and Eisenmenger syndrome
- Infective endocarditis
- Associated valvular lesions – aortic regurgitation

What are the investigations you would like to perform in this patient?

- **ECG**
  The ECG is normal in small VSD's
  In patients with a large VSD there is evidence of left atrial and ventricular enlargement
  In patients with pulmonary hypertension there is evidence of right atrial and ventricular enlargement
- **CXR**
  The CXR is normal in patients with a small VSD
  In patients with a large VSD there is left ventricular enlargement and pulmonary plethora
  If there is associated pulmonary hypertension there is enlargement of the proximal pulmonary arteries with narrow peripheral vessels and pulmonary oligaemia
- Echocardiogram confirms the diagnosis and assesses the severity of the lesion

How would you manage this patient?

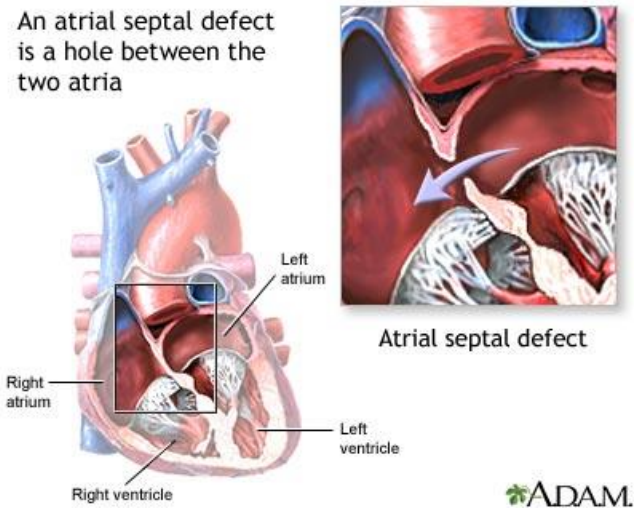
- Surgical closure of the lesion is the treatment of choice. The following are indications for surgery
  VSD with associated pulmonary hypertension
  VSD with symptoms and signs of heart failure
  Associated valvular regurgitation

Atrial septal defect

What is the diagnosis?

What are the anatomical types of ASD?

- Ostium primum defect
- Ostium secondum defect



What are the complications associated with an ASD?

- Atrial arrhythmias
- Pulmonary hypertension
- Reversal of the shunt and Eisenmenger syndrome

What are the investigations you would like to perform in this patient?

- ECG
  Usually shows right axis deviation and an incomplete RBBB
- CXR
  Shows pulmonary plethora
- Echocardiography
  This is used for confirmation of the lesion and anatomy. It also quantifies the severity of the lesion

How would you manage this patient?

- Surgical closure may be considered in symptomatic patients and those with evidence of pulmonary hypertension

Cyanotic heart disease in adults

Possible cases for the exam

- Tetralogy of Fallot
- Eisenmenger syndrome

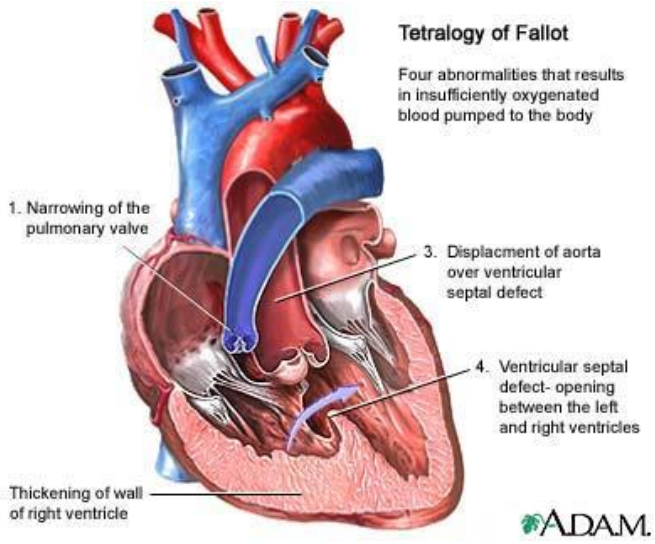
Differentiation between these two lesions

TOF	Eisenmenger syndrome
Cyanosis and clubbing Ejection systolic murmur at the mid left sternal edge Soft and single second sound	Cyanosis and clubbing (differential clubbing in Eisenmenger syndrome due to reversal of a PDA) Features of tricuspid regurgitation and pulmonary regurgitation Loud and palpable P2

Tetralogy of Fallot

What is the diagnosis?

What are the components of the lesion in TOF?



- Pulmonary infundibular stenosis
- Right ventricular hypertrophy
- Ventricular septal defect
- Overriding aorta

What are the complications associated with TOF?

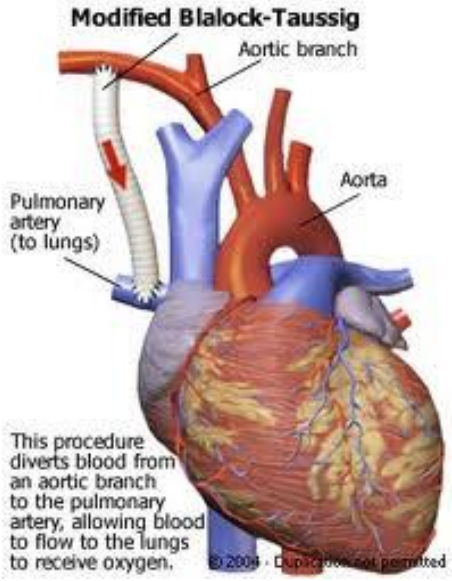
- Hypercyanotic spells
- Infective endocarditis
- Cerebral abscess
- Strokes – secondary to polycythaemia

What are the investigations you would like to perform in this patient?

- ECG – shows evidence of right ventricular hypertrophy
- CXR – shows a boot shaped heart and pulmonary oligaemia
- Echocardiogram is the investigation of choice for confirmation of the diagnosis
- Cardiac catheterization is usually performed in order to identify the anatomy before surgical correction of the lesion

What are the options for management?

- Total correction is the preferred treatment option
- However if the patient is not fit for total correction or has hypoplastic pulmonary arteries a Blalock –Taussig shunt procedure may be performed as a palliative procedure



Eisenmenger syndrome

What is the diagnosis?

What are the investigations you would like to perform in this patient?

- ECG – shows right ventricular hypertrophy
- CXR – shows dilation of the pulmonary artery with narrowing of the peripheral vessels and right ventricular enlargement
- Echocardiogram

What are the options for management of this patient?

- Monitoring and follow up
- Avoid pregnancy
- Pulmonary vasodilators

Abnormalities of the arterial pulse

Abnormalities of the arterial pulse may be classified into abnormalities of rate, rhythm and character. The following details are important and may be asked at any point during a case. Rarely examination of the pulse may be the only part of your CVS short case

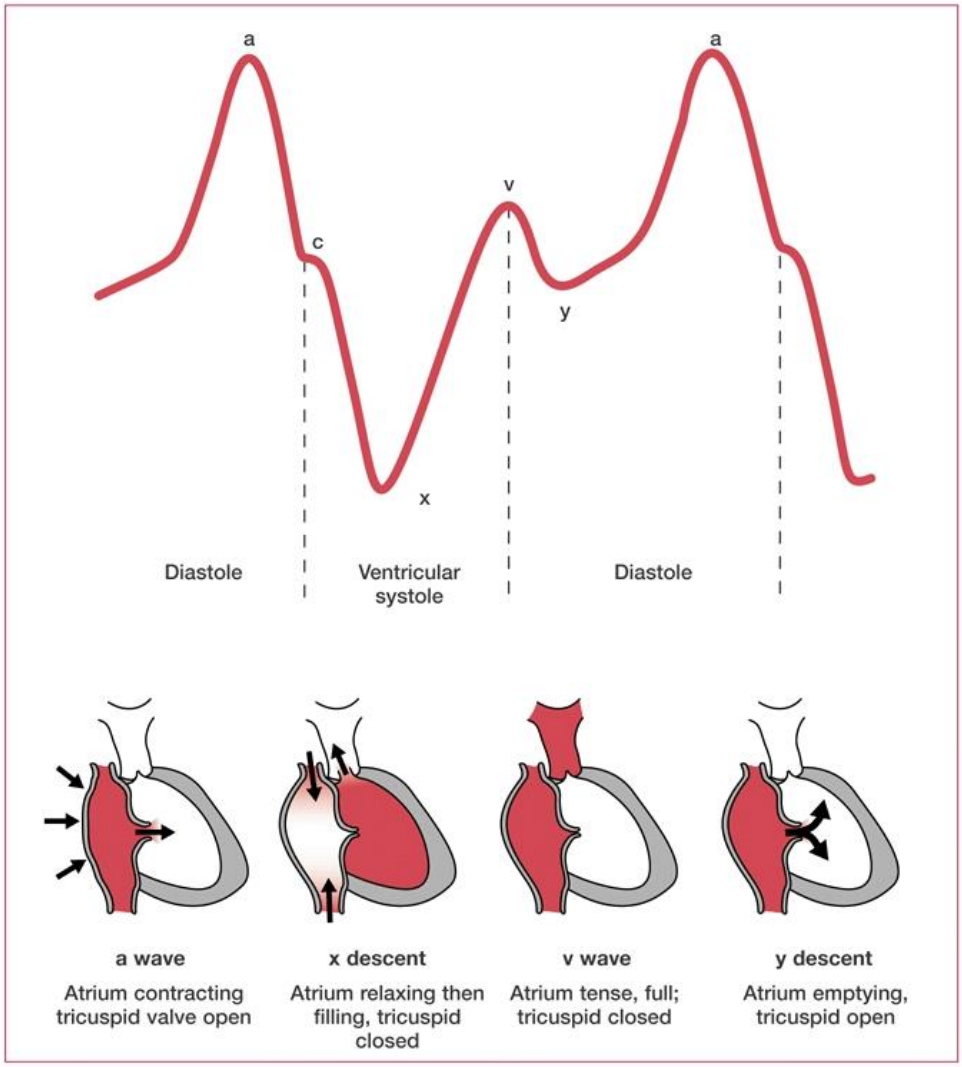
	Abnormality	Causes
Rate	Tachycardia	**Sinus tachycardia** **Arrythmias** Atrial fibrillation Atrila flutter SVT Ventricular tachycardia
	Bradycardia	**Sinus bradycardia** **Arrythmias** Sick sinus syndrome Second degree heart block Complete heart block
Rhythm	Irregular pulse	Sinus arrhythmia Atrial or ventricular ectopics Atrial fibrillation Atrial flutter with variable block
Character	Slow rising pulse	Aortic stenosis
	Collapsing pulse	Aortic regurgitation Patent ductus arteriosus A-V fistula Causes of a hyperdynamic circulation
	Pulsus alternans (Alternating small and large beats)	Severe left ventricular failure
	Bisfriens pulse	Mixed aortic valve disease
	Pulsus paradoxus (Excessive fall of pulse pressure during inspiration)	Cardiac tamponade Constrictive pericarditis

Discussion

Attach tachycardia and bradycardia management algorithms ALS here

Abnormalities of the JVP

Note



Interpretation of the JVP is extremely difficult at undergraduate level but the following points may be asked during a discussion

Abnormality	Causes
Non pulsatile raised JVP	SVC obstruction
Loss of “a” wave	Atrial fibrillation
Prominent “a” wave	Tricuspid stenosis Pulmonary hypertension
Cannon “a” wave	Complete heart block
“cv” waves	Tricuspid regurgitation
Steep “x” and “y” descent	Constrictive pericarditis

Respiratory system

Examination routine

- Introduce yourself and take consent
- Ensure adequate exposure and request for a chaperone if necessary
- Proper positioning of the patient at 45 degrees and make sure that the patient is comfortable

Inspection

- **Go to the foot end of the bed**
  Look around for any clues
  Get a general impression of the patient. Is he/she wasted? Are there signs of respiratory distress?
  Look at the chest for any asymmetry, abnormal shape, deformities and surgical scars
  Look for any apical flattening – apical fibrosis
  Observe the chest movements
  Count the respiratory rate

General examination

- **Do a general examination relevant to the respiratory system**
  Face
  Febrile/not
  Look for central cyanosis, pallor, evidence of Horner's syndrome
  Neck
  Look at the neck veins, assess JVP, look for cervical lymphadenopathy assess the position of the trachea and the distance between the cricoid and the suprasternal notch (These last 3 steps require the patient to sit up and may be done at a later stage- see examination outline)
  Upper limbs
  Look for clubbing, cyanosis and nicotine stains in the fingers. Examine the pulse for a bounding pulse – carbon dioxide retention
  Look for flapping tremors – respiratory failure
  Look for a mantoux scar
  Lower limbs
  Look for lower limb edema – cor pulmonale

Respiratory system proper

- **Move on to the examination of the respiratory system proper. Ask to start the examination from the back of the chest as this will reveal the most physical signs**

Palpation

- **Examine the location of the apex**
- **Feel for a palpable P2 which indicates pulmonary hypertension**
- Examine the chest movements and look for any asymmetry
- Examine the tactile vocal fremitus
- Remember to examine all zones of the lung

Percussion

- Percuss in all zones of the lung
- Percuss over the liver to look for a pushed down liver dullness

Auscultation

- Auscultate the 3 zones of the lung and listen to the breath sounds. Is there vesicular breathing or bronchial breathing? Are there any added sounds?
- Examine the vocal resonance
- Remember to examine all zones of the lung

Interpretation of individual signs in the respiratory system

	Sign	Causes
General examination	Clubbing	**Infective** Empyema Lung abscess Bronchiectasis **Neoplastic** Bronchial carcinoma **Other** Fibrosing alveolitis
Trachea	Deviation towards the lesion	

	Deviation away from the lesion	
Auscultation **Breath sounds**		
Quality	Bronchial breathing	Consolidation Localized area of fibrosis Upper limit of a pleural effusion
Intensity	Low intensity	Pleural effusion Consolidation Pneumothorax Collapse Fibrosis
Added sounds	**Crepts** Early inspiratory	Pneumonia, bronchiectasis
	Late inspiratory	Fibrosing alveolitis, pulmonary edema
	Ronchi	Asthma, COPD

Pleural effusion

What are the possible causes you would like to consider?

- Pleural effusions can be categorized into transudative effusions and exudative effusions. Exudative effusions are commonly unilateral while transudative effusions are commonly bilateral

Transudative effusion	Exudative effusion
Nephrotic syndrome	**Infective**
Cardiac failure	Parapneumonic effusion
Hepatic failure	Empyema
Hypothyroidism	Tuberculosis
	Malignant
	Bronchial carcinoma
	Secondary deposits in the pleura
	Lymphoma
	Mesothelioma
	Connective tissue disorders
	Rheumatoid arthritis
	SLE
	Rare
	Pulmonary embolism
	Pulmonary infarction
	Subphrenic abscess

What are the investigations you would like to perform in this patient?

- Investigations should be performed in this patients with the following objectives in mind
  Confirmation of the diagnosis
  Assessment of the severity
  Determination of the composition
  Determination of the underlying cause
- Chest X ray PA and lateral
- Pleural aspiration

Send for – full report, cytology, glucose, gram stain and culture, other special investigations (AFB, LDH, adenosine deaminase)

Investigation	Interpretation
Full report	Neutrophills are raised in acute pleural inflammation – parapneumonic effusion Lymphocytes may be raised in malignancy and TB
Glucose	Glucose content is reduced in parapneumonic effusion, empyema, TB and malignancy
Protein	Important in differentiating between exudative and transudative effusions

How would you differentiate between an exudative and a transudative effusion?

- The protein content in an exudative pleural fluid is >3g/l
- However for a more accurate diagnosis Light's criteria is used
  Pleural fluid protein: serum protein > 0.5
  Pleural fluid LDH: serum LDH > 0.6
  Pleural fluid LDH more than 2/3 of the upper limit of normal serum LDH
- If 2 or more of the above criteria are met the effusion is classified as exudative

Discuss the principles of management of a pleural effusion

- The basic principles of management are based on the underlying cause of the effusion. The following table gives a guide to the management

	Parapneumonic	Empyema	TB	Malignant
Diagnosis	History of pneumonia	Is an infection of the pleural space Is characterized by pH <7.2 LDH > 1000 Neutrophills ADA	History suggestive of TB Demonstration of organism in pleural fluid	Systemic features Malignant cells in cytology
Management	Simple aspiration	Aspiration to dryness With adequate antibiotic cover	Treatment regimen similar to that of pulmonary TB	Observe if asymptomatic Therapeutic aspiration Pleurodesis

Consolidation

What are the causes you would like to consider in this case?

- The causes for consolidation of the lung are
  Bacterial pneumonia
  Bronchial carcinoma
  Pulmonary infarction
- The most likely cause is bacterial pneumonia

What are the investigations you would like to perform in this patient?

- CXR for confirmation of the diagnosis
- As the most likely cause for consolidation is pneumonia the further investigations would target this
- FBC – to look for neutrophil leucocytosis
- Blood culture and ABST
- Sputum culture and ABST

What are the principles of management of suspected bacterial pneumonia?

- Assess the severity
  This is usually done by the CURB 65 criteria
  Confusion
  Urea > 7mmol/l
  Respiratory rate > 30 breaths per minute
  Blood pressure (Systolic <90 or diastolic <60)
  Age > 65
- A patient who scores 2 or more of the above criteria should be hospitalized
- Obtain the required samples for cultures and commence empirical antimicrobial therapy. These are usually given intravenously but can be switched to oral antibiotics once the patient is clinically better
- Monitor the vital parameters of the patient and also the response to the antibiotics
- The patient should begin to respond within 48-72 hours of initiation of therapy
- Continue antibiotics for 5-7 days
- CXR may be repeated after about 7-12 days

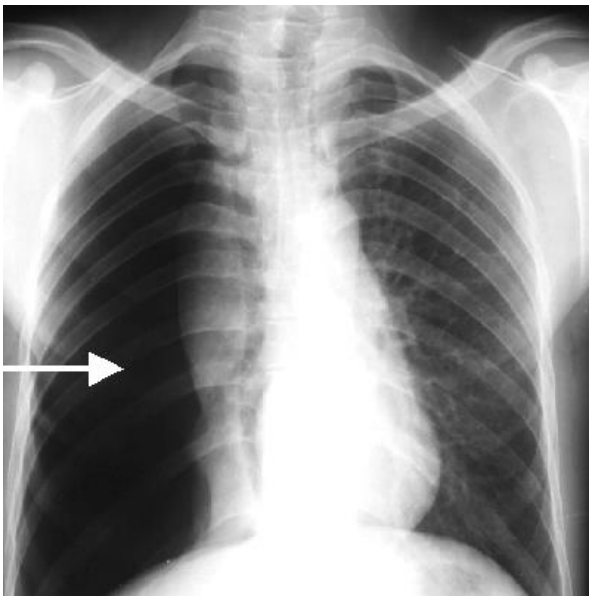
What will you do if the patient fails to respond to the treatment?

- Reconsider the diagnosis
- Reconsider the choice and/ or dose of antibiotics

- Investigate for rare organisms causing pneumonia
- Think of the probability of a malignancy

Pneumothorax

What are the causes you would like to consider in this patient?



- The causes of pneumothorax can be classified as follows
- Spontaneous
  Primary – Seen in patients who do not have a previous history of lung disease. Commonly in tall thin individuals
  Secondary – In patients with lung disease, COPD, bronchial asthma
- Traumatic

How would you manage this patient?

- Assess the severity of the condition
  Immediate aspiration may be indicated without radiographic confirmation in a patient with a tension pneumothorax – severe dyspnoea, marked mediastinal shift, hypotension and bradycardia
- Confirm the diagnosis with CXR PA and lateral
- **Primary pneumothorax**
  Aspiration is recommended if the patient is breathless and/or there is a rim of air >2cm on the chest radiograph
  IC tube insertion may be considered if recurrent aspiration is unsuccessful
- **Secondary pneumothorax**
  Is considered to have a worse prognosis. Therefore the management plan is different
  IC tube insertion is recommended if the patient is breathless + >50 years + rim of air >2cm on CXR
  If the above criteria are not met simple aspiration may be attempted

Lung fibrosis

What are the causes you would consider for pulmonary fibrosis?

The causes of lung fibrosis can be classified as follows

Upper lobe fibrosis	Lower lobe fibrosis
TB	Rheumatoid arthritis
Ankylosing spondylitis	Scleroderma
Sarcoidosis	Asbestosis
Silicosis	Pneumoconiosis
Radiation	Paraquat poisoning
Drugs	

Diffuse parenchymal lung disease

- Are a heterogeneous group of conditions which share similar symptoms pulmonary signs, pulmonary function abnormalities and radiological changes
- **Clinical**
  Exertional dyspnoea, cough with minimal sputum production
  Clubbing
  Fine, end inspiratory bi basal crepitations

  CXR
  Reticulonodular shadowing
  With advanced disease
  "honeycombing" of the lung

  Pulmonary function testing
  Restrictive pattern with reduced diffusing capacity of CO

- **Classification**

Category	Further classification and causes
DPLD of known cause or associations	**Connective tissue diseases** SLE, rheumatoid arthritis, scleroderma **Drugs** Amiodarone Chemotherapeutic agents

	Antirheumatic agents – gold, penicillamine **Environmental exposures**
Idiopathic interstitial pneumonias	Idiopathic pulmonary fibrosis (formerly known as fibrosing alveolitis) Other
Granulomatous DPLD	Sarcoidosis
Other rare forms of DPLD	Histiocytosis X

What are the investigations you would like to perform in this patient?

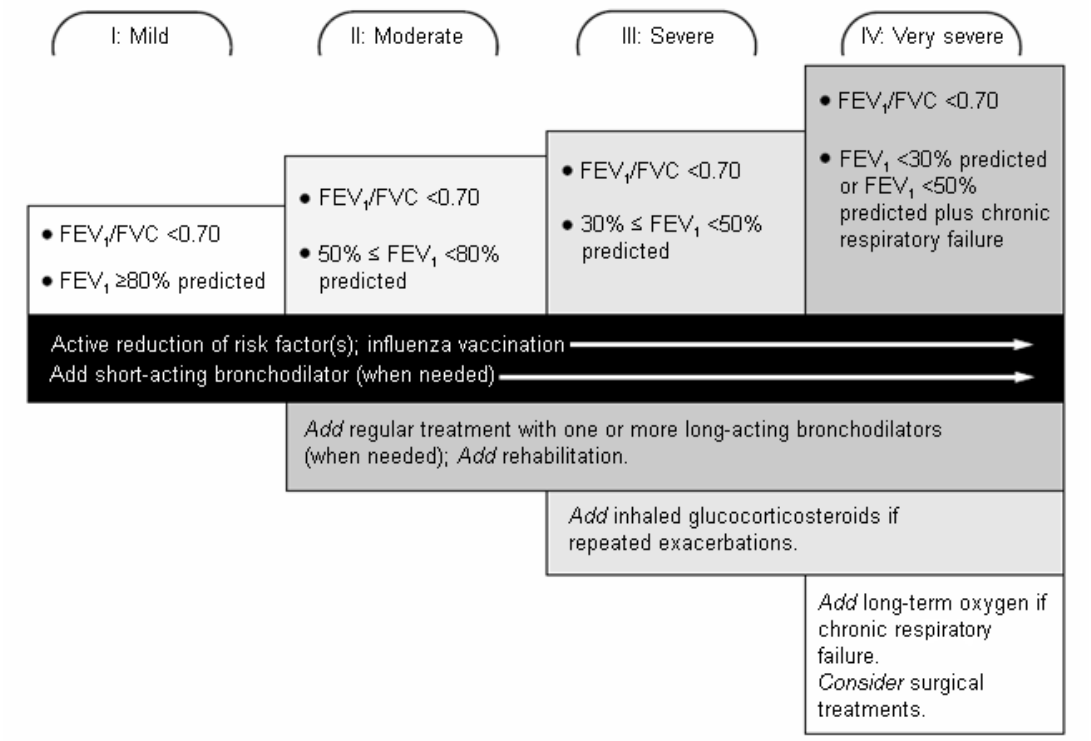
- The following investigations are used in the investigative process of a suspected DPLD
  Imaging
  CXR
  HRCT

  Pulmonary function tests
  Bronchoscopy – Bronchoalveolar lavage
  Consider lung biopsy

COPD

What are the principles of management of this patient?

- Given below is a diagram depicting the management of COPD.



Bronchiectasis

What are the causes you would like to consider in this patient?

- Congenital
  Cystic fibrosis
  Ciliary dysfunction syndromes
  Primary hypogammaglobulinaemia
- Acquired
  Pneumonia (complicating pertussis or measles)
  Tuberculosis
  Bronchial carcinoma

What are the investigations you would like to perform in this patient?

- CXR
  Ring shadows and tramline shadows
- HRCT
- Sputum culture

What are the principles of management of this patient?

- Chest physiotherapy and postural drainage
- Antibiotics
- Bronchodilators
- Surgical options

Abdominal examination

Examination routine

- Introduce yourself and take consent
- Ensure adequate exposure and request for a chaperone if necessary
- Position the patient adequately on the bed with one pillow for the head and the arms comfortably resting at the sides

General examination relevant to the abdomen

- **Face**
  Look for pallor, Icterus, xanthelasmata
  Perioral pigmentation
  Oral ulcers
  Angular stomatitis
  Glossitis
- **Chest**
  Gynaecomastia
  Spider naevi
- **Upper limbs**
  Hands
  Clubbing, leukonychia, koilonychia
  Palmar Erythema
  Dupuytren's contracture
  Flapping tremors
  Arms
  A-V fistulae
  Axillary hair
- **Lymph nodes**
  Examine both cervical and axillary nodes
- **Lower limbs**
  Ankle edema
- **Skin**
  Rashes

Inspection of the abdomen

- Go to the foot end of the bed and start your inspection from there
- Look for the following
  Distension
  Symmetry of movement of the abdomen
- Now come closer and look for the following details

Surgical scars
Dilated veins
Visible pulsations
State of the umbilicus
Any obvious masses or visible peristalsis
Hernial orfices

Palpation

- Start the palpation of the abdomen with a superficial palpation moving from quadrant to quadrant. Always keep your eye on the patient's face for tenderness
- Palpate for the major organs in the abdomen
  Liver
  Percuss for the upper and lower borders
  Spleen
  Kidneys
- Ask for permission to examine the groin and external genitalia

Percussion

- Remember to complete the percussion of any abdominal mass detected then and there
- At this stage percuss for free fluid. Initially look for flank and shifting dullness and then go on to look for horseshoe shaped dullness
- If you suspect gross ascites, look for the fluid thrill

Auscultation

- Examine for a bruit over the enlarged liver
- Examine for a splenic rub
- Examine for a renal bruit
- Examine the bowel sounds

General points on the presentation and discussion

- The usual cases which will be given for the exam usually involve palpable organs. Therefore one should know how to present a liver, spleen and renal mass
- See the table below for the method of presentation

Liver	Spleen	Renal mass
Right hypochondrial mass Cannot get above Moves with respiration Dull to percussion over the mass and continues with the liver dullness	Left hypochondrial mass Cannot get above Moves diagonally with respiration Notch felt/not Dull to percussion over the mass and the dullness is continuous with the splenic dullness	Mass in the loin Can get above (If the mass is very large you may not be able to get above it) Moves with respiration Ballottable Dull to percussion over the mass but with the presence of resonant bands
Mention the distance from the costal margin to the lower border of the liver in the mid clavicular line Mention the site of the upper border of the liver (mid clavicular line State the span of the liver	Mention the distance from the costal margin to the tip of the spleen	Measure the size of the lump from upper to lower pole
Describe the lower border of the liver, the surface and the consistency	Describe the consistency of the spleen	Describe the consistency
Mention if there is a hepatic bruit/not	Mention if there is a splenic rub/not	

- The next step is to know the differential diagnosis for hepatomegaly, splenomegaly, hepatosplenomegaly and renal mass
- Study the following table for the differential diagnosis

Narrowing down your differential diagnosis

Jaundice

	Pre hepatic	Hepatic	Cholestatic
Clinical findings	Look for pallor	Look for signs of chronic liver disease	Scratch marks Cachexia
Causes	Hemolytic anaemia	Chronic liver disease Viral hepatitis Drug induced hepatitis Metabolic	**Intrahepatic cholestasis** Billiary cirrhosis Sclerosing cholangitis Drug induced **Extrahepatic** Pancreatic carcinoma Gallstones

Hepatomegaly

Clinical picture	Diagnosis
Febrile patient +/- jaundice	Infective hepatitis
Peripheral stigmata of chronic liver disease	Cirrhosis
Hard nodular liver +/- peripheral stigmata of chronic liver disease (There may be a hepatic bruit)	Hepatocellular carcinoma
Pulsatile liver	Heart failure, tricuspid regurgitation

Splenomegaly

Based on the size of the spleen

Massive splenomegaly >8cm or crossing the midline	Moderate splenomegaly 4-8cm	Mild splenomegaly Just palpable or 2-4cm
CML Myelofibrosis Chronic malaria Visceral leishmaniasis	Myeloproliferative disorders Lymphoma CLL Cirrhosis with portal hypertension Hemolytic anaemia (Thalassemia)	Myeloproliferative disorders Lymphoma CLL Cirrhosis with portal hypertension **Infections** Glandular fever IE Typhoid **Hemolytic anaemia**

Clinical picture	Diagnosis

Pallor	Hemolytic anaemia
Lymphadenopathy +/- Pallor	Leukaemia, lymphoma

Hepatosplenomegaly

Clinical picture	Diagnosis
Pallor	Hemolytic anaemia Myeloproliferative disease Leukaemia, lymphoma
Lymphadenopathy	Leukaemia, lymphoma
Peripheral stigmata of liver disease	Cirrhosis with portal hypertension

Possible discussion topics from a case of hepatomegaly, splenomegaly or hepatosplenomegaly

Cirrhosis

Hepatocellular carcinoma

Infective diseases of the liver

Hemolytic anaemias

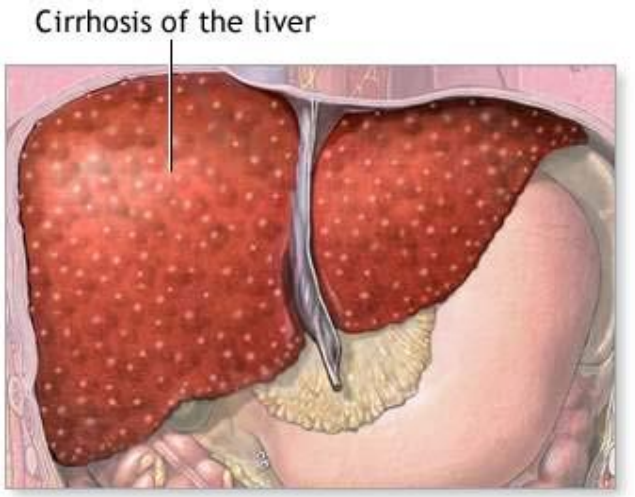
Hematological malignancy

Cirrhosis

- Is a pathological term used for progressive diffuse fibrosis of the liver with distortion of the hepatic architecture and formation of regenerative nodules

What are the possible causes you would like to consider in this patient?

- Alcohol
- Non alcoholic fatty liver disease
- **Infective**
  Chronic viral hepatitis
- **Immune**



Autoimmune hepatitis

- **Bililary**
  Primary billiary cirrhosis
  Secondary billiary cirrhosis
  Sclerosing cholangitis
- **Genetic**
  Wilson's disease
  Hereditary hemochromatosis
  Alpha 1 antitrypsin deficiency

What are the complications of cirrhosis of the liver?

- Portal hypertension leading to variceal hemorrhage
- Ascites and spontaneous bacterial peritonitis
- Hepatic encephalopathy
- Hepatorenal syndrome
- Coagulopathy
- Hepatocellular carcinoma

How would you clinically assess the severity of cirrhosis in this patient?

- Severity is assessed by the presence of features of decompensation. These are as follows
  Jaundice
  Hepatic encephalopathy
  Ascites
  Bleeding
  Portal hypertension

What are the investigations you would like to perform in this patient?

- Investigations should be performed to confirm the diagnosis, look for an underlying cause and to assess the severity and prognosis
- **FBC**
  To look for pancytopenia – this could indicate hypersplenism
- **Liver function tests –** these are used to assess severity and prognosis
  Serum bilirubin
  AST/ALT
  PT/INR
  Serum albumin
- **Ultrasound scan of the abdomen**
  This is used to visualize the liver, look for splenomegaly and ascites
- **Investigations to look for a cause**
  Remember that alcoholic liver disease is the most common cause, but other causes may be considered if the history is not suggestive
  Hepatitis B serology

Serum autoantibodies
Serum iron and ferretin – Haemochromatosis
Serum ceruloplasmin – Wilson's disease
Liver biopsy

What are the principles of management?

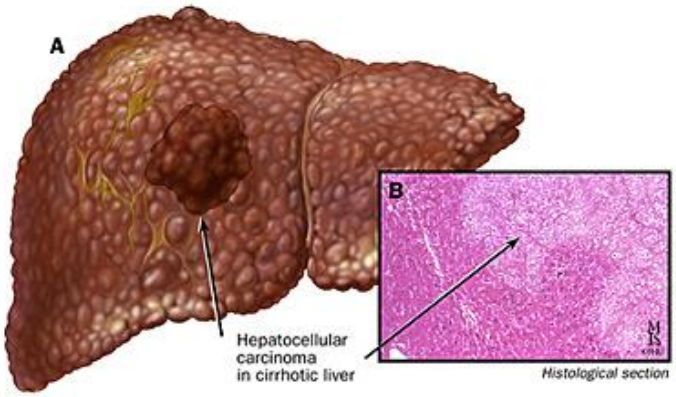
- Treat the underlying cause if possible
- Manage complications
  Ascites
  Hepatic encephalopathy
  Variceal bleeding
  (See long cases in medicine for a further discussion on these topics)
- Liver transplantation
  This is based on a scoring system – i.e. Child-Pugh classification or MELD scoring system

Hepatocellular carcinoma

What are the causes you would consider in this patient?

- Remember that most of the time hepatocellular carcinoma occurs in a cirrhotic liver. Therefore all causes for cirrhosis may be considered

What are the investigations you would like to perform in this patient?



- **Ultrasound scan of the abdomen**
  This will detect any focal lesions in the liver
- **Serum markers**
  Alpha fetoprotein
- Remember that liver biopsy is not indicated as seeding of the tumor can occur along the biopsy tract

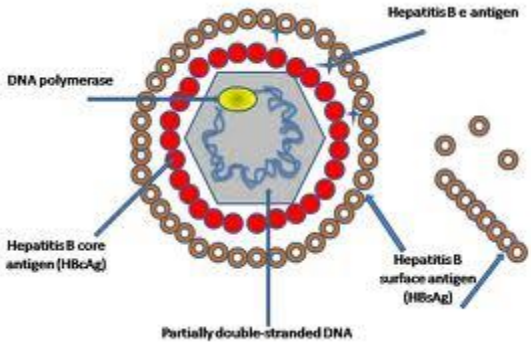
What are the options for management in this patient?

- The therapeutic options for management are
  Resection
  Liver transplant – preferred in patient with cirrhotic livers
  Percutaneous ablation

TACE (Transarterial chemo-embolization)
Chemotherapy

Viral hepatitis

- Is caused mainly by hepatitis viruses A-E but can also be caused by other viruses such as CMV and EBV
- The clinical course is usually described in 4 phases. These are the incubation, prodrome, icteric phase and recovery phase
- Investigations in the initial phase
  FBC – Decreased WCC with a relative lymphocytosis
  Liver function tests – Elevated AST with normal serum bilirubin
- Specific investigations
  Include those for the diagnosis (See table below)
- Management is usually supportive



	Microbiology	Complications	Investigations	Management and Prevention
Hepatitis A	RNA virus Transmission is via the faeco-oral route	Acute fulminant hepatitis	**Prodrome** HAV in stool (by electron microscopy or RNA detection. This is not commonly used **IgM HAV** Positive at the onset of symptoms. Persists for about 4-6 months	Personal hygiene Vaccination
Hepatitis B	DNA virus Transmission is by the intravenous route, sexual contact Vertical transmission can also occur	Chronic infection Asymptomatic carrier Cirrhosis Hepatocellular carcinoma Acute fulminant	**HBsAg and positive IgM anti-HBc** Indicates acute hep B infection **HBeAg** Indicates severe	Vaccination

		hepatitis is rare	infection **HBsAg persisting for more than 6months with positive IgG anti-HBc and negative IgM anti-HBc** Indicates chronic infection	
Hepatitis C	RNA virus Transmission is via the intravenous route and by sexual contact	Higher risk of developing chronic liver disease, cirrhosis and hepatocellular carcinoma	**Anti HCV**	Vaccine?
Hepatitis D	Incomplete RNA particle Can only replicate in the presence of hepatitis B infection This can be superinfection (infection in a person already having hep B) or coinfection	Acute fulminant hepatitis more common in coinfection Chronic liver disease more common in superinfection	**Co infection** IgM anti HDV and IgM anti HBc **Superinfection** IgM anti HDV and IgG anti HBc	Vaccine?
Hepatitis E	RNA virus Similar to Hep A	Fulminant hepatitis	HEV RNA in stools	No effective prophylaxis

Hemolytic anaemia

- Basic investigation of a suspected hemolytic anaemia starts with the FBC and blood picture. The blood picture has a specific appearance according to the type of hemolytic anaemia
- **Other investigations**
  Reticulocyte count - Increased reticulocytes

Unconjugated hyperbilirubinaemia
Hemoglobinuria and hemosiderinuria in intravascular hemolysis
Bone marrow – erythroid hyperplasia

- After confirmation of a hemolytic anaemia investigations should focus on finding the cause or underlying pathology
- Classification of hemolytic anaemia is into congenital and acquired

Congenital	Acquired
Membrane defects Hereditary spherocytosis Hereditary elliptocytosis	**Immune** **Autoimmune** Warm Cold **Alloimmune** Transfusion reactions Hemolytic disease of the newborn
Metabolic defects G6PD deficiency PK deficiency	**Red cell fragmentation syndromes** Prosthetic valves HUS, TTP, DIC
Disorders of hemoglobin Thalassemia Sickle cell anaemia	**Systemic disease** **Infection** **Toxins**

Congenital hemolytic anaemias

Disease	Investigations	Management
Hereditary spherocytosis (AD)	Blood film – Spherocytes Osmotic fragility test	Definitive management is splenectomy which is usually planned when the child is 5-6 years of age In the meantime the management is based on symptoms with severe anaemia managed by blood transfusion
G6PD deficiency (X linked)	Hb normal between attacks Blood film – Bite cells, blister cells, Heinz bodies Features of intravascular hemolysis G6PD levels in the RBC	Is precipitated by antioxidant drugs and substances Avoiding these is the most important aspect of the management A crisis can be treated with blood transfusions as necessary
Thalassemia **β – Thalassemia major**	Blood film - microcytic hypochromic anaemia Target cells, nucleated red blood cells Reticulocyte count may be low Serum iron studies	Management is with recurrent blood transfusions Consider splenectomy **Monitor for the complications of iron overload** Cardiomyopathy

	Bone marrow – Erythroid hyperplasia Serum hemoglobin electrophoresis- Absent HbA with increased HbF and HbA2	Liver disease Endocrine organ dysfunction – growth, hypothyroidism, diabetes **Iron chelation therapy** Subcutaneous desferrioxamine Can cause auditory and ophthalmological side effect **Counseling and parent education**
Sickle cell anaemia	Blood film - Sickle cells, Howell-Jolly bodies Sickling test Hb electrophoresis - HbS	Rare in SL Avoid precipitants Management of crisis with analgesia, adequate fluid and transfusion

Acquired hemolytic anaemia

	Warm AIHA	Cold AIHA
Type of antibody	IgG	IgM
Causes	Idiopathic Autoimmune – SLE Lymphoma CLL Drugs – Methyldopa	Idiopathic Infections – EBV, CMV Mycoplasma
Investigations	Those of hemolytic anaemia Spherocytes on the blood film	Those of hemolytic anaemia Less spherocytes Cold agglutination test
Management	Treat cause Blood transfusion of necessary Steroids Immunosuppressive drugs – Azathioprine, cyclophosphamide IVIg Splenectomy is also a final option of there is poor response to the medical management	Treat cause Keep warm

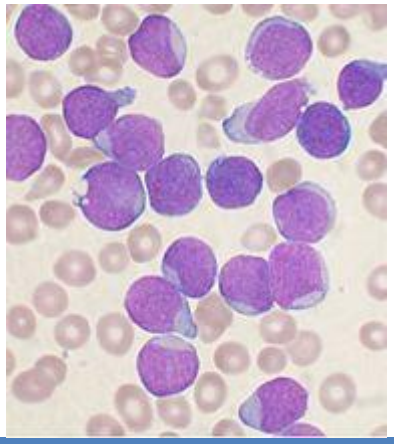
Hematological malignancy

Leukaemias

- Classified as acute and chronic leukaemias
- In acute leukaemia there is proliferation of primitive stem cells causing accumulation of blasts in the bone marrow

In chronic leukaemia the malignant cells retain the ability to differentiate causing accumulation of cell of various levels of differentiation

- Acute leukaemia occurs at all ages. ALL has a peak incidence at 1-5 years
- Chronic leukaemia occurs in middle and old age individuals



Acute leukaemia

	ALL	AML
Clinical presentation	Features of bone marrow failure Organ infiltration	Features of bone marrow failure Organ infiltration Can have other features DIC, gum hypertrophy
Associations	Trisomy 21 Translocation (9:22)	
Investigations	FBC – Pancytopenia Blood picture – Blasts Bone marrow - >30% blast cells Special stains and classification tests	Similar Positive for myeloperoxidase stain
Management	General supportive therapy Chemotherapy **Induction of remission** Prednisilone **Consolidation of remission** Intensive multi agent chemotherapy CNS prophylaxis **Intensification** **Maintenance chemotherapy**	General supportive therapy **Induction of remission** **Consolidation**

Chronic leukaemia

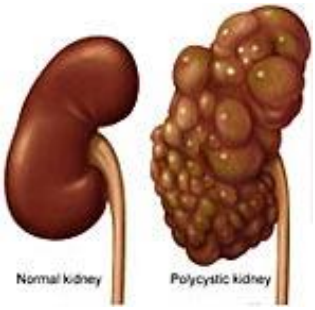
	CML	CLL
Clinical presentation	Presents usually with insidious features Can also present mimicking an acute leukaemia. This is known as a blast crisis	Presents with insidious features

Associations	Philadelphia chromosome (95%)	
Investigations	**FBC** High leucocyte count Normocytic normochromic anaemia Thrombocytosis **Blood picture** Full range of granulocyte precursors and mature neutrophils	**FBC** Lymphocytosis Associated autoimmune hemolytic anaemia **Blood picture** Lymphocyte precursors and mature lymphocytes
Management	This varies on the stage of the disease **Chronic stage** Tyrosine kinase inhibitors - Imatinib **Accelerated and blast phase** Tyrosine kinase inhibitors if the patient has not already been given one Hydroxycarbamide	Has a better prognosis. Treatment is only required if the patient is progressively symptomatic or if there is evidence of marrow failure Chlorambucil

Lymphomas

	Hodgkin's lymphoma	Non Hodgkin's lymphoma
Clinical	Lymphadenopathy usually begins from 1 group of peripheral lymph nodes and spreads contiguously to the others Can have mediastinal involvement Extra nodal spread rare Leukaemic phase rare Constitutional symptoms common	Has a more unpredictable and haphazard spread Involves oropharyngeal lymph nodes Extra nodal spread common Leukaemic phase more common Constitutional symptoms rare
Investigations	Lymph node biopsy shows Reed – Sternberg cells	No RS cells
Management	**Early stage disease** Radiotherapy **Advanced disease** Chemotherapy +/- radiotherapy	Multi agent chemotherapy

Polycystic kidney disease



What are the main genetic categories of PCKD?

- Autosomal dominant PCKD – presents with renal failure in adults
- Autosomal recessive PCKD – presents with renal failure in childhood

What are the complications known to be associated with PCKD?

- **Renal complications**
  Renal failure
  Hypertension
  Cyst rupture
  Cyst infection
  Renal calculi
  Urinary tract infection
- **Extra renal complications**
  May be associated with cysts in other sites – liver, ovary, pancreas
  Berry aneurysms

Ascites

What are the causes you would like to consider in this patient?

- There are 2 clinical patterns of ascites encountered in clinical practice. These are
- **Ascites out of proportion to ankle edema**
  Portal hypertension
  Intra abdominal malignancy
  Tuberculous peritonitis
  Constrictive pericarditis
- **Ascites as a part of generalized edema**
  Heart failure
  Nephrotic syndrome

How would you clinically evaluate this patient for a cause?

Clinical pattern	Cause	Further examination
Ascites out of proportion to ankle edema	Portal hypertension	Stigmata of chronic liver disease, splenomegaly
	Intra abdominal malignancy	Ovarian carcinoma is a recognized cause. Therefore suspect this in cachetic females
	Constrictive pericarditis	Steep x and y descent in the JVP Pulsus paradoxus Pericardial knock
Ascites as a part of generalized edema	Heart failure	Elevated JVP Hepatomegaly
	Nephrotic syndrome	Ask to examine the urine for protein

What are the investigations you would like to perform in this patient?

- Ultrasound scan of the abdomen – to visualize the intra abdominal organs
- UFR
- Tumor markers – CA 125 in females
- Diagnostic peritoneal tap – full report, cytology

How would you determine whether the ascitic fluid is exudative or transudative?

- This is based on the serum albumin – ascitic fluid gradient (SAAG)
- If the SAAG is <1.1g/dl it is exudative
- If it is more than 1.1g/dl it is transudative

What are the principles of management?

- Treat the underlying cause
- Symptomatic treatment of ascites
  Dietary management with salt restriction
  Diuretics – spiranolactone and frusemide
  Therapeutic paracentesis

Nervous system

Examination routine

Examination of the cranial nerves

- Introduce yourself and take consent
- Ask for a chaperone if required
- Look for any obvious abnormalities – ptosis, squint, facial asymmetry

Cranial nerve 1

- Routinely not tested but the patient can be asked on his/her sensation of smell

Cranial nerve 2

- Start with examination of the visual acuity using a pocket Snellen chart. If the patient cannot see this go on to the finger counting method
- Examine the visual fields using the confrontation method. The technique of examination is extremely important
- Examine the pupils – size, shape, symmetry, direct and consensual light reflex and accommodation reflex
- Ask the examiner for the ophthalmoscope to examine the fundi

Cranial nerves 3, 4 and 6

- Look for ptosis
- Examine the eye movements in all directions
- Note any nystagmus

Cranial nerve 5

- **Motor**
- Test the masseters by asking the patient to clench his/her teeth. Feel the muscle bulk of the contracting muscle
- Test the pterygoids by asking the patient to open his/her mouth. Look for any deviation. Test again after applying resistance
- Test the jaw jerk
- **Sensory**
- Examine the facial sensation
- Examine the corneal reflex

Cranial nerve 7

- Test the muscles of facial expression in the upper and lower half of the face respectively

- Taste is not tested routinely

Cranial nerve 8

- This is also not routinely examined at the short cases
- Whispering test
- Rinne's and Weber's test

Cranial nerve 9 and 10

- Ask the patient to open his/her mouth. Observe the symmetry of the palate
- Ask the patient to say "aah" and look for any deviation of the palate or a lack of movement

Cranial nerve 11

- Examine the trapezius and sternocleidomastoid muscles

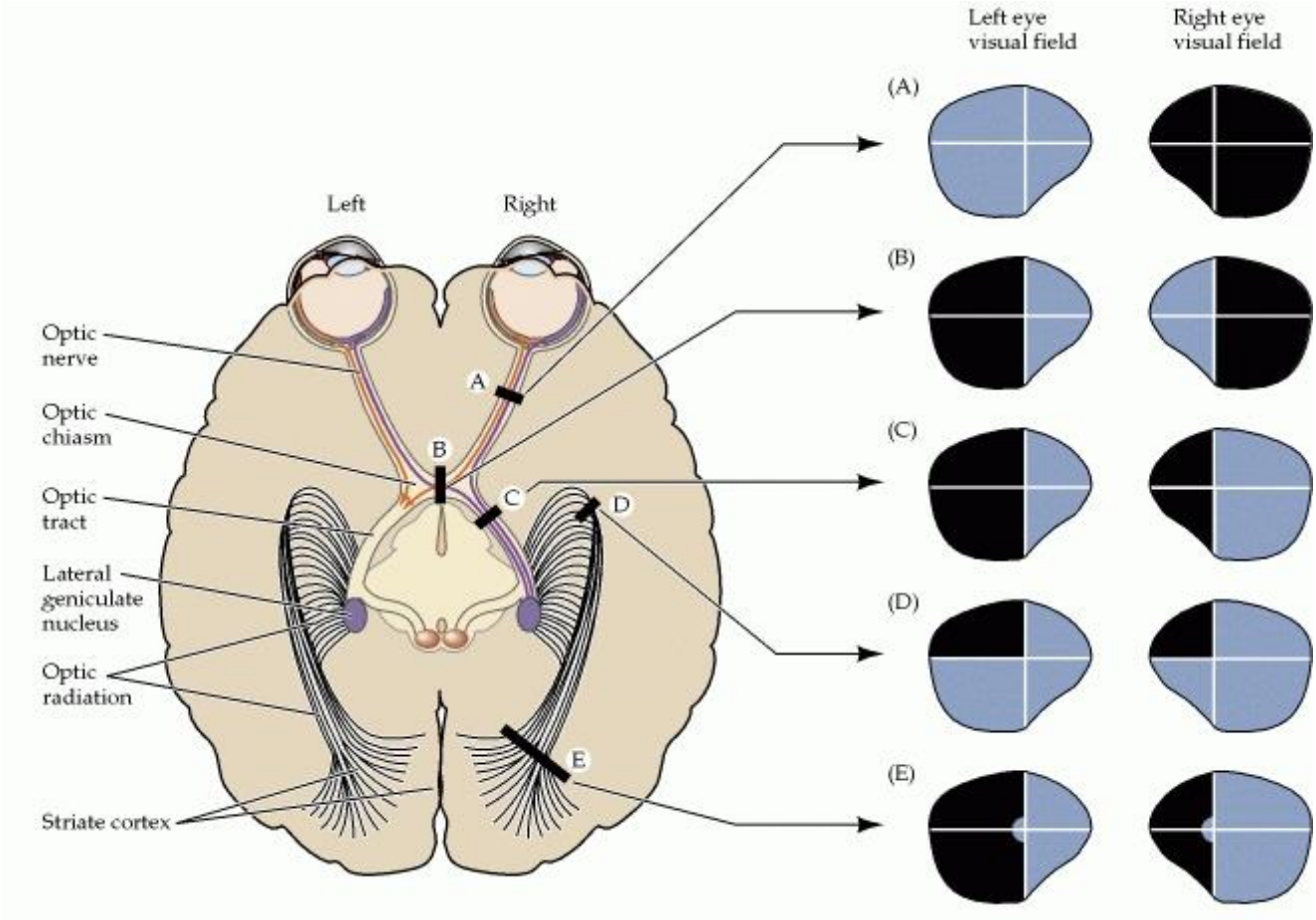
Cranial nerve 12

- Inspect the tongue in the resting position within the mouth. Look for wasting and fasiculations
- Ask the patient to protrude the tongue and look for any deviation
- Check the power of the tongue

Remember that in the exam usually the command is to examine only the motor component of the cranial nerves

Lesions of the visual pathway

Where is the lesion?



What is the pathology?

- The most important lesion of the above pathway is that at the optic chiasm. Abnormalities of the pituitary gland is responsible most of the time
- Given below are the causes
  Pituitary tumor – Look for features of acromegaly
  Craniopharyngioma
  Suprasellar meningioma
  Gliomas
  Vascular lesions – aneurysms

Ptosis

Where is the lesion?

Ocular myopathy (Muscle)	Myasthenia gravis (NMJ)	3rd nerve palsy (Nerve)	Horner's syndrome (Sympathetic)
Bilateral and symmetrical	Initially one side is more affected than the other Pupils are not involved Fatigability	Ptosis Affected eye deviated laterally and downwards Pupil may or may not be dilated	Partial ptosis Constricted pupil Enophthalmos Anhydrosis

3rd nerve palsy

Where is the lesion?

- The classification of 3rd nerve palsy is as follows
- Surgical 3rd nerve palsy – Here the pupillomotor fibers of the 3rd nerve are affected causing dilation of the pupil
  Seen in lesions of the midbrain and compression of the 3rd nerve along its course
- Medical 3rd nerve palsy – Here the pupil is unaffected

Surgical 3rd nerve palsy

- The lesion can be localized based on the pathway of the 3rd cranial nerve

Midbrain	Exit from the midbrain	Cavernous sinus	Orbit
Lies in close relationship with the corticospinal tracts and the red nucleus	Lies in close relationship with the posterior communicating artery	Lies in close relationship with the 4th and 6th cranial nerves and the ophthalmic and maxillary branches of the 5th CN	Lies in close relationship with the 4th and 6th cranial nerves and the ophthalmic branch of the 5th CN
Lesion 3rd nerve palsy + contralateral hemiplegia (Weber syndrome)	**Lesion** Isolated surgical 3rd nerve palsy	**Lesion** Associated 4th and 6th nerve palsies and sensory loss of the face in the ophthalmic and	**Lesion** Associated 4th and 6th nerve palsies and sensory loss of the face in the ophthalmic

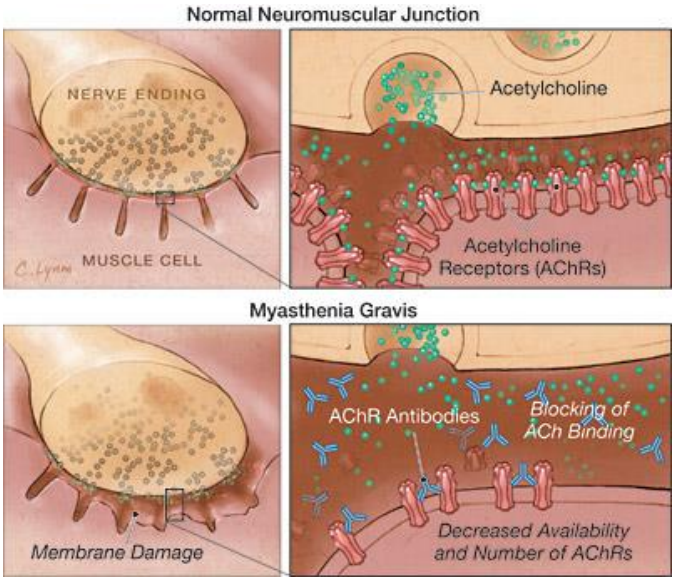
	maxillary divisions	division
3rd nerve palsy +red nucleus - tremor and involuntary movements (Benedikt's syndrome)		

What is the pathology?

	Site	Pathology
Surgical 3rd nerve	Midbrain	Vascular lesion – infarct Tumor
	Exit from the midbrain	Posterior communicating artery aneurysm
	Cavernous sinus	Cavernous sinus thrombosis
	Orbit	Tumor
Medical 3rd nerve	Nerve	Hypertension, DM, vasculitis, MS

Myasthenia gravis

What are the investigations you would like to perform in this patient?



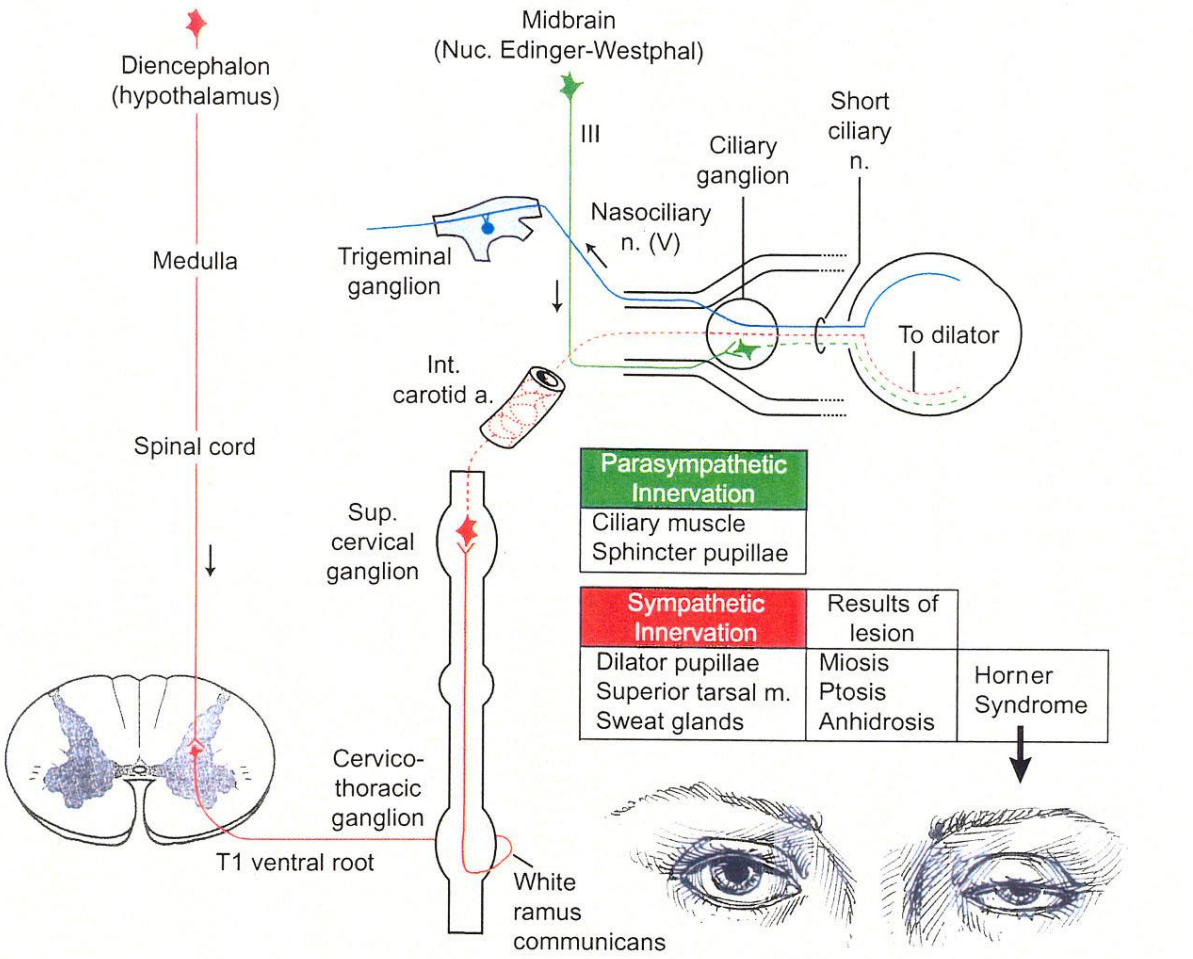
- **Edrophonium (Tensilon test)**
  This is an important test to aid in the diagnosis. Edrophonium is a short acting acetylcholinesterase inhibitor. This is injected IV and response is observed
- **EMG**
  Repetitive stimulation test will show a decremental response
- **Antibody testing**
  Acetylcholine receptor antibodies (over 80% of cases)
- **CXR/ CT thorax**
  To look for evidence of a thymoma

What are the options for management in a patient with myasthenia gravis?

- Anticholinesterase drugs
  Pyridostigmine
- Thymectomy
- Corticosteroids
- Other immunosuppressant therapy – azathioprine
- IV immunoglobulin or plasma exchange in a myasthenic crisis

Horner syndrome

Where is the lesion?



Given below is the localization of the important sites of the lesion in a patient with Horner syndrome

Brainstem	T1 root	Neck
Associated with lateral medullary syndrome	Look for small muscle wasting in the hands Examine the lungs to look for evidence of a tumor in the apex of the lung (Pancoast's syndrome)	Look for local lymphadenopathy, masses, aneurysms

6th nerve palsy

Where is the lesion?

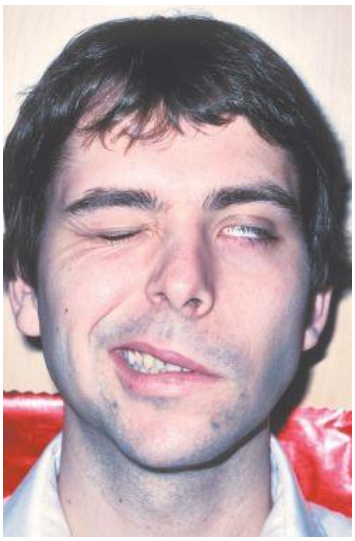
What is the pathology?

At the brain stem (Pons)	Intracranial course (nerve)	Cavernous sinus	Orbit
The 6th nerve nucleus lies in close proximity to the nucleus of the 7th nerve and the corticospinal tract	Has the longest intracranial course of all cranial nerves. Lies close to the tip of the petrous temporal bone	Lies in close relationship with the 3rd and 4th cranial nerves and the ophthalmic and maxillary branches of the 5th CN	Lies in close relationship with the 3rd and 4th cranial nerves and the ophthalmic branch of the 5th CN
Lesion 6th nerve palsy + LMN 7th nerve palsy + contralateral hemiplegia	**Lesion** Isolated 6th nerve palsy	**Lesion** Associated 3rd and 4th nerve palsies and sensory loss of the face in the ophthalmic and maxillary divisions	**Lesion** Associated 3rd and 4th nerve palsies and sensory loss of the face in the ophthalmic division
Pathology Infarction of the pons Tumor	**Pathology** Increased intracranial pressure Basal meningitis Inflammation of the petrous tip Diabetes Hypertension	**Pathology** Cavernous sinus thrombosis	**Pathology** Tumor of the orbit

7th nerve palsy

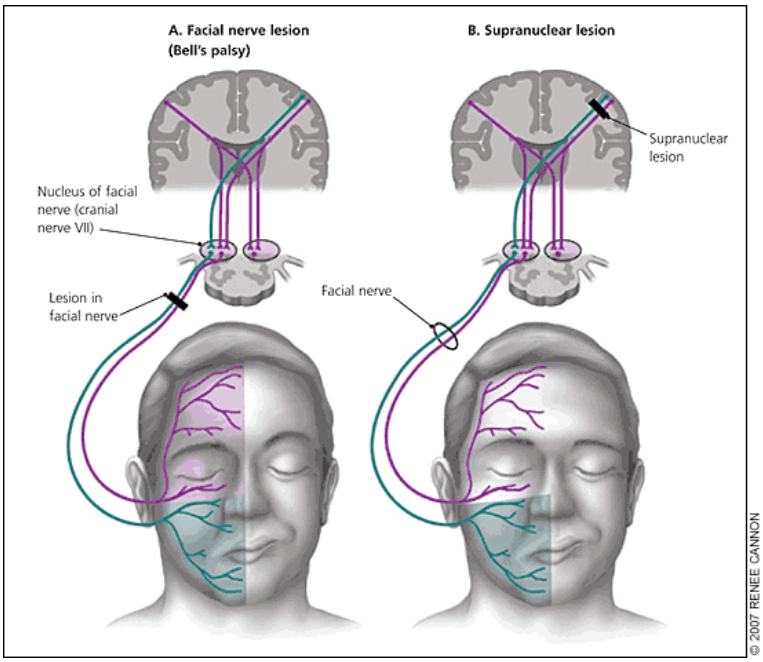
Where is the lesion?

What is the pathology?



- The most important step in localizing the site of lesion in a facial nerve palsy is to determine whether it is a UMN lesion or a LMN lesion
- Both upper and lower parts of the face would be affected in a LMN lesion while only the lower part of the face would be affected in an UMN lesion

- See the diagram given below



Clinical type	Site of lesion	Associated features	Pathology
UMN	Above the facial nucleus located in the pons Usually at the level of the cortex or internal capsule	**Cortical lesions** Associated dysfunction of speech (dysphasia) and loss of other higher functions Hemiplegia	Infarction Tumor
LMN	Pons	Associated ipsilateral 6th nerve palsy and contralateral hemiplegia	Infarction Tumor
	Cerebellopontine angle	Associated 5th nerve palsy and 8th nerve palsy	CP angle tumor – acoustic neuroma
	Internal acoustic meatus	Associated 8th nerve palsy	
	Inner ear	Check for hyperacousis and taste of the anterior 2/3 of the tongue, look for vesicles in the external auditory canal	CSOM, cholesteatoma, Ramsay Hunt syndrome
	External acoustic meatus	Isolated 7th nerve palsy	Bell's palsy
	Parotid gland		Parotid tumors

What are the treatment options available for Bell's palsy?

- Most patients recover spontaneously
- Physiotherapy
- Electrical stimulation of the facial nerve
- Steroids
- Acyclovir

Nystagmus and cerebellar signs

- There are clinical types of nystagmus. These are jerky nystagmus and pendular nystagmus
- **Pendular nystagmus**
  Oscillations are equal in speed and amplitude in both directions. Seen in patients with severe refractory error and macular disease
- **Jerky nystagmus**
  Has a fast phase and a slow phase. Seen in patients with cerebellar disease, vestibular disease and disorders of their central connections

What are the cerebellar signs you would elicit in this patient?

- Scanning dysarthria
- Past pointing
- Rebound phenomenon
- Dysdiadochokinesia
- Pendular knee jerk
- Heel shin test

What are the causes of cerebellar syndrome?

- Congenital anomalies – Agenesis of the cerebellar vermis, Dandy – Walker malformation
- Cerebellar infarction
- Demyelination - MS
- Cerebellar tumors - Medulloblastoma
- Infections
  During infections – coxsackie, echo, EBV
  Postinfectious – varicella
- Degenerative conditions – Friedrich's ataxia, ataxia telangectasia, Batten's disease
- Drugs and toxins – Phenytoin, alcohol
- Paraneoplastic syndromes – bronchial carcinoma

Lesions of the lower cranial nerves

- This involves lesions of the CN 9, 10 and 12

- Try to identify the clinical pattern of the lesion. These are given below

Bulbar palsy and pseudobulbar palsy

How would you differentiate between bulbar palsy and pseudobulbar palsy?

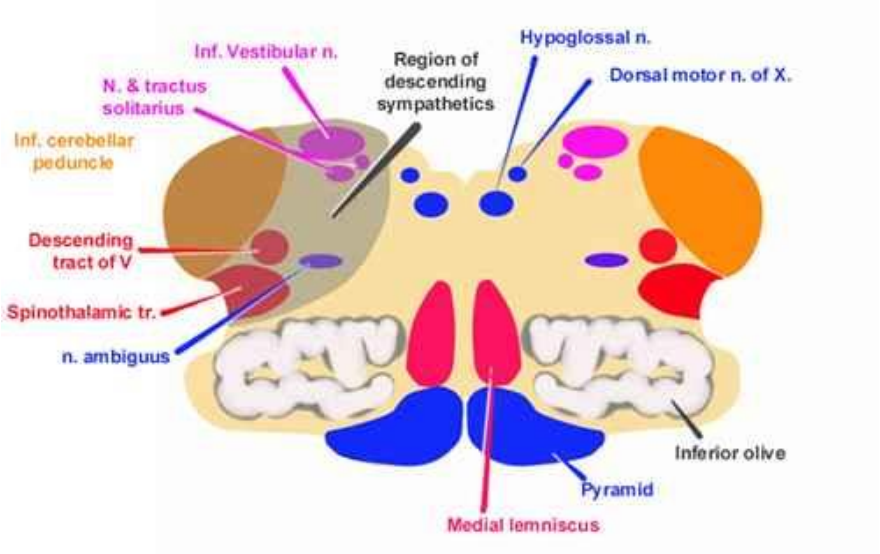
	Pseudobulbar palsy	Bulbar palsy
Anatomical basis	Is an upper motor neuron lesion of the cranial nerves 5, 9, 10, 11 and 12 (The cranial nerves arising from the medulla)	Is a lower motor neuron lesion of the cranial nerves 5, 9, 10, 11 and 12
Features	Emotionally labile Dysarthria ('Donald Duck' speech) Dysphagia Spastic tongue Palatal movements impaired Exaggerated jaw jerk	Not emotionally labile Dysarthria (nasal speech) Dysphagia + nasal regurgitation Tongue wasting and fasciculation Palatal movements impaired Normal or absent jaw jerk

What are the causes for bulbar palsy and pseudobulbar palsy?

Pseudobulbar palsy	Bulbar palsy
Stroke	TB meningitis
Demyelinating disease	MND
	Myasthenia gravis

Lateral medullary syndrome

- Is characterized by the following
- Ipsilateral Horner syndrome
- Ipsilateral 10th nerve palsy (palate)
- Ipsilateral cerebellar signs
- Ipsilateral sensory loss of the face
- Contralateral sensory loss of the body



12th nerve palsy

Where is the lesion?

What is the pathology?

- The most important aspect of a 12th nerve palsy is to identify whether the lesion occurs as a part of bulbar palsy or in isolation

Neurological examination of the lower limbs

Examination routine

- Introduce yourself and obtain consent
- Ask for a chaperone if necessary
- Ensure adequate exposure of the lower limbs

Start with an examination of the gait

Inspection

- Make sure to inspect the limbs carefully for muscle wasting and fasiculations. Tap the muscles of the thigh and leg to elicit fasiculations if they are not seen
- Look for scars – muscle biopsy scars and tendon release scars

Tone

- Ask the patient to relax
- Assess the tone of the lower limbs around all major joints – hip, knee and ankle
- Then put your hand behind the patient's knee and flick it upward – this assess the tone around all 3 joints at the same time
- If the tone is high check for ankle clonus and patellar clonus

Power

- Given below are the important muscle groups to be examined and their root values
- Remember to initially examine the movement without resistance and then with resistance

	Muscle action	Root value	Muscle and nerve
Hip	Flexion	**L1, L2**	Iliopsoas – Femoral
	Adduction	**L2, L3**	Adductors – Obturator
	Abduction	**L4, L5**	Gluteus medius – Superior gluteal
	Extension	**L5, S1**	Gluteus maximus – Inferior gluteal
Knee	Extension	**L3, L4**	Quadriceps – Femoral
	Flexion	**L5, S1**	Hamstrings - Sciatic
Ankle	Dorsiflexion	**L4, L5**	Tibialis anterior – common per.
	Plantarflexion	**L5, S1**	Gastrocnemius – tibial nerve
	Inversion	**L4, L5**	Tibialis posterior – tibial nerve
	Eversion	**L5, S1**	Peroneal – common per.

Grading of muscle power

Grade	Description
5	Normal power
4	Can move against resistance but sub optimal power
3	Can move against gravity but not against resistance
2	Cannot move against gravity but can move when the effect of gravity is eliminated
1	Flicker of movement
0	No movement

Reflexes

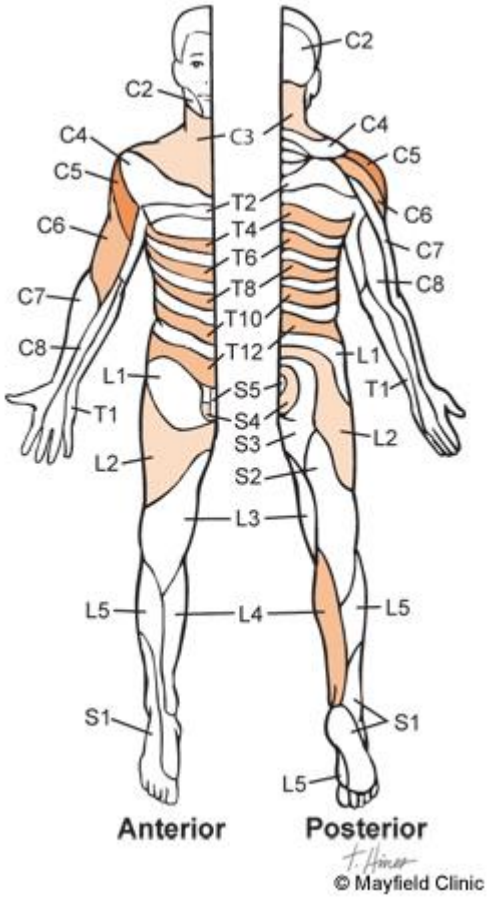
- Examine the following important reflexes. Make sure that the patient is relaxed
  Knee – L3, L4
  Ankle – L5, S1
  Plantar reflex
- If the reflexes are not elicited use reinforcement and re check

Coordination

- Perform the heel-shin test to look for coordination
- Remember that this test should only be performed if the muscle power is normal

Sensory

- Usually not examined at the exam



Interpretation of physical signs – LL examination

The two most important questions to be answered in a neurology case are

- Where is the lesion
- What is the pathology

The interpretation of physical signs starts with

UMN lesion or a LMN lesion?

Upper motor neuron lesion	Lower motor neuron lesion
Increased tone	Decreased tone
Increased reflexes	Diminished or absent reflexes
Ankle and patellar clonus may be present	
Extensor plantar response	Plantars may be flexor or equivocal

Approach to a LMN lesion

If the lesion is a lower motor neuron lesion further analyze your findings to localize the site of the lesion

Site of the lesion	Pattern of neurological signs
Muscle	Bilateral and symmetrical weakness Proximal>Distal weakness Reflexes – Knee jerk is lost while the ankle jerk may be preserved Waddling gait and Gower sign positive No sensory impairment
NMJ (Not given at the exam)	Fatigable weakness
Peripheral nerve	**Polyneuropathy** Bilateral and symmetrical weakness Distal>Proximal weakness Sensory may or may not be impaired. If impaired will be in a glove and stocking distribution **Mononeuropathy** Motor and sensory pattern related to the supply of the nerve **Multifocal neuropathy** (Rarely given as cases) Patchy involvement of peripheral nerves
Root	Will have motor and sensory loss in a root distribution
Anterior horn cell	Bilateral and symmetrical weakness Proximal>distal Prominent wasting and fasiculations No sensory impairment
Spinal cord lesions	Spinal cord lesions may present as LMN lesions Associated bladder and bowel incontinence Sensory level

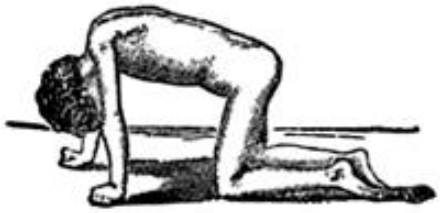
Possible further discussions from a case of LMN of the lower limbs

- Proximal myopathy
- Peripheral neuropathy
- Foot drop

Proximal myopathy

- There are two important categories of muscle disorders causing proximal myopathy. These are myopathies and muscular dystrophies
- The only important muscular dystrophy at undergraduate level is Becker's muscular dystrophy
- Myopathies can be classified according to their aetiology as given below

	Causes
Congenital	Metabolic myopathies Due to disorders in carbohydrate and lipid metabolism
Inflammatory	Dermatomyositis Polymyositis
Endocrine and metabolic	Hypo and hyperthyroidism Cushing's syndrome Conn's syndrome Hypokalemia
Toxic	Alcohol Organophosphates
Drugs	Corticosteroids Statins
Neoplastic	Paraneoplastic syndromes



Peripheral neuropathy

Polyneuropathy

- The following table lists the possible causes of polyneuropathy

Congenital	Acquired
Hereditary motor and sensory neuropathy (HMSN)	**Infection** Leprosy Diphtheria **Inflammatory** Guillain- Barre syndrome CIDP Vasculitis and connective tissue disease **Metabolic and endocrine** DM Vitamin deficiency – B1, B6, B12, E **Organ failure** Chronic renal failure **Drugs** **Toxins** Arsenic Lead Organophosphates **Malignancy**

- Investigation of a suspected neuropathy should be started with a nerve conduction study. Then specific investigations should be performed to find the possible cause
- The NCS identifies 2 major categories of peripheral neuropathy
  Demyelinating – Reduced nerve conduction velocity
  Axonal – Reduction in the amplitude of the action potential with relative preservation of the conduction velocity

Guillain – Barre syndrome

- Is a post infectious demyelinating disease
- Diagnosis is on clinical suspicion. Presents as an ascending paralysis which may follow a respiratory tract infection or an episode of diarrhea

Management

- The most important aspect of the management is close monitoring of the patient. The following are the most important
- **Progression of the neurological symptoms and signs**
- **Respiratory function**
  This is done with the single breath count and cough effort at the bedside. A more accurate assessment can be made by a respirometer
- **Autonomic function**
  A life threatening complication is autonomic dysregulation. Therefore monitor the pulse rate and blood pressure
- If there is deterioration in the respiratory function ICU care is necessary
- IV IG or plasmapharesis is used as the definitive management
- The child should be given limb and chest physiotherapy and DVT prophylaxis until recovery
- Proper nursing care is essential

Investigations

- Confirmatory investigations
- LP – shows cytoprotein dissociation with elevated proteins and normal white cell count
- Nerve conduction study

Foot drop

Where is the lesion?

There are 2 types of foot drop encountered in clinical practice. These are UMN type foot drop and LMN type foot drop. The latter is usually given as a short case

Cortex and spinal cord	L4/L5 root lesion	Sciatic nerve	Common peroneal	Polyneuropathy
UMN lesion	Weakness of inversion	Weakness of all muscles from the knee downwards (Knee extension spared – femoral)	Weakness of dorsiflexion and eversion. No weakness of inversion	B/L foot drop
	Ankle reflex preserved	Ankle reflex lost	Ankle reflex lost	Loss of ankle reflexes B/L
	Sensory loss in the L4, L5 dermatomes	Sensory loss in sciatic territory	Sensory loss over the lateral calf and dorsum of the foot	Stocking type sensory loss

Approach to an UMN lesion

After identification of an upper motor neuron lesion think of the clinical pattern

Clinical pattern	Possible sites of the lesion	Further localization
Hemiplegia	Cortex	Look for associated disturbances of higher function + UMN facial nerve palsy
	Internal capsule	Usually presents only with motor manifestations
	Brain stem	Examine for cranial nerve palsies
Spastic quadriplegia	Cervical cord	Look for associated bladder and bowel incontinence and a sensory level
	Brain stem	Examine for cranial nerve palsies
Spastic paraplegia	Spinal cord between T1 and L1	Look for associated bladder and bowel incontinence and a sensory level Examine the superficial abdominal reflexes for further localization

Remember –

- When you pick up UMN signs in the lower limbs always examine the upper limbs and do a quick cranial nerve examination to localize the lesion as given above

Possible discussions from and UMN lesion in the lower limbs

- Hemiplegia
- Spastic quadriplegia
- Spastic paraplegia

Hemiplegia

Stroke

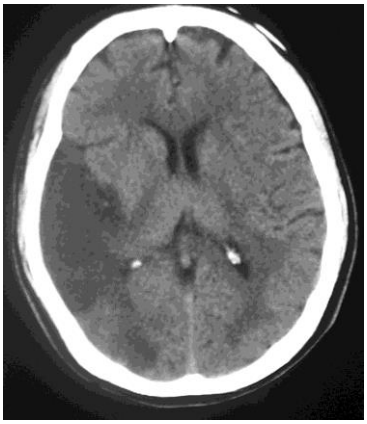
What are the causes you would like to consider in this patient?

- There are two major categories of stroke. These are ischaemic stroke and hemorrhagic stroke. Given below are the causes of ischaemic stroke

Pathology	Causes/ risk factors
Athero-thromboembolism	DM, smoking, Hyperlipidaemia, hypertension
Cardioembolism	Atrial fibrillation Infective endocarditis Intramural thrombus secondary to an MI
Arterial dissection	Carotid artery dissection
Vasculitis	SLE Infective vasculitis – HIV, syphillis
Hematological	Hemoglobinopathy – Sickle cell anaemia Hyperviscosity syndrome – polycythaemia, MM, macroglobulinaemia Hypercoagulable states – Protein c deficiency, protein s deficiency, factor V Leiden APLS
Other	Hyperhomocysteinaemia

- The aetiology depends on the age of the patient. Atherosclerosis would be the commonest cause in most patients but the other causes should be excluded in young patients

What are the aspects of management in a patient with a stroke?



- Stabilize the A,B,C of the patient
- Check and stabilize the blood glucose of the patient
- Imaging studies should be carried out – CT/ MRI
- Look for the possibility of administering thrombolytics (rtPA). Check inclusion and exclusion criteria
- Continue monitoring the vital signs of the patient
- Nursing care – bladder, bowel, skin
- Nutrition
- Rehabilitation
- Management of risk factors

What are the investigations you would like to perform in this patient?

- After the acute stage most of the investigations would be focused on finding an aetiology for the stroke
- Lipid profile, FBS

- Echocardiogram
- Vasculitic screen
- HIV testing, VDRL
- Clotting studies
- Serum homocysteine

Spastic quadriplegia and spastic paraplegia

Spinal cord disease

What are the causes you would like to consider in this case?

- Spinal cord disease can be classified as compressive and non compressive. The following table gives the causes

Compressive spinal cord disease

Site	Causes
Vertebral	Trauma Intervertebral disc prolapse Metastatic carcinoma Myeloma TB
Meninges	Tumors – meningioma, neurofibroma, lymphoma Epidural abscess
Spinal cord	Tumors – Glioma Metastasis

Non compressive spinal cord disease

Type	Causes	Important features
Congenital	Hereditary spastic paraplegia	AD Onset usually in adult life
Infective/ inflammatory	Transverse myelitis	
Vascular	Anterior spinal artery thrombosis	Dorsal columns (Proprioception and vibration) spared
Metabolic	Vitamin B12 deficiency	Loss of propriception, loss of ankle jerks due to associated peripheral neuropathy
Degenerative	MND Syringomyelia	Mixture of UMN, LMN, bulbar palsy Dissociated sensory loss

What are the investigations you would like to perform on this patient?

- X rays of the spine
- MRI of the spine
- Other investigations to look for a cause

Abnormalities of the gait

Gait	Description	Further examination	Possible causes
Hemiplegic	Circumduction with the upper limb of the affected side flexed at the elbow and pronated	LL examination for UMN signs, UMN facial nerve palsy	Stroke Tumor
Bilateral spastic	Looks as if the patient is wading through water	UMN signs in the lower limbs	Spinal cord disease
Cerebellar	Broad based and unsteady gait	Look for other cerebellar signs	Alcohol Cerebellar degeneration
Parkinsonian	Short shuffling steps Lack of arm swing Stooped posture Festinant gait	Other signs of Parkinson's disease – bradykinesia, rigidity, asymmetrical resting tremor	Parkinson's disease
Sensory ataxic gait	Stamping gait, broad based, patient looks to the floor to aid unsure steps	Positive Romberg's sign Impaired proprioception and vibration sense	Subacute combined degeneration of the cord Tabes dorsalis
High stepping	Foot drop	Examine LL	See discussion on foot drop
Waddling gait		Look for possible causes of proximal myopathy	Proximal myopathy

Neurological examination of the upper limbs

Examination routine

- Introduce yourself and take consent
- Ask for a chaperone if necessary
- Ensure adequate exposure of the upper limbs

Inspection

- Make sure you inspect the upper limbs very carefully for evidence of muscle wasting. Look especially over the deltoids, inspect the area over the scapula and look at the muscles of the palmar and dorsal surface of the hand
- Look for fasiculations in the major muscle groups of the upper limbs. Tap over the muscle to elicit fasiculations
- Look for surgical scars
- Ask the patient to hold out the hands and observe for any abnormal movements
- Look for wrist drop
- Look for pronator drift

Tone

- Ask the patient to relax
- Examine the tone around all important joints of the upper limbs – shoulder, elbow and wrists

Power

	Muscle action	Root value	Muscle and nerve
Shoulder	**Abduction**		
	30 degrees	**C5, C6**	Supraspinatus – Suprascapular
	Further	**C5. C6**	Deltoid - Axillary
	Adduction	**C6, C7**	Pec major, lat dorsi
	Internal rotation	**C5, C6**	Subscapularis – Subscapular
	External rotation	**C5, C6**	Infraspinatus - Suprascapular
Elbow	Flexion	**C5, C6**	Biceps – Musculocutaneous
	Extension	**C7, C8**	Triceps - Radial
Wrist	Flexion	**C7, C8**	Wrist flexors – Ulnar and median
	Extension	**C7, C8**	Wrist extensors - Radial
Fingers	Flexion	**C7, C8**	Wrist flexors – Ulnar and median

	Extension	**C7, C8**	Wrist extensors – Radial
	Abduction	**T1**	Dorsal interrossei – Ulnar
	Adduction	**T1**	Palmar interossei – Ulnar
Thumb	Flexion, extension, opposition	**T1** **T1**	Flexor pollicis, opponens pollicis - Median

Grading of muscle power

Grade	Description
5	Normal power
4	Can move against resistance but sub optimal power
3	Can move against gravity but not against resistance
2	Cannot move against gravity but can move when the effect of gravity is eliminated
1	Flicker of movement
0	No movement

Reflexes

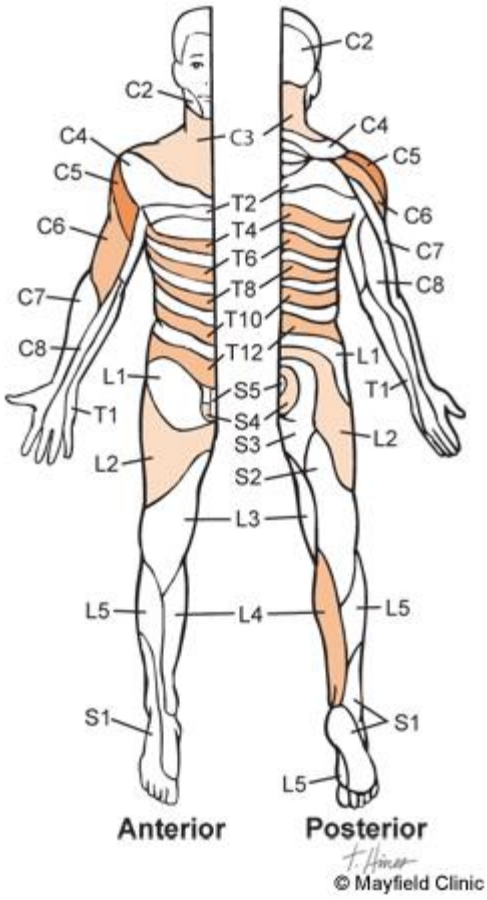
- Examine the following important reflexes. Make sure that the patient is relaxed
  Biceps – C5, C6
  Triceps – C7, C8
  Supinator – C6, C7
- If the reflexes are not elicited use reinforcement and re check

Coordination

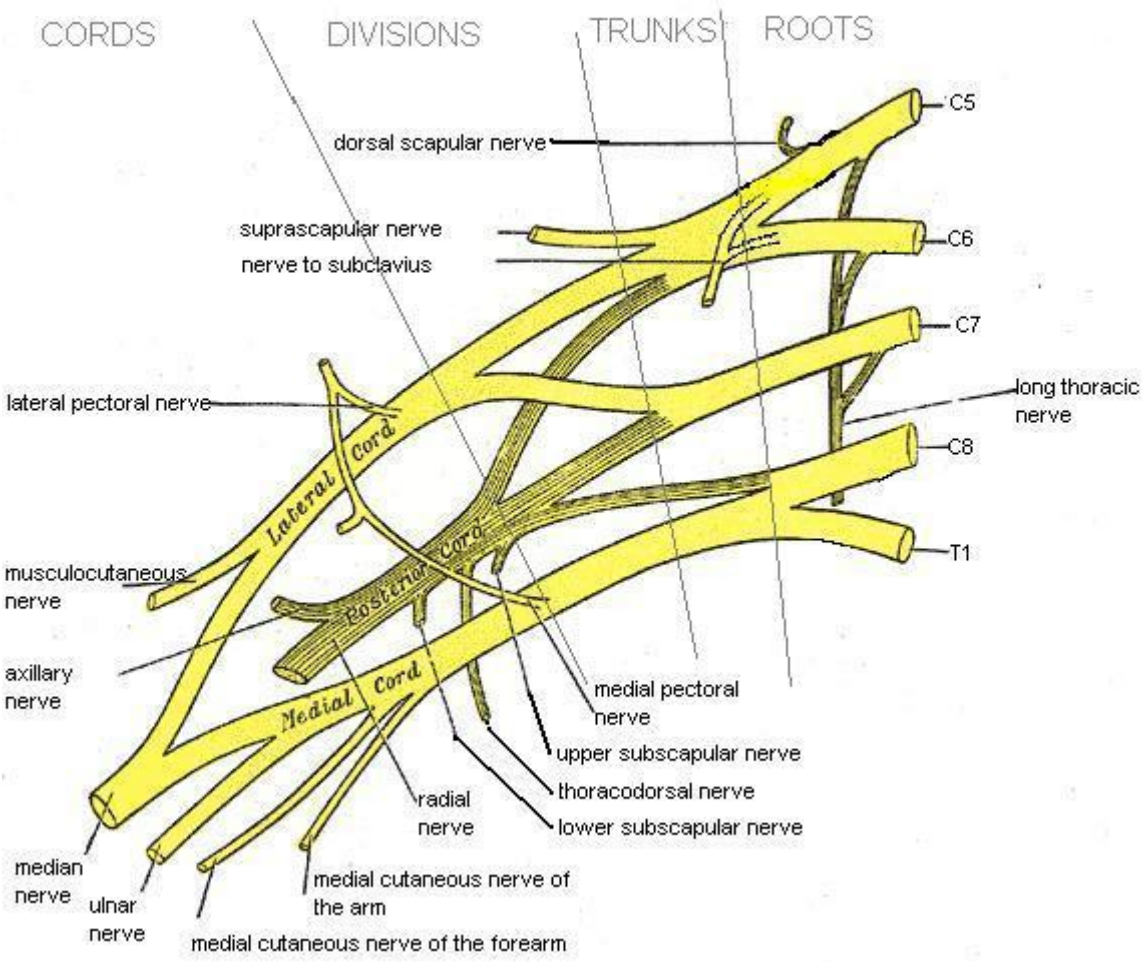
- Do the finger nose test and examine for dysdiadokokinesia
- In order to examine for coordination the muscle power should be normal

Sensory

- Examine the dermatomes



Anatomy of the nerves of the upper limb



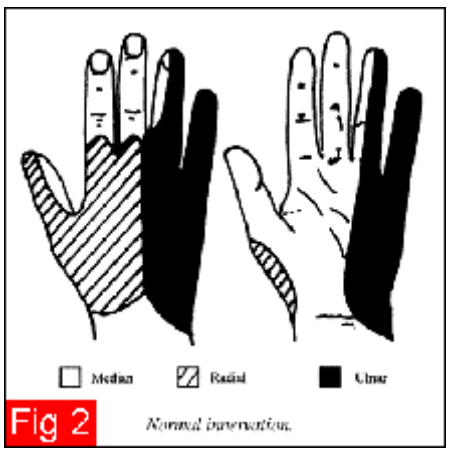
Wasting of the small muscles of the hand

Where is the lesion?

What is the pathology?

- The 1st step is to determine the pattern of muscle wasting and muscle weakness
- There could be 3 possible scenarios
  All muscles involved
  Ulnar nerve lesion
  Median nerve lesion

- 1st observe the pattern of wasting



- See if all muscles are weak – those supplied by the ulnar and median nerves. Two muscles are extremely important
  Abductor pollicis brevis - median
  Interossei – ulnar
  Another important confirmatory sign is to check for sensory loss
- If all muscles of the hand are involved follow the table given below

All muscles involved

Site of the lesion	Associated features	Causes
Muscle	B/L wasting and fasiculations (may be slightly asymmetric) No sensory impairment Associated bulbar/ pseudobulbar palsy and UMN signs in the LL	Motor neuron disease
Peripheral nerve	**Polyneuropathy** B/L Associated glove type sensory loss **Combined ulnar and median nerve** Sensory loss in the ulnar and median nerve distributions	Causes of polyneuropathy
T1 root lesion	Sensory loss in the T1 dermatome	Cervical spondylosis Syringomyelia Cervical rib Pancoast tumor

- If the ulnar nerve is involved further localization is necessary

Ulnar nerve lesion

Site of the lesion	Features	Causes
Above the cubital fossa	Flexor carpi ulnaris affected	Pressure palsy Trauma Fracture Mononeuropathy
At the wrist	More clawing (ulnar paradox) Flexor carpi ulnaris preserved	Compression in Guyon's canal

Median nerve lesion

Site of the lesion	Features	Causes

At or above the elbow	Weakness of the flexor digitorium superficialis and the lateral half of the flexor digitorium profundus Index and middle finger held in extension	Trauma Fracture
At the wrist (commonly in the carpal tunnel)	FDS and FDP spared Palmar cutaneous branch spared (sensory loss to the center of the palm. This branch goes above the flexor retinalculum) Positive tinel's test and phallen's sign	**CTS** DM, hypothyroidism

Claw hand

- Look at the picture given. The following will give a discussion of how to approach such a case

Where is the lesion?

Site of the lesion	Associated features
Muscle (MND)	Wasting and fasiculations of all small muscles of the hand Weakness of all muscles B/L involvement No sensory impairment
Peripheral nerve	**Polyneuropathy** Wasting and fasiculations of all small muscles of the hand Weakness of all muscles B/L involvement Sensory loss in a glove pattern **Ulnar nerve palsy** Weakness of muscles supplied by the ulnar nerve Interossei Note that the abductor pollicis brevis will be spared (median) Froment' s sign +
Root (T1)	Sensory loss in the T1 dermatome

Wrist drop

Where is the lesion?

Site of the lesion	Features
Muscle	**Unlikely**
Peripheral nerve	**C7 root lesion** **Polyneuropathy** B/L **Radial nerve palsy**

Further localization in radial nerve palsy

Site of the lesion	Features	Causes
Lesion above the junction of the upper and middle thirds of the humerus	Triceps affected Brachioradialis affected	Trauma Fracture
Lesion at the middle 3rd of the humerus	Triceps affected Brachioradialis spared	Trauma Fracture
Lesion at the wrist	Finger drop only Triceps and brachioradialis spared	Trauma Fracture