Medical students Guide to Paediatrics

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Contents

Personal profile	Page 3
Normal values	Page 5
Bronchiolitis	Page 10
Cerebral Palsy	Page 12
Developmental Assessment	Page 16
Congenital Dislocated Hip	Page 18
Epilepsy	Page 19
Febrile Convulsions	Page 22
Jaundice in the Neonate	Page 24
The Child with Painful Joints	Page 28
Asthma	Page 32
Guide to clinical Examination	Page 37

Personal Profile

Topic	Clinical	Theory	Revision
Normal Values			
Urine Dipstix			
X-ray Interpretation			
History Taking x12			
Abdominal Examination x5			
Respiratory Examination x5			
Cardiovascular Examination x5			
Developmental Examination x5			
Neurology Examination x2			
Gait Examination x5			
ENT Examination x5			
Asthma			
Bronchiolitis			
Croup			
Pneumonia			
Respiratory Distress syndrome			
Bronchopulmonary Dysplasia			
The Preterm Neonate			
Febrile convulsions			
Epilepsy			
Cerebral palsy			
Infantile spasms			
Renal Failure			
Leukaemia			
Resuscitation			

Paediatrics at Darlington

Introduction

Welcome to the undergraduate course in paediatrics at Darlington.

My role as your tutor has two main components: firstly, I am there to ensure that you leave DMH with a good basic knowledge in paediatrics. From your point of view this probably means that you pass the exam! Secondly, I am available to help with any problems you may have which may not be directly to do with the course.

It is up to you to let me know of any circumstances which you feel may interfere with your progress in the course and I will try my best to help you with them.

The emphasis of the course is in developing your paediatric clinical skills. Attendance is given a lot of weight in your final assessment.

How to get hold of me

My Secretary, Vicki Greener holds my diary and will be able to connect you with me: vicki.greener1@nhs.net

If it is urgent you can text me on: 07967822808 and my personal email is tim.ubhi@e-hospital.co.uk

Dr Tim Ubhi
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Paediatric Normal Values

During your time as a medical student you will increasingly be expected to look at laboratory results and interpret them. In order to do this, you need to learn common "reference ranges". Once you qualify, these ranges will be a part of everyday life, so I think it is a good idea to become familiar with them at an early stage. Do not consider them as a burden which you need to learn to pass the exam, rather use them as tools to help you towards making a diagnosis in a patient.

In order to help you I have listed the normal values you should learn followed by a few tasks which I would like you to complete.

Biochemistry	Value
Sodium	133-145 mmol/l
Potassium	3.3-5.5mmol/l
Urea	2.5-6.5mmol/l
Creatinine	20-80 μmol/l
Bicarbonate	18-25mmol/l
Chloride	96-110mmol/l
Albumin	35-55g/l

Haematology

Haemoglobin	11.5-15.5* g/dl
White cell count	4.5-14.5 $x10^9$ /l
Platelets	150-450 x 10^9 /l
Mean cell volume	77-95 μm^3
Mean cell Haemoglobin conc.	31-37 pg/cell

Arterial bood gas

Ph	7.35-7.45
PaO_2	11-14 kPa
$PaCO_2$	4.5-6.0 kPa
HCO_3	18-25 mmol/l
Base Excess	-3 to +3 mmol/l

Infection

C-Reactive Protein (CRP)	< 10 mg/l

Exercise 1

If a patient is involved in any incident which results in a loss in intravascular volume such that they are shocked, what is likely to happen to their capillary refill time?

……

How do you test the capillary refill time?

……

Why may the blood pressure be normal?

……..

What are the consequences to cell respiration?

……

Predict the arterial blood gas in such a patient

……..

Exercise 2

A patient is referred to you with a history of lethargy. The Referring GP provides the following full blood count;

Hb = 7.7g/dl
Wcc = 9.0
Platelets=200

MCV =52
MCHC = 26

What abnormalities are present?

……

How would you describe this blood picture?

……

What treatment would you start?

……

How does this differ from beta thalassaemia?

……

Exercise 3

Describe the biochemical abnormalities seen in pyloric stenosis

……

What is the initial management?

……

What are the daily fluid requirements of a 1-year old child?

……

What are the daily fluid requirements for a 5-year-old child?

……

What is the radiological investigation of choice in pyloric stenosis?

……

What is the surgical procedure used to treat pyloric stenosis?

……

Exercise 4:

You are asked to see a 5 year old patient in casualty referred because of recurrent episodes of epistaxis.

A full blood count is taken which shows the following results

Hb = 135
Wcc = 7.5
Platelets= 8

Assuming that the blood film is normal, what is the likely diagnosis?

……

What important differentials must you bear in mind?

……

what is the treatment?

……

What is the prognosis?

……

Exercise 5

Plotting growth charts

Bronchiolitis

Definition:

Coryza for one to two days followed by persistent cough, breathlessness, hyperinflation, and expiratory wheeze in a 1 to 12 month old. Fine crackles and cyanosis correlate with severe disease. **Respiratory syncitial virus** (RSV) is the usual cause (occasionally ***parainfluenza***).

Epidemiology:

1. 1-2 % of all infants are admitted to hospital with bronchiolitis.
2. Occurs in winter to early spring.
3. Breast feeding and parental avoidance of smoking is protective.

Pathology:

1. Inflammation of the bronchioles: secretion of mucus, necrosis of ciliated epithelium and oedema of the submucosa leading to airways obstruction.
2. Hyperinflation and patchy segmental collapse of the lung.

Clinically:

1. Tachypnoea, cyanosis and inability to feed.
2. Head bobbing and unresponsiveness.
3. 1% progress to respiratory failure.
4. Apnoea, especially in the premature.
5. Cardiac failure is unusual.

Investigations:

1. RSV detected by immunofluorescence of nasopharyngeal secretions.
2. CXR shows square chest, horizontal ribs, hilar streaking, with subsegmental collapse or
consolidation in 35%.
3. Blood gases show hypoxia

Differential:

1.Asthma
2. Bronchopneumonia
3. Cystic fibrosis
4. Heart failure

Management:

1. Admit to hospital if respiratory or feeding difficulties.
2. Feed via iv or NG if indicated.
3. Oxygen for pallor or cyanosis. Pulse oximetry.
4. Bronchodilators
6. Mechanical ventilation.

Prognosis:

Recovery in 7-10 days.
Recurrence of wheeze in 50-75%

CEREBRAL PALSY

Definition:

A non-**progressive** disorder of the developing brain affecting **movement** and **posture**. It is a group of conditions with various causes and neurological dysfunctions.

Aetiology and incidence

1.Antenatal (60%) Intra-uterine infection, placental dysfunction, drugs.
2.Birth asphyxia (10%)
3. Low birthweight (10%). Children < 1.5kg have a ten-fold risk of CP.
4. Post neonatal (10%). Infection, trauma & hypoxia.
5. Familial & Genetic (10%). eg due to Joubert's syndrome
(AR abnormal eye movements, hyperventilation, mental handicap, ataxic cerebral palsy later.)

Overall incidence is 1/500 children. 30% mild, 70% moderate and severe

Nomenclature

Diplegia	(both legs > arms) - PVL, HII.
Hemiplegia	(half the body, arm > leg) - congenital, HII, brain malformation, child abuse, meningitis, prolonged seizure.
Quadriplegia	(All 4 limbs arms > legs) - mainly congenital
Paraplegia	(lower limbs only) - this is usually a diplegia.
Monoplegia	(one limb) - usually a hemiplegia

Motor pattern.

1. Spastic. 70% = increase in tone.
2. Dyskinesia. 15% = constant change in tone
   (i) dystonia = writhing movements with prolonged abnormal postures.
   (ii) athetosis = continuous slow writhing movements.
   (iii) chorea = sudden jerky movements
3. Ataxia (5%). = incoordinate movement, often with hypotonia.
4. Ataxic diplegia (10%) = mixed CP

Clinical presentation

1. Floppy infant
2. Delayed motor development
3. Strong hand preference beginning under 1 year.
4. Failure to thrive
5. Bottom shuffling (diplegia)
6. Persistent drooling with speech delay.
7. Preservation of primitive reflexes: moro, tonic neck reflex,
8. Affected side underdeveloped in hemiplegia.
9. Mental handicap, epilepsy, squint and hearing defects are commoner.
10. Immobility causes windswept posture, kyphoscoliosis, dislocation of hips .
11. Contractures.

Management.

Aim: maximum independence and self dignity

A. Therapy

1.Physiotherapy. Advice on handling, bathing, sitting etc. Prevent secondary deformity.

2. Speech therapy. Help in drooling, sign language (e.g. Makaton), communication and voice synthesizers.

3. Occupational therapy. Home aids, hand skills, Portage scheme.

B. Medication

1. For epilepsy

2. To reduce spasticity- Baclofen, diazepam, L - dopa in dystonic CP.

3. Counselling for parents. Support groups.

4. Aim to introduce those of normal intelligence into mainstream school.

5. Orthopaedic assessment. Shoes and splints. Brace or surgery to correct kyphus or scoliosis. Surgery to lengthen heel cords, cut adductors.

6. Botulinum toxin

Prognosis

Normal intelligence in 1/3.

Seizures in 50%

Exercise

1. What is cerebral palsy?

2. What is the aetiology?

3. How common is it?

4. What is the most common type and why?

5. What is the prognosis for intelligence in a child diagnosed with Cerebral palsy.

6. Imagine you are the doctor seeing a patient just diagnosed with spastic diplegia. List the treatment modalities you would discuss with the parents.

Developmental Assessment

AGE	GROSS MOTOR	FINE MOTOR	LANGUAGE SKILLS	SOCIAL & PERSONAL
1M	Lifts head while prone	Visual following to midline		Watches face
2M	Lifts head erect open when sitting	Hands predom. Stills to Mums voice. Startles.	Vocalizes & coo's.	Smiles responsively
3M	Lifts head tp 90' while prone Loss of Morrow & Grasp.	Visual following past midline	Laughs	
4M	Head steady when held	Plays with hands together	Goos & gurgles	Excited by approach of food
5M	No head lag Rolls over	Palmar grasp Holds rattle	Squeals	Smiles spontaneously
6M	Lifts head forward when pulled to sit Weight on hands when prone.	Passes block hand to hand	Turns to voice Responds to own name.	Friendly to all comers Chews on biscuit.
7M		**Transfers** from hand to mouth	"Ma" & " Da"	
8M	Sits without support *Saving Reflexes	Object Permanence	Repetition of syllables	Feeds self biscuit
9M	Crawls Rolls supine-> prone.		"Mama" Understands " no"	
10M	Stands holding on	Crude pincer grip.(Index approach.)	"Mum" & "Dad" without meaning	Shows object to mother.
12M	Walks holding onto furniture	Good pincer grip. Reaches behind. Throws objects	Imitates sounds Shakes head Bangs 2 bricks	Gives up toy Waves Bye-bye. Arm out for sleeve.

15M	Walks alone	Neat pincer grasp	"Mum" & "Dad" with meaning	Indicates wants
1.5Y	Walks well Climbs stairs	tower of 2-4 blocks	6 words apart from mum/dad	Drinks from cup Symbolic play & Feeds with spoon Pulls at nappy.
2Y	Walks down steps & runs Kicks ball	Scribbles Tower of 6-8 One page / time	Points to 3 part of body	Gives dolly a drink.
3Y	Pedals tricycle	Imitates vertical line. Copies circle/ +	Uses 4 word sentences Count to 10	Puts on clothes Dry by day Pleurals/ preps.
4Y	Balances on one foot for 5 sec.		Gives first & last name	Dresses with supervision.
5Y	Hops on one foot Catches ball.	Draws man in three parts Copies x,sq. & triangle	Knows age & some colours Counts to 20 Knows 3 coins. Nursery rhymes Knife & Fork	Dresses without supervision Shoelaces Wipes bottom

* The " Parachute Reflex " occurs before the child begins to walk and involves extension of the upper extremities when the child is suspended by the trunk and sudden forward flexion is produced, as if the child were to fall.

Other primitive reflexes include the " rooting ", " Moro ", " Grasp " and " tonic neck reflex".

Developmental Dysplasia of the Hip

Developmental dysplasia of the hip
(Aka " Congenital dislocated hip") is
Classified into 2 major groups:

1. Typical - in a neurologically normal infant

2. Teratologic- when there is underlying neuromuscular disorder

Aetiology:

-Multifactorial
- 9:1 female preponderance
- 60% are first born
- 30 -50% are breech
- Associated with congenital muscular torticollis & metatarsus adductus

Clinical tests:

1. Barlow's test- Determines whether hip is *"DISLOCATABLE".*
   The pelvis is fixed with one hand and the opposite hip is flexed and abducted applying a posterior force.

2. Ortolani test- Determines whether the hip is *" DISLOCATED"* and involves a manoeuvre to reduce the dislocation. The thigh is flexed and abducted and the femoral head is lifted anteriorly into the acetabulum.

Hip Clicks-

These are not usually pathological and may be due to :
1. Breaking of surface tension across the hip joint
2. Snapping of gluteal tendons
3. Patellofemoral motion
4. Femorotibial rotation

Investigation:

The femoral head ossific nucleus does not appear until 3-7 months of age. Therefore, ultrasound scan in the newborn is a good first line of investigation. Older children may require A-P and frog lateral x-rays.

Treatment:

In the newborn, need to maintain the hip flexed and abducted. Therefore may use the Pavlik or Frejka splint. Alternatively, the use of double or triple nappies may be indicated.

Complications: Avascular necrosis in 5-15%

EPILEPSY

Definition:

" More than 1 episode of altered consciousness sensation or movement, primarily cerebral in origin, un-associated with infection, metabolic state or fever."
(Ross et al., 1980).

In a very simplistic way one could consider epilepsy as a *tendency towards seizures*.

Causes of seizures

AEIOU - C-DPT

A= Apoplexy from hypertension or intracranial bleed.

E= Epilepsy

I= Infection

O= Oxygen lack

U= Uraemia and other metabolic disturbances [hypoglycaemia, hyponatraemia, Reye's syndrome, hypernatraemia, hypocalcaemia, hypomagnesaemia, pyridoxine deficiency. Inborn errors such as PKU, hyperammonaemias.

C= Congenital brain malformation. Hydrocephalus, cyst.

D= Drugs and withdrawal.

P= Pseudoseizure. Hysterical and Munchausen by proxy.

T= Trauma, Tumour and Toxins.

Classification of childhood epilepsy.

1.Partial

Focal onset in a part of one cerebral hemisphere.

(i) Simple - No loss of consciousness.

(ii) Complex - With loss of consciousness (includes temporal lobe)

(iii)Secondary generalization . Start as simple or complex and progress to generalized tonic/clonic seizure.

2. Generalised. (From onset)

(i) Absence seizures: petit mal, atypical absence seizures

(ii) Myoclonic seizures: infantile spasms.

(iii)Tonic + Tonic clonic seizures: Grandmal and reflex epilepsy

3. Unclassifiable

Neonatal seizures.

4. Reactive febrile convulsions.

Management of Epilepsy.

Many parents believe their child is dying during the first observed fit.

1. Medication

Start after 2nd or 3rd seizure if time between seizures is < 12 months.

administer once or twice daily.

Monotherapy is effective in 80%. 10 - 15% need 2, 5% need 3.

If drug therapy fails, consider surgery.

Note on drugs:

1. Valproate is 2nd line in under two's - hepatotoxicity.
2. Carbamazepine preferred to phenobarbitone and phenytoin with less cognitive dysfunction.
3. Vi***gaba***trin, a GABA transaminase inhibitor, may be useful in children > 3yrs old. Complex partial, infantile spasms and tuberous sclerosis.
4. Lamotrigine, a folate antagonist, may help atypical absence attacks.
5. Steroids & ACTH help in infantile spasms.
6. Hypoallergic diet may help refractory epilepsy.

2. Parental advice

Reassure

Avoid precipitants

Avoid overprotectiveness

Give antipyretics for fever.

3. Protection

No cycling

Supervised swimming until seizure free for 6 months.

Avoid contact sports

If photosensitive, sit at 45 degrees and 3m away from TV with back light.

Computer screens are safe.

4. Psychological support

5.Surgery

If unresponsive to treatment for > 2 years.

In partial epilepsy with defined focus.

Causing significant handicap.

Driving and Epilepsy.

Sufferer must be seizure free for1 year, or if nocturnal only, for 3 years.

If adolescent, may prefer to stay on medication and learn to drive.

Epilepsy after 5 yrs old excludes holding a public service vehicle or HGV licence.

Febrile Convulsions

Definition:

A convulsion occurring when febrile and no other cause is found in a child between 6 months and 6 years and where there is no past history of afebrile convulsions.

Incidence:

3% of all children are affected. 30% will have more than one fit.

Cause :

Usually associated with viral URTI but always consider meningitis and UTI

Investigation:

CXR, urine and lumbar puncture in all children in whom no source of infection is found and in all children less than 18 months even with URTI.

Treatment

1. Oxygen by mask
2. 5mg diazepam pr
3. If still fitting - iv Lorazepam 0.1mg/kg.
4. Treat source of pyrexia.

Prevention

1. 10mg/kg paracetamol every 4 hours or 5mg/kg ibuprofen every 6 hours when pyrexial.

2. Undress, tepid sponge and fan in the room but not on the child.

Follow up:

1. No follow up or EEG unless frequent febrile convulsions.
2. Consider valproate or phenobarbitone if > 5 febrile convulsions. Continue for 2 years. Risk of epilepsy is unaltered.
3. Risk factors for recurrence: - Onset < 15 months
   - Prolonged fit (> 30 mins)
   - Focal
   - Developmental or neurological abnormality
   - sib or parent with epilepsy

Advise Parents:

1. If pyrexial institute antipyrexial measures
2. Supply rectal diazepam and give basic CPR training.
3. Take to hospital if fit > 15 minutes.
4. No immunisation is absolutely contraindicated
   - give regular paracetamol for 10 days following MMR.

A rough guide to counselling:

A. *Nature of Febrile convulsion*;

1. Due to high temperature
2. Especially rapidly rising temperature
3. Possible recurrence (30%)
4. Occurs between 6 months and 6 years.
5. Usually of short duration (< 10 mins)
6. Good prognosis
7. No long-term anticonvulsant treatment
8. Very common

B *Home management*;

1. Treatment of fever - Skin exposure / light clothing
- encourage oral fluids
- Electric fan / cool room
- Regular paracetamol

2. Treatment of convulsion
Lie on side in safe place
Do not force object between teeth / gums

if convulsion not stopped by 5 mins call 999 or administer rectal valium.

C. Ability to communicate and empathise:
- Explanation understandable to a lay person.

Jaundice in the Neonate

Physiological jaundice is very common in the neonatal period. This type of jaundice peaks at approximately 2-5 days of life, disappears within 2 weeks, is worse in breast fed infants (due to poor steroid handling) and is worse in the premature baby. Walking around the neonatal unit you will often see babies under ultraviolet lamps undergoing phototherapy. This is the mainstay of treatment in straightforward physiological and is important to avoid kernicterus (bilirubin encephalopathy).

Clinical features suggesting liver disease:

1. Pale stools & dark urine
2. Dysmorphic features
3. Bruising, petechiae or bleeding
4. Hepatomegaly
5. Failure to thrive

Investigations:

1. Urinary bilirubin - present only in liver disease
2. Split plasma bilirubin - if more than 25% is conjugated, liver disease should be investigated.
3. Alanine aminotransferase (ALT)< 40 IU
4. Alkaline phosphatase 600 - 1000 IU / litre
5. Prothrombin time (PT)

Causes of prolonged unconjugated Hyperbilirubinaemia

1. Physiological
2. Haemolysis - a) Immune
   b) RBC membrane abnormality
3. Metabolic disorders
   a) Crigler-Najjar syndrome
   b) Gilberts syndrome
   c) Galactosaemia
   d) Fructosaemia
   e) Hypothyroidism
4. Sepsis
5. Hypoxia

Differential diagnosis of prolonged neonatal Jaundice (> 2 weeks)

1. Biliary abnormality
2. Neonatal hepatitis
3. Metabolic disorders
3. Iatrogenic (TPN & drugs)

Biliary Atresia

Biliary Atresia occurs in 1/14000 births worldwide. Aetiology is unknown and there is no genetic basis. Destruction of the extrahepatic and intrahepatic biliary ducts leads to cholestasis, fibrosis and cirrhosis. 25% have associated abnormalities such as ASD, VSD, situs inversus and polysplenia. Diagnosis MUST be made before 70 days to prevent irreversible damage.

Clinically: Jaundice and pale stools from day 2. Failure to thrive. Hepatomegaly is an early feature and splenomegaly implies fibrosis.

Investigations: Conjugated bilirubin is > 100 µmol/l, ALT 100- 200 IU/l. PT and albumin are normal in the early stages.

Diagnosis:
-Absent or contracted gallbladder on abdominal uss after 4 hour fast.
-Demonstration of failure of excretion of radioisotope (DISIDA) from liver into bowel 24 hours after administration. (Pre-treatment with phenobarbitone for 5 days, at 5mg/kg, improves the diagnostic accuracy)
- Liver histology
- Operative cholangiogram

Management:
Surgical removal of the fibrosed biliary tree and formation of a Roux-en-Y anastomosis (Kasai portoenterostomy) which achieves biliary drainage in 80 % if performed before 7 weeks.
Sufficient calories to prevent malnutrition and to overcome fat malabsorption can be provided by supplying a high calorie protein feed with 50% medium chain triglycerides. In addition, fat soluble vitamins are needed in the following amounts:

Vitamin A : 5-15,000 IU/day
Vitamin D : 50ng/kg/day (alphacalcidol)
Vitamin E : 50 - 200 mg /day
Vitamin K : 2.5 - 5 mg / day

Prognosis:
Recurrent cholangitis, cirrhosis and portal hypertension are inevitable.

Choledochal Cysts

These are cystic dilatations of all or part of the common bile duct.
Commoner in Japan
Four times commoner in girls.
Leads to biliary fibrosis, cirrhosis and liver failure.

Clinically:
Obstructive jaundice
hepatomegaly
diagnosis is based on USS, PTC or ERCP.

Management:
Surgical excision with formation of hepatojejunostomy

Prognosis:
2.5% risk of malignancy in later life.

Gallstones

These may occur secondarily to haemolysis in the neonate.

Biliary hypoplasia (*Alagille's syndrome*)

Autosomal dominant
1/100,000 live births.
Present with persistent cholestasis, dysmorphic features, butterfly vertebrae, abnormal digits, peripheral pulmonary stenosis, renal tubular acidosis, posterior embryotoxin and severe failure to thrive . Mental retardation is seen in 30%.
Diagnosis is by liver biopsy.

Management:
Nutritional support, pruritus is intractable and managed cholestyramine 1-2g/day, phenobarbitone 5-15mg/kg/day and rifampicin 50mg/kg/day.

Prognosis:
50% of children will regain normal liver function by adolescence. Liver transplantation may be indicated if there is progression to cirrhosis and portal hypertension or intractable pruritus.

Neonatal hepatitis

Significant histological overlap with biliary atresia occurs.
Babies tend to be small for gestational age, stools may be pigmented and dysmorphic features may be obvious. Biochemically, ALT 200-300 IU/l and conjugated bilirubin is greater than 100mcmol/l.
Liver histology demonstrates a giant cell hepatitis with fibrosis,extramedullary haemopoiesis, cholestasis and biliary ductule proliferation.

Causes:
1. CMV (IgM antibody) - 60% completely resolve. Of the remainder, 10% require transplantation.
2. Rubella
3. Toxoplasmosis
4. Herpes
5. Syphillis (treponema pallidum haemagglutination test)

Metabolic causes

Alpha 1 anti-trypsin deficiency-autosomal recessive, 1/4000 births, < 0.9g/l
30% recover, 30% develop inactive fibrosis or cirrhosis, 40% CLF

Tyrosinaemia type 1

- autosomal recessive disorder presenting with acute liver failure in the neonate or chronic liver disease in the older child. Due to deficiency of enzyme Fumaryl- aceto-acetase. Results in cirrhosis, hepatocellular carcinoma, hypertrophic cardiomyopathy, renal tubular acidosis, vitamin D resistant rickets' and peripheral neuropathy. Diagnosis is by isolating urinary succinyl acetone.

Galactosaemia.

A rare autosomal recessive disorder.
1/40,000 live births
presents with hypoglycaemia, jaundice, hepatomegaly and cataracts.
Diagnosis is confirmed by measurement of galactose 6 phosphate uridyl transferase which is deficient leading to accumulation of galactose 1 phosphate in tissues.
management is with a galactose free diet.

Cystic fibrosis

May present with jaundice. Sweat test at 6 weeks is needed.

Niemann pick disease type C

A neurovisceral lipid storage disorder which presents with foetal ascites or neonatal hepatitis. hepatosplenomegally is always present. Find Foam cells in liver and bone marrow.

Other conditions presenting with neonatal jaundice:

1. Inherited disorders of bile salt metabolism
2. Chromosomal Disorders (trisomy 18 & 21)
3. Peroxisomal disorders (VLCFA in plasma)
4. Cholestasis secondary to TPN.

The Child with Painful Joints

In a toddler or infant pain may present as a reluctance to use a limb or as a pseudo-paresis.

Assessment.

Normal history. Note that **pathological fracture** should be considered even if no history of significant trauma. Also need to consider underlying pathology even with a history of significant trauma.

Examination is the usual **LOOK**, **FEEL**, **MOVE** (active and passive).

Comment on the child's general appearance. Does the child look sick? Is the child holding a limb protectively?
Is there any evidence of muscle spasm or muscle wasting?
Is there any swelling?

Feel the child's pulse and skin for fever and sweating.
Slide the fingers down the length of a suspected bone.
If swollen comment on:

1. Position
2. Colour
3. Temperature
4. Tenderness
5. Size
6. Shape
7. Composition
8. Relation to surrounding structures
9. Surface

Movement essentially involves an assessment of function., ACTIVE and PASSIVE with limitations to the degree of movement should be recorded.

Percussion is sometimes useful. For example, looking for tenderness over the spine.

Differential diagnosis of the painful joint.

Swelling	**No Swelling**
Septic arthritis	Irritable hip
Haemophilia	Slipped upper femoral epiphysis
Acute rheumatism	Pulled elbow
Tuberculous Arthritis	Adolescent disc protrusion and spondylolisthesis
Juvenile rheumatoid arthritis.	
Intra-articular fracture	
Henoch-Schoenlein purpura.	

1. Septic Arthritis.

Hot, swollen, tender joints.
Toxic, feverish child.
One or more affected joints being held in position of maximum comfort.
Investigation: Blood culture, CRP, FBC, X-ray. Consider joint aspiration.

Commonest organisms: ***Staphylococcus aureus***, ***Haemophilus influenzae***, ***Escherichia coli***, ***streptococci*** and ***salmonella***.

Commence intravenous flucloxacillin urgently.
Apply traction or splintage to reduce pain.

If persisting pain, consider repeat aspiration or open drainage to minimise joint destruction.
Remember to watch for anaemia.
Physiotherapy.

Prognosis: Usually good. Osteoarthritis.

2. Haemarthrosis with Haemophilia or Christmas disease

Swollen tender joint with ROM
No systemic disturbance
Known bleeding disorder
Management is with cryoprecipitate 2 units / kg 12 hourly for 48 hours.

Monoarticular juvenile chronic arthritis.

Hot swollen joint with muscle spasm
Usually affects Knee joint.
Muscle wasting often present
Family history of HLA B27
Positive ANF
Iridocyclitis on slit lamp examination.

Management involves anti-inflammatory drugs and physiotherapy.

Rheumatic Fever
Acute febrile systemic disorder affecting mainly the heart and joints. Caused by a group A beta-haemolytic streptoccoccus. Usually occurrs between 5 - 15 yrs.
Recent sore throat
Ill child with high fever
Hot swollen painful joint.

Duckett jones criteria:

Major	Minor
Carditis	Fever
Arthritis	Arthralgia
Nodules	Raised ESR
Erythema Marg.	Raised ASO
Chorea	Prolonged PR interval

Need 2 major criteria or 1 major & 2 minor criteria both with evidence of previous streptococcal infection.

Management: Aspirin and penicillin.

Tuberculous Arthritis.
Siffness, muscle spasm, weakness
Insidious onset.
Cool swollen joint
sysytemic upset with loss of energy, appetite, night sweats and low grade fever.
Heaf test , EMU, CXR

Management: Rifampicin , Isoniazid, Traction and gentle mobilization.

Intra-articular fracture

Warm, swollen, painful joint.
History of trauma.
ROM
Management: Reduction and immobilization.

Henoch- Schoenlein Purpura

A common vasculitis of immune mechanism (IgA and C3 deposition)
Non thrombocytopaenic purpura,arthritis, abdominal pain and nephritis.
Path: Vasculitis of venules and capillaries, with proliferative glomerulonephritis.
Assoctd. with URTI esp. streptococcal. Peak age 5 - 15 years.
Clinically:
1. Skin 100% Maculopapules / urticaria on extensor surfaces and buttocks.
2. Joints 80% Painful periarticular swelling of knees, elbows and small joints of hands. Resolves in days.
3. Abdominal pain 70% ; Malaena 20%: Intussusception 5%
4. Nephritis 50%

Investigation: Urine for blood and protein. Renal biopsy if persistent.

Management: Supportive. Corticosteroids used by some for abdominal pain

Prognosis; Recovery within 7 - 10 weeks. Nephrotic syndrome in 5%
Accounts for 10% of chilhood end stage renal failure.

Irritable hip (Transient synovitis, coxalgia fugax)
Onset of limp and pain in one hip or knee
No other evidence of illness
No swelling but restriction of movement at the hip joint.
Management is bed rest and tractio for 7 - 10 days.

Slipped upper femoral epiphysis.
Pain in hip, thigh or knee
No swelling but ROM
May have true shortening
X - ray of hip joint shows capital epiphysis sliding downwards and backwards.

Due to imbalance of growth and sex hormones.. Relatively low sex hormone.
Bilateral in 25%
Retinacular vessels may be damaged.
Management: If < 1/3 slip - threaded screws. Otherwise allow spontaneous fusion . Then consider osteotomy of Dunn or Souther. OA may result.

Pulled elbow.
Sudden jolt to arm results in painful arm with reluctance to move.
Pseudoparalysis.
Cause is distal dislocation of radial head through the annular ligament.
Requires manipulative reduction.

Adolescent disc protrusion.

Abnormal gait and tilted posture.
Lumbar muscle spasm and restricted straight leg raising.
Needs bed rest and immobilization.

Other "pain free" conditions to read about:

Perthes
Juvenile chronic arthritis
Congenital dislocation of hip
Osgood schlatter disease.

Asthma

Definition
Reversible bronchoconstriction occurring secondarily to airway hypersensitivity.

Asthma clinically consists of a triad of *cough, dyspnoea and wheeze*. The airway narrowing that occurs, results from a combination of bronchiolar muscle spasm, mucosal oedema and increased mucus production.

There is a strong association with atopy. 70% of children with asthma are atopic. This includes eczema, urticaria and hayfever.
Asthma affects 10-15% of the population and its prevalence is increasing having risen 3 fold over the past 40 years. Approximately 80% develop symptoms by 5 years of age.

Diagnosis and treatment are important, as there are approximately *50 child deaths* per year in the UK.

Pathophysiology of bronchoconstriction

Acute, late and *chronic* changes

Acute:

Precipitant--------> IgE (mast cell)--------> histamine + leukotriene
or
Increased airway hyperresponsiveness via a vagal response.

Late:

Onset 4 - 6 hours and lasting up to 10 days, a result of mast cell and alveolar macrophage release of leukotrienes, prostaglandin's and thromboxanes. Chemotactic factors also attract neutrophils, eosinophils and macrophages. Platelet activating factor results in inflammation and oedema of the airways.

Chronic:
Results from damage to respiratory epithelium leading to hyperresponsiveness.

Precipitants:
1. URTI
2. Exercise
3. Cold air + humidity (fungal spores)
4. Emotional
5. Pollen, mould
6. House dust mite
7. Animals
8. Food such as cola drinks, ice, cooking oil
9. Irritants such as paint, smoking and paraffin heating.

Symptoms and signs:
Persistent / recurrent nocturnal cough, wheeze, breathlessness, abdominal pain and vomiting due to forceful coughing.
Atopic eczema which often improves during exacerbation's.
Stunted growth and delayed puberty.
Poor school attendance.
Nasal flaring.
Tracheal tug.
Tachypnoea.
Pigeon chest deformity
Widespread wheeze with coarse crackles.
Remember the silent chest in severe asthma – this is a terminal sign
Cyanosis.
Difficulty speaking in sentences.
Pulsus paradoxus.

Investigations:
1. Peak expiratory flow rate
2. Response of PEFR to bronchodilators
3. FVC
4. CXR
5. FBC
6. RAST

Management:
1. Allergen avoidance
2. Home record card
3. Medication-

(i) Beta agonists used as required
(ii) Sodium cromoglycate
(iii) Low dose inhaled steroid
(iv) Long acting beta 2 agonists
(v) Slow release theophylline
(vi) Ipratropium bromide
(vii) High dose inhaled steroids
(viii) LTA
(ix) Oral steroids

Delivery systems

1. Metered dose inhalers +/- Aerochamber / volumatic / nebuhaler
2. Dry powder devices - Accuhaler, Turbohaler, diskhaler, rotahaler
3. Breath activated devices
4. Nebulisers
5. Syrups – questionable role

Group exercise1

1. Define asthma.

_____

2. How common is it?

_____

3. How many children die each year as a result of asthma?

_____

4. What conditions are associated with it?

_____

5. What factors may precipitate an attack?

_____

6. What are the Symptoms & signs associated with asthma?

7. What form of treatments are available to treat asthma?

_____

Group Exercise2

A 9-year-old boy presents to casualty with increasing cough and wheeze.
He is a known asthmatic having been diagnosed at 4 years of age.
His current treatment consists of ventolin (salbutamol) 200mcg whenever he needs it and flixotide (fluticasone) 125mcg twice daily using a metered dose inhaler (MDI) directly into his mouth.
He is 130cm tall and his peak expiratory flow rate on arrival in casualty is 150 l/min. (Expected =275)

1. What would be your immediate management in casualty?

_____

2. Referring to the expected PEFR chart, what can you say about his regular treatment?

_____

3. What is your assessment of the mode of drug delivery?

_____

4. Name 3 types of Inhaled steroids.

_____

_____

_____

5. Name 2 types of short acting beta 2 agonists

_____ _____

6. For each of the following device, give the age range at which use would be appropriate:

(a) Aerochamber _____
(b) Volumatic _____
(c) Turbohaler _____
(d) MDI _____

Paediatric clinical examination

Squint

1. Comment on general appearance; sclera etc
2. Check symmetry of light reflection.
3. Check eye movement in all directions (draw union jack).
4. Look for corneal opacities & catarracts.
5. Determine whether squint is alternating or paralytic - *COVER TEST* *
6. Check red reflex (Leukocoria = catarract, retinoblastoma or ROP).
7. Fundoscopy
8. Assess visual acuity

Understanding paralysis of eye movement.

3 nerves involved: (i) Oculomotor (III) nerve
(ii) Trochlear (IV) nerve
(iii) Abducens (VI) nerve

Oculomotor Nerve.

Oculomotor innervates superior, inferior and medial rectus as well as the inferior oblique and levator palpebra superioris.

Paralysis results in:
1. Ptosis
2. Pupil dilatation
3. Eye in " Down and out " position.

Trochlear Nerve.

Supplies the superior oblique muscle. Paralysis therefore results in the eye to deviate upward and outwards

Abducens Nerve.

This is the most common due to the long path of the VI th nerve.
Supply is to the Lateral rectus muscle. Paralysis therefore results in medial deviation of the eye and inability to abduct beyond the midline.

Causes of Nerve palsies:

1. *Third nerve palsy*:
Third nerve palsies are usually congenital. They are often associated with developmental anomalies or birth trauma. If acquired, be suspicious of an intracranial neoplasm or aneurysm. Other causes include inflammatory or infectious lesions, head trauma, post viral syndromes and migraine.

2. Fourth nerve palsy:
Because this nerve has a long intracranial course, it is susceptible to trauma.

In children they are usually congenital rather than the result of trauma.
Children usually present with a head tilt to the shoulder opposite the affected eye.
This minimises deviation and the associated double vision.

3. Sixth nerve palsy:
Congenital sixth nerve Palsies are rare. In neonates, a transient palsy may occurr which resolves spontaneously by six weeks .A benign sixth nerve palsy may occurr in older children often preceded by a febrile illness or URTI.
other causes
- Hydrocephalus
- Intracranial tumours
- Trauma
- Vascular malformations
- Meningitis
- Gradenigo syndrome (Otitis media, mastoiditis and petrosistis with oedema of the dura which pinches the sixth nerve against the petrosphenoidal liganent.

Duane Syndrome
Congenital disorder of ocular motility characterised by retraction of globe on adduction. This is due to cocontraction of medial and lateral recti. Patients may exhibit impairment of abduction, adduction or upshoot or downshoot of the eye.
Occurrence is either sporadic or Autosomal Dominant.

Mobius syndrome
Congenital facial paresis and abduction weakness. Facial palsy is commonly bilateral, asymmetric and incomplete. Uncertain aetiology.

Brown syndrome- Restricted elevation of eye in adduction

Gait Examination

Introduction

Introduce yourself, ask the child's name and ask if they have ant difficulty with walking.
Remember that 30% of children with cerebral palsy have normal intelligence.

Observation - Comment on presence or absence of:

- Any visible aids in the room or on child*
- Any postural deformity
- Muscle wasting
- Scars – Look at the heels (achilles lengthening), Hamstrings and Hip Adductors if accesible.
- Look at the childs shoes for evidence of unequal wear and or shoe raises.

Gait - comment on

- Stability
- Associated movement and position of arms and hands
- Base - narrow/broad-based
- Posture of legs - i.e. flexor/extensor/scissoring**
- Feet position/heel strike i.e. toe walking

Further assessment

- ask to run
- ask to walk on heels/toes/outside of feet (Fog test)
- heel toe walking in straight line
- hopping & balancing on one leg

Finally

Summarise your findings and suggest a diagnosis e.g. normal, spastic diplegic, hemiplegic, etc.

* Splints, crutches, walking frames, shoe raises.

** Refer to lecture for demonstration of muscle groups involved in lower limb

Notes