2003 OSCE Handbook

The world according to Kelly, Marshall, Shaw and Tripp
Our OSCE group, like many, laboured away through 5th year preparing for the OSCE exam. The main thing we learnt was that our time was better spent practising our history taking and examination on each other, rather than with our noses in books.

We therefore hope that by sharing the notes we compiled you will have more time for practice, as well as sparing you the trauma of feeling like you’ve got to know everything about everything on the list. You don’t! You can’t swot for an OSCE in a library!

This version is the same as the 2002 OSCE Handbook, except for the addition of the 2002 OSCE stations.

We have used the following books where we needed reference material:

- J. Murtagh, General Practice, McGraw-Hill, 1994

These are good books – buy them!

*Warning: This document is intended to help you cram for your OSEC. It is not intended as a clinical reference, and should not be used for making real life decisions. We’ve done our best to be accurate, but don’t accept any responsibility for exam failure as a result of bloopers....*

Hope it goes well,

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Students at the Wellington School of Medicine

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Introductory Tips

Approach to a Station

General Approach

- Read instructions carefully
- *Stop and think*: despite the pressure of time this is important – don’t just launch in
- ALWAYS introduce yourself to the patient
- Appear warm and confident to the patient
- Use your real bedside manner – don’t step out of the ‘role play’

At the end:

- Forget the station – especially if it went badly. Focus on the next station
- Don’t rush between stations. There’s plenty of time. Take some time to breath deeply!
- Check the sample questions at the end of this book to give you a feel for what they’re after

History Station

- Form the differential diagnosis as soon as you know the presenting complaint - (if the PC is given in the station instructions don’t look up from the instructions until you have the differential in your head). See the "History outline" section for more tips
- Always start with open ended questions
- Try and phrase closed questions as open ones – rather than ‘were there coffee grounds in your vomit’ try ‘was their anything different you noticed about your vomit’. They’ll tell you it was green even though you were about to forget to ask about bile. Once they’ve said ‘no’, for the examiners sake ask ‘So no blood, bile or coffee grounds then?’
- Only ever do a focused history based on the presenting problem (you’ve only got 4 – 5 minutes):
  - History of presenting illness: relevant positives and negatives only
  - Past Medical History
  - Medications and allergies
  - Family history
  - Social History – especially smoking and alcohol
- Consider:
  - What organ system am I dealing with
  - What are the likely causes
  - What risk factors may have contributed
  - What are the possible complications
- If the patient (or you!) dries up:
  - Repeat their last statement
  - Don’t rush. *Silence is OK*
- At the end if you have time:
  - Summarise back to the patient (especially if you’ve run out of ideas – buys time!): ‘So the main problem is…. Anything we’ve missed that you think is important…. This has left you feeling … You’d like me to ….‘
  - Ask if they have any questions or anything else that they would like to tell you

Education Stations

- Begin by determining the patient’s current knowledge. Emphasise accurate knowledge and correct any misconceptions:
  - Explain findings
  - Ask what they already know
  - Discuss prognosis
  - Outline plan
- At the end get them to explain it back
- ‘How does all that make you feel’
- Consider preventative measures:
  - Alcohol/drug/smoking (Quitline number is 0800 778 778)
  - Counselling
- Screening: breast, cervical, skin
- Infectious diseases: STDs, Vaccination
- Diet and exercise
- Stress reduction
- Environmental/occupational hazards
- Injury prevention (especially kids)

**Behaviour change stations**

- Help patient clarify problem - where in the cycle of change are they?
  - Do you want to change?
  - Why do you think you need to change
- Knowledge - what do they know about the problem
- Attitudes and fears - how do they feel about changing
- Practices - what have they already tried to change
- Barriers - what stopped them succeeding
- Validate their concerns and attempts
- Educate - problem is common, difficult to change on first attempt etc.
- Suggest strategies for change - need to link the change in behaviour with something that is very important to them eg stopping smoking and living to see the grandchildren grow up, stopping drinking and been able to stay at work.
- Let patient choose the strategies most appropriate to them
- Set goals and prioritise
- Arrange follow-up and on-going support

**Examination Stations**

- Ask permission to do the exam
- Don’t wash hands – the purists can point out that they normally would but don’t have time!
- Check the light
- Check the patient’s position
- Tell the patient what you’re going to do
- Show consideration for pain
- Keep patient draped – ask permission before uncovering/removing clothing. Get the patient to do as much as possible
- Assist the patient off the table
- Invite them to get dressed
- Explain findings

**Communication Skills**

- Remember their name and use it
- The two things patients want most from their doctor is for the doctor to listen and to explain conditions, investigations and treatment plans clearly
- Good listening skills: good eye contact, use silence, don’t interrupt
- Acknowledge the emotions they show
- Care about their condition – don’t forget the empathy
- Offer praise: ‘you seem to be coping well’
- When discussing impressions or findings, identify their emotional response
- Don’t give false reassurance or information you’re unsure of
- If discussing a poor prognosis, discuss family and community supports
- Address the patient’s concerns!

**History Outline**

- Presenting complaint - form your differential diagnosis now to guide your questions. This list should be brief (eg max 5-6 diseases) otherwise you won’t be able to keep it in the front of your mind. The differential list is made up of two “types” of disease:
  - The most likely diseases which could cause the symptoms
  - The most serious diseases which could cause the symptoms
- History of Presenting Complaint
• Start with open-ended questions eg ‘Tell me about…’ Don’t interrupt till they dry up – after they’ve done this for the preceding 30 students they’ll recite their whole blurb if not interrupted!
• Symptom assessment: explore the presenting symptom fully using more direct questions. (Eg SOCRASAP = site, onset/duration, character and severity, radiation, aggravating/reliving factors, associated symptoms, previous episodes)
• Review of systems (ROS): ask the ROS questions for the systems which could be involved. Eg for chest pain you need to ask the CVS, respiratory and GI ROS questions.
• Risk assessment: try to determine the level of risk a patient has for each differential diagnosis. Eg for chest pain ask about smoking, high cholesterol, diabetes (DM), hypertension and family history of MIs
• ‘Is there anything you think is relevant that we’ve missed?’, “any other symptoms or changes you’ve noticed”.
• General Medical History: Any other illnesses, ever been in hospital, blood pressure
• Medications and Allergies, including OTC drugs, pain killers, contraceptives
• Family History
• Social History: Consider the following as you have time
  • Smoking, alcohol, drugs
  • Lifestyle factors: diet, exercise, sexual history
  • Occupation: either it could be causative (eg asbestos exposure in a respiratory history) or illness will affect their ability to work
  • Problems/stressors
  • If appropriate, exposure to allergens – pets, occupational exposure, hobbies etc
  • Overseas travel
  • Who lives at home with you? What other supports do you have?
  • If elderly:
    • Activities of daily living: DEATH: dressing, eating, ambulatory (ie getting around), toileting, hygiene
    • Instrumental activities of daily living: SHAFT: Shopping, housework, accounting (ie money management), food preparation, transport
• Systems review:
  • A systems review in one minute, for systems not covered in the HPC. Only if you’re stuck. It will have a very low yield of both responses and marks! For each system keep the questions open (it’s quicker and they’ll just give you the positives)
  • General: Any changes in weight, appetite, sleep or energy. Fevers
  • Cardiovascular: any chest pain, unusual heart beats or ankle swelling
  • Respiratory: any trouble with your breathing or a cough
  • Neuro: any dizziness, blackouts, strange sensations or weakness
  • Psych: How have you been feeling in yourself lately
  • Endocrine, metabolic: covered in general and in renal
  • Renal: Any problems with your waterworks (including going more often)
  • GI: any vomiting, tummy pain, or change in your bowel motions
  • Gynaec: Any change in your periods, any vaginal discharge
  • Musculo-skeletal: any problems with your joints or with weakness
  • Skin: anything different you’ve notice about your skin (eg rashes, itching, etc)
• Summarise back:
  • Key issues for patient,
  • Their biggest worry, etc
  • ‘Any questions you’d like to ask’?

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  - Psychiatric
  - Metabolic/Endocrine/Blood
  - Gastro-intestinal
  - Renal
  - Genito-urinary
  - Obstetrics/Gynaecology
  - Musculo-skeletal
  - Skin

Management Options

- Listen!!!
- Reassurance
- Education
- Lifestyle:
  - Smoking/drinking
  - Diet
  - Exercise
  - Stress reduction
- Supports/support groups
- Psychotherapy
- Medication (remember O2 if acute, and pain relief for everything)
- Surgery
- Referral/multidisciplinary involvement
- FOLLOWUP

Past OSCE Stations

2002 OSCE Stations

- History:
  - History of headaches, plus differential (tension headaches) and two reasons
  - History of abdominal pain, plus differential (recurrent biliary obstruction)
  - History of a rheumatological condition, give most likely diagnosis (Polymyalgia rheumatica) plus two tests to do
  - History of a 49 year old woman with menorrhagia and irregular periods, plus two differentials (peri-menopausal changes and dysfunctional uterine bleeding), plus two treatment options
  - History of failure to thrive in a 9 month old, plus 2 differentials
  - History of sudden onset chest pain in 34 year old male. Give 2 differentials (pneumothorax)
  - History of sudden loss of vision, plus differential (temporal arteritis), plus immediate management plan
  - Alcohol history for 8 minutes, plus diagnoses (Hazardous drinking, alcohol dependence), plus reasons for diagnoses
- Examination:
- Take blood pressure in a healthy volunteer plus questions on technique
- Examination of complete CV system excluding BP and legs, plus present findings (there was a murmur), plus diagnosis
- Neuro Exam of the lower limb, limited to reflexes, light touch and vibration sensation in an actor, plus differential (peripheral neuropathy)
- Examination of the hand in an actor with simulated ulnar nerve damage, plus name which nerve was damaged, plus two reasons for that diagnosis
- Examination of pulse and heart for rhythm and rate only (patient had AF), plus read a ECG rhythm strip for rate, plus 3 causes of atrial fibrillation
- Investigations:
  - Interpretation of abdominal CT with renal tumour extending into the renal vein and IVC, plus differential in order of probability, plus any additional imaging
- Advice/Explanation:
  - Educate a parent about the first febrile convulsion in a 2 year old
  - Explain an educational pamphlet to a woman who had had a bowel resection, plus determine what follow up she needed, plus refer to appropriate allied health professional
  - Education about steroid withdrawal

2001 OSCE Stations

- History:
  - Haemoptysis: Short history of coughing up blood. Take history. NB ask occupation – he was a plumber. In the final minute, interpret x-ray (apical consolidation + trachial deviation)
  - Failure to thrive in a 6 month old. Take history from the mother. Was her first child and she was slow to introduce solids. What management would you recommend. (Include examine the child, further investigations and education).
  - Urinary retention. Elderly man was writhing in pain. Once you’d established he was in urinary retention, you couldn’t get any further history from him until you said he needed to be catheterised. He then relaxed and you could continue the history.
  - Dyspepsia (GP station). History of chronic epigastric pain. Give preferred diagnosis (ie had to decide between ulcer and cancer pain)
  - Dysphagia: patient with trouble swallowing
  - Patient with melena and rectal bleeding
  - Antenatal booking history at 8 weeks. Take a history then state 3 risk factors for the pregnancy
  - Psych station: take a history of someone presenting with panic attacks. Give differential diagnosis
- Examination:
  - Peripheral vascular on leg: history of claudication. Exam the leg. They watched carefully for where you palpated and auscultated the femoral pulses. ‘What simple bedside check could you use for peripheral arterial blood flow?’ → Ankle Brachial Index (ABI) using Doppler ultrasound
  - Examine hands of a patient with advanced Rheumatoid Arthritis
  - Listen to a murmur. What is it and why? Go straight to auscultation – forget inspection, etc
  - Shoulder exam on a patient pretending to have a dislocated shoulder (but he could still move it). What investigations and management would you recommend?
  - Read an ECG (3 minutes given) – it had ischaemic change and was in AF. Asked about two treatments/drugs you would recommend.
  - Assess a patient’s visual fields and state the deficit. He had his glasses on (needed to ask him to take them off)
- Advice/Explanation:
  - Teach a patient how to use a GTN spray, including side effects, when to use it, how to use it, and what to do if it doesn’t work
  - Advise a patient on how to start a combined oral contraceptive. They gave you a pack (and it was a two track pack that none of us had ever seen – reading the instructions on the pack helped a lot!)
A patient needs a blood transfusion. Obtain informed consent.

2000 OSCE Stations

History Stations:
- Seizure: presented in A&E after a collapse in a supermarket. Faint or fit (he had a previous head injury)
- Headaches: 16 year old girl with history of migraines
- Dysuria: Male with a cheesy discharge from his urethra
- Diarrhoea: had campylobacter enterocolitis
- Depression (11 minute station): Not sleeping, take history, summarise findings, give a diagnosis
- Infertility: was PID. Needed to give differential
- Dysphagia: Guy has lost weight, drinks and smokes. What investigations would you do
- Chest pain: angina

Examination:
- Chest: respiratory: Young guy with asthma. After 4 minutes interpret spirometry results
- Peripheral vascular on leg: Diabetic with history of claudication. Examine his leg
- Neck: examine the thyroid
- Knee
- MMSE: on a woman who has recently had a fall, then give a differential

Imaging: Barium Enema of apple-core lesion (not observed): What is it, how to do it, what does it show, possible diagnosis, 5 things that are done to optimise the interpretation

Advice/Explanation:
- Cervical smear: advice on what an abnormal smear is
- Thyroid replacement therapy
- Child sleeping problem: advise a mother how to manage a 2 year old’s sleeping

1999 OCSC Stations

History:
- Haematuria
- Breast lumps: history and differential
- Diarrhoea and vomiting in a child (not present) and differential
- Sleep apnoea: history and differential
- Head injury: assessment and GCS (scoring sheet given)
- Alcohol: abuse or dependence?

Exam:
- Retinal exam (on a model)
- Neurology of the leg
- Abdominal exam
- Identify the murmur: Aortic Regurgitation

Investigations:
- Xray – aortic trauma
- Urinanalysis: how to do it and 6 differentials of proteinuria

Advice/Education:
- Patient wanting an unnecessary ECG – reassure him he doesn’t need it
- Depression: What drugs
- Pre-op assessment: risk factors for General Anaesthetic and Operation
- Starting OCP
- Asthma: purpose of reliever and preventer, and effects of smoking

1998 OCSE Stations

History:
- Menorrhagia and Dysmenorrhoea in a 35 year old lady
- Claudication: Prognosis, severity, differential
- Abdominal pain: Gallstone like. Differential
- Depression: presents with trouble sleeping

Examination:
• Deaf left ear, increasing over 2/12. Test CN V, VII, VIII. Determine what sort of deafness and give differential
• Developmental Assessment: 1-minute video of a 12/12 child. List features shown. What is normal, abnormal for a child this age
• CPR: 5 cycles on a mannequin. Name 5 ECG traces. Treatment for VF, VT, Asystole
• Knee Exam
• Heart: JVP, heart rate, pulse
• MMS: 70-year-old man admitted for a ‘fall’. Differential: dementia, stroke, delirium, etc
• Investigations:
  • Blood film/results. Megaloblastic anaemia due to pernicious anaemia. Diagnosis, treatment and complications of B12 deficiency
  • Radiology: Large bowel obstruction. Why do a CXR also? Further tests (eg Barium enema)
• Education/Advice:
  • Explain how to use a spacer and steroid inhaler
  • Explain to a man why it was necessary to undergo cystoscopy, given history of haematuria. As soon as ‘cancer’ was mentioned, he went off
  • Newly diagnosed diabetic: Diet, exercise, general health care
  • Immunisation: discuss 3/12 schedule with Mum: importance, listen to concerns, reassure
• Starting a woman on erythromycin (remember contraindication, side effects, ?OCP use → 7 days rule)

**Older OSCE Stations**

• Set 1
  • Teach peak flow
  • Look in ear: Otitis Media
  • MSE: higher cognitive function (delirious patient)
  • Examine hip
  • Lower leg neurology
  • Blood pressure
  • Alcohol History
  • Explain febrile convulsion to a hysterical mother
  • History of PR bleeding
  • Drug interactions
  • Incontinence history
  • Explain oesophageal reflux: prognosis/treatment
  • Dipstick urine diagnosis
  • Abdominal exam
  • PE history
  • CXR: child
  • Haematology: Fe studies, anaemia
  • X-ray: subdural haematoma
  • Micro: Hep A, B, C and CMV results
  • O&G: Growth in utero
  • CXR: Pneumothorax
  • DM: retinopathy
• Set 2:
  • Paediatrics: Developmental milestones
  • Haematology: anaemia
  • Medicine: History of RUQ pain radiating to back
  • Pharmacology: Drug history, explain effect of antibiotics on oral contraceptive
  • GP: explain use of asthma inhaler
  • Radiology: interpret plain AXR and CXR
  • Psych: history of poor sleep
  • Psych: history of post-natal depression
  • Orthopaedics: examine knee
  • Medicine: examine cranial nerves
- Medicine: CV examination
- Surgery: history of painless jaundice
- Surgery: history of claudication
- Medicine: MMSE – evaluate causes of confusion in the elderly
Fever is >37.2°C

- Rigors: uncontrolled shivering or violent shaking. Usually due to more serious diseases eg pneumonia, pyelonephritis, cholangitis
- Night sweats: moderate sweats are common with anxiety, stress or too much bedding. Drenching sweats suggest infection of lymphoproliferative disease
- Remember lymph nodes in your exam

**History**

- Symptom assessment: onset of fever, pattern etc
- ROS: headache, neck stiffness, photophobia, cough, SOB, diarrhoea and vomiting, pain anywhere, dysuria, weight loss, night sweats, tiredness
- Risk assessment:
  - Overseas travel
  - Infectious contacts
  - Food
  - Sexual history
  - Contact with animals, insect bites, 
  - Immunisations, recent antibiotics
  - Immunosuppressive illness or drugs
  - For ?hepatitis, add in:
    - Sexual history
    - IV drugs
    - Tattooing or body piercing
    - Transfusions/transplants
    - Health care worker

**PUO**

- Infection
  - Abscesses - subphrenic, hepatic, pelvic, renal
  - Viral - CMV, EBV, VZ, HIV, dengue fever
  - Bacteria - Tb, brucellosis, typhoid, leptospirosis, Q fever, actinomycosis, salmonella
  - Parasites - Malaria, toxoplasmosis
- Neoplasms
  - Lymphomas (a fever associated with leukaemia is usually infective)
  - Solid tumours esp GI and renal cancers
- Autoimmune/inflammatory conditions - cranial arteritis, SLE, PAN, RA, polymyalgia rheumatica, Still's disease, IBD, rheumatic fever
- Drug reaction - especially penicillins and sulphonamides. May start months after starting the drug.
- Other - intracranial pathology, factitious, familial conditions
- Exam: nails, teeth, skin, temporal arteries, nodes, hepatosplenomegaly, joints, vaginal, PR
- Investigations: cultures, FBC, ESR, CRP, U+E, LFTs, MSU, CXR

**In children**

- Many children with fever > 39°C have no focus found. Most of these have a viral infection. 5% have UTI, 1-3% have pneumococcal bacteraemia which may rarely (5%) cause meningitis
- All children with unexplained fever should have a urine dipstick performed
- Those at risk of serious infection require referral. All others should be reviewed in 24hrs if there is no improvement or earlier if they deteriorate
- At risk children: neonates, immunocompromised, multiple congenital abnormalities, toxic appearance, epidemiological risk factors eg Maori or PI in some areas, and possibly those not fully immunised especially against HIB
In the post-operative patient
- Mild fever may be due to tissue damage but need to consider pneumonia, incision site infection, DVT, peritonitis, UTI, IV cannulae sites, meningism, endocarditis.
- Ix: consider blood cultures, CXR, MSU, abdo USS

In the neutropenic patient
- Fever > 38C for 4 hrs, > 38.5C at any time or patient is "unwell"
- History: pain anywhere, superficial infections, perianal, respiratory, GIT and UTI symptoms
- Exam: vitals, feel peripheries, mouth/throat, skin and lines, chest, abdo, perianal (no PR)
- Investigations: Blood cultures - central line vs peripheral, CRP, CXR, Swabs
- Rx: empiric ABs eg gentamicin + ticarcillin or imipenem +/- vancomycin (good for infected lines and staph). If fever for 72hrs consider change of ABs or antifungal Rx (amphotericin)

NB - the absence of a fever does not exclude the existence of a serious infection and the need to do blood cultures. If someone does have a high fever do a blood culture.

67: Pain
- The pain itself
  - Site
  - Onset and time course
  - Character/Quality
  - Radiation
  - Aggravating and relieving factors (eg movement, breathing, eating, exertion, drugs which help, etc)
  - Severity – get in detail: scale of 1 to 10, how far can you walk for, does it wake you at night, etc
  - Associated symptoms
  - Previous episodes
  - Anything you think might be the cause
- If relevant:
  - Do you get it at night
  - Affect on function
- Then questions related to likely diagnoses:
  - Other symptoms for each diagnosis
  - Risk factors for each diagnosis

69: Pallor

Causes
- Racial
- Familial
- Cosmetic
- Pathological
  - Anaemia
  - Shock
  - Stokes-Adams attack
  - Vasovagal faint
  - Myxoedema
  - Hypopituitarism
  - Albinism

Anaemia
- Symptoms
  - Tiredness
  - Muscle weakness
  - Headache
  - Lack of concentration
  - Faintness/dizziness
- Dyspnoea on exertion
- Palpitations

**Signs**
- Pallor
- Features of hyperdynamic circulation: tachycardia, systolic flow murmur
- Specific signs
  - Jaundice – haemolytic anaemia
  - Koilonychia – iron deficiency

**History**
- Fe deficiency:
  - Intake: diet
  - Losses: GI, menorrhagia, malignancy, NSAIDs, anti-coagulants
- Folate deficiency:
  - Intake: diet, especially with pregnancy and alcoholism
  - Losses: small bowel disease
- Vit B12 deficiency:
  - Intake: diet, alcohol
  - Losses: previous gastric surgery, ileal disease or surgery; pernicious anaemia
- Haemolysis: Abrupt onset anaemia with mild jaundice
- Alcohol
- Marrow infiltration

**Differential**
- Microcytic: Fe deficiency, thalassemia, sideroblastic anaemia
- Normocytic: Acute haemorrhage; chronic disease; renal failure; pregnancy; hypothyroidism; haemolysis; bone marrow failure
- Macrocytic: B12 or folate, alcohol, liver disease, hypothyroidism, cytotoxic drugs; marrow infiltration; haemolysis

**Treatment:**
- IDA: correct identifiable cause; diet; iron preparations (ferrous gluconate)
- B12: Vit B12 (1000µg) IM every 2-3 days until body stores replenished THEN maintenance with 1000µg injections every 3rd month
- Folate: Oral folate 5mg/day (replenishes body stores in about 4 weeks)

**Ref:** Murtagh, General Practice 2nd Edition, Chapter 19, pp 156-163

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**54: Jaundice**

**Two types of bilirubin:**
- Unconjugated: If ↑↑unconjugated in a neonate → kernicterus: athetoid cerebral palsy, deafness, ↓IQ
- Conjugated: water soluble, conjugated in liver by glucuronyl transferase

**Causes in a Neonate**

- **Early onset (in 1st 24 hours):**
  - Always pathological
  - Causes: Haemolysis of any cause or sepsis:(respiratory distress + jaundice - not common)
  - Exam:
    - Maybe hydrops fetalis, large liver, large spleen (site of haemopoiesis in newborn)
    - Sepsis: especially breathing (indrawing and difficulty)

- **Jaundice in 1st week:**
  - Due to immature liver. Emphasis on extent of the jaundice (as well as consideration of the cause)

- **Treatment:** Phototherapy or exchange transfusion

- **Persistent jaundice:**
  - Causes:
    - ↑Conjugated bilirubin (go green) – needs treatment:
    - Liver obstruction abnormalities (eg biliary atresia, secondary to liver damage from infection, toxins, etc)
- Hepatitis/liver inflammation
  - Unconjugated (yellow) – needs treatment if high. Eg Breast milk jaundice – progesterone in breast milk delays maturation
  - Diseases picked up on Guthrie card causing jaundice:
    - Hypothyroidism
    - Galactosaemia
    - Cystic Fibrosis

**Causes in an Adult**

- Unconjugated hyperbilirubinaemia
  - Overproduction: Intravascular or extrahepatic haemolysis
  - ↓ Hepatocellular uptake: drugs, sepsis, starvation
  - ↓ Hepatocellular conjugation: Gilbert’s syndrome, neonatal jaundice, drugs, diffuse hepatic disease
- Conjugated bilirubinaema (cholestatic jaundice)
  - Impaired hepatocellular secretion: various syndromes, duct stricture, biliary cirrhosis, steroids
  - Hepatocellular cholestasis – impaired secretion plus liver injury as well: viral infection (eg EBV, Hep C), drugs, alcohol, cirrhosis
  - Extrahepatic obstruction: stones, carcinoma, strictures and congenital atresia

**History**

- Differentials:
  - Liver disorders
  - Bleeding/RBC disorders
- Symptom assessment: onset, duration (as per pain screen), previous occurrences
- ROS: fever, abdominal pain, pruritis, anorexia, nausea, easy bruising
- Risk assessment: IV drug use, overseas travel, tattooing, body piercing, sexual activity, health care worker, infectious contacts, overseas travel, immunisations, drug and medication use
- Stool colour: if pale then ↓ bile ⇒ obstructive jaundice
- Urine colour: if dark then ↑ urobilinogen ⇒ ↑ conjugated bilirubin
- Past medical history: including previous abdominal surgery (eg biliary)
- Medications
- Family history
- Social history: alcohol

- Ref: Murtagh General Practice 2nd Edition, Chapter 53, pp 532-547

91: Weight gain

**Differential diagnosis**

- Exogenous obesity
- Depression
- Endocrine: Hypothyroid, Cushing’s, acromegaly, hyper-prolactinaema, hypogonadism
- Oral contraceptives
- Pregnancy
- Fluid overload: Cardiac failure, liver failure, nephrotic syndrome

**Predisposing factors**

- Genetic    Family tendency
- Sex        More likely in women
- Activity   Lack of physical activity
- Psychogenic Emotional deprivation, depression
- Social class
- Alcohol
- Smoking    Cessation of smoking
- Drugs      Tricyclics
Consequences
- Cardiovascular (MI, hypertension, etc)
- Metabolic (NIDDM, etc)
- Mechanical (OA, obstructive sleep apnoea)

History
- Specific details of diet?
- Exercise?
- Do you have any special problems, such as getting bored, tense, upset or depressed

Examination
- BMI, degree and distribution of body fat
- Blood pressure
- Blood and urine sugar
- Visual fields, reflexes etc if appropriate
- MSE

Investigations
- BMI
- Waist-hip ratio
- Cholesterol/triglycerides
- Glucose (fasting)
- LFT
- TSH, T4
- U&E

Management
- Children: aim to maintain weight at current level as they often grow out of it during puberty. Must also consider emotion issues of obesity and address these
- Adults
  - Reduced energy intake
  - Change in diet composition
  - Increased physical activity
  - Behavioural therapy

Ref: Murtagh General Practice 2nd Edition, Chapter 70, pp 724 - 731

92: Weight Loss

Probable cause
- Stress and anxiety
- Non-coping elderly (adverse psychological factors, neglect, and drug effects)

Serious causes
- Poor intake:
  - DEPRESSION
  - EATING DISORDERS
  - Drug dependency (weight loss secondary to reduced nutrition)
- ↑Utilisation
  - DIABETES: thirst, polyuria, weight loss.
  - MALIGNANCY
  - Chronic renal failure
  - Chronic infections (Tb, HIV)
  - CHF
  - Hyperthyroidism
- ↑Losses, failure of utilisation, or catatonic state
  - Malabsorption
History
- Careful documentation of weight loss
- Determine food intake and appetite
- Psychological enquiry
  - Stress and anxiety
  - Depression
  - Self induced vomiting, distorted body image, etc
- Malabsorption
  - Describe motions
  - Difficult to flush
- Diabetes: Thirst, polyuria, weight loss
- Cancer: Smoking, general health
- Drugs

Examination
- Vitals
- Thyroid
- Abdomen
- Rectal

Investigations
- Bloods: FBC, ESR, Thyroid, glucose
- CXR
- Urinalysis

- Ref: Murtagh General Practice 2nd Edition, Chapter 71, pp 732 - 740

40: Difficulty Sleeping

Differentials
- Psychiatric disorder
  - Anxiety, stress
  - Depression
  - Nightmares
  - Dementia
- Drugs/Alcohol
  - Caffeine, alcohol, beta-blockers
- Medical conditions
  - Sleep apnoea
  - Restless legs syndrome (try gentle stretching of the legs, especially hamstrings and calf muscles, before going to bed. Drugs (if desperate) try clonazepam
  - Physical disorders: eg arthritis (or anything causing pain)
  - Bed-wetting/ nocturia
  - Reflux disease

Management
- Treat causes
- Explanation and reassurance
- What helps them to settle best: bath, music, etc
- Establish a routing before going to bed
- Avoid alcohol and caffeine in the evening
- Avoid heavy evening meals
- Have a warm milk drink before bed
- Sex last thing at night!
- Relaxation therapy, meditation, stress management

Fatigue differential
- Poor sleep
- Anaemia
• Depression
• Hypothyroid
• Any acute or chronic disease
• Medication (e.g., β-blockers and anti-histamines)
• Chronic fatigue

66: Oedema

• Refer to 13: Ankle Swelling on page 98
• May be generalised or localised – periorbital, peripheral or an arm (lymphoedema)

Causes
• Decreased plasma volume
  • Hypoalbuminaemia (nephrotic, chronic liver disease, malnutrition)
• Increased plasma volume
  • Congestive heart failure
  • Chronic renal failure
  • Drugs: NSAIDs, certain anti-hypertensives
• Idiopathic: Women; cyclical or persistent; unrelated to menstrual cycle; abdominal bloating; may affect hands and face as well as feet; often made worse by diuretics associated with headache, depression, tension.

Puffiness of face and eyelids
• Similar causes to generalised oedema, but must consider
  • Renal disease
  • Hypothyroidism
  • Cushing’s disease and corticosteroid treatment
  • Mediastinal obstruction (SVC obstruction)
  • Angio-oedema
  • Skin sensitivity (e.g., drugs, cosmetics, etc)

Treatment
• Treat cause where known
• Salt (sodium restriction)
• Diuretics: loop or potassium sparing

• Ref: Murtagh General Practice 2nd Edition, Chapter 70, pp 730 - 731

62: Nausea/vomiting

General Differential
• GI infections
• Systemic infections
• Surgical causes (i.e., GI obstruction)
• Other:
  • Alcohol/drugs/medication
  • Metabolic (e.g., DKA)
  • Pregnancy
  • ↑ICP
  • Psychological

Common Causes
• All ages:
  • Acute gastroenteritis
  • Motion sickness
  • Drugs: cytotoxics, antibiotics, opiates.
  • Various infections
  • Psychogenic (ALWAYS CONSIDER BULIMIA)
• Neonates: feeding problems
• Children:
• Viral infections/fever
• Otitis media
• UTI
• BILE STAINED VOMITING IN AN INFANT IS ALWAYS SERIOUS
• Adults:
  • Gastritis
  • Alcohol intoxication
  • Pregnancy
  • Migraine

**Serious causes**
• Bowel obstruction from any cause
• Severe infection
• Malignancy
• Intracranial disorders
  • Malignancy
  • Cerebellar haemorrhage
• Acute appendicitis
• Acute pancreatitis
• Acute MI (painless)

**Rarer causes**
• Organic failure: liver, renal, heart
• Labyrinthine disorders: Meniere’s, labyrinthitis
• Poisoning
• Achalasia
• Hypercalcaemia

**History**
• Drug intake
• Psychogenic factors (self-emesis)
• Weight loss
• Other GIT symptoms
• Symptoms suggestive of systemic disease

**Differentiating cause**
• Nature of vomit
  • Faeculent
  • Blood
  • Coffee-grounds
  • Bile
  • No nausea, possible projectile ↑ intracranial pressure
  • Surgical cause unlikely if no abdominal pain

**Examination**
• If fever present look for source of infection: ears, meninges, urinary tract, pneumonia, sepsis
• Abdominal examination
• Neurological examination: look for ↑ICP
• Level of hydration

**Investigations**
• Consider underlying cause
• Biochemical abnormalities from fluid and electrolyte loss

**Treatment**
• Drug induced: Metoclopramide 10mg orally, ondansetron, domperidone
• Motion sickness: Promethazine theooclote 25mg oral 60 mins prior to travel
• Vestibular disturbances: Phenothiazine derivatives, eg cyclizine
• Gastroenteritis: Metoclopramide 10mg orally
40: Enlarged Lymph Nodes

- Important features: size, tenderness, mobility, consistency (normal, rubbery or hard)
- Reactive (tender)
  - Infective - systemic (e.g. EBV, CMV, HIV, toxoplasmosis) or local (e.g. cellulitis occurring distally)
  - Non-infective - sarcoïd, connective tissues disease, dermatopathic (eczema, psoriasis), drugs
- Infected (abscess)
  - Staph or strep - tender, fluctuant, may require drainage if large or antibiotics fail
- Infiltrated
  - Benign - histiocytosis, lipidoses
  - Malignant - lymphoma (rubberly), metastasis (hard and fixed)

85: Unexplained Lump

- Also see 63: Neck swelling, lump or pain and 15: Breast lump

- The eight S’s:
  - Site
  - Size
  - Shape
  - Surface (texture)
  - Slipperiness (mobility)
  - Sensation (pulsation/warmth)
  - Sound (bruit)
  - Special tests (e.g. illumination)

63: Neck swelling, lump or pain

Midline lump
- Submental nodes
- Thyroglossal cyst (fluctuant and moves with swallowing and protrusion of tongue)
- Dermoid cyst (beneath chin and may also occur at angle of the eyes) = benign cystic teratoma
- Ectopic thyroid (rare. Often the only thyroid tissue present. Prone to becoming hypothyroid)

Lateral lumps
- Lymph nodes
  - reactive lymphadenopathy (NB tonsils and throat drain to anterior cervical nodes)
  - lymphadenitis (with or without abscess),
  - fixed and hard: tumour - primary or secondary (check for ENT tumours), mycobacteria (if < 5 then usually Non MTb, if > 5 then MTb), cat scratch (B. henslæ).
- Anterior triangle
  - branchial cyst (upper part of triangle, usually < 20yrs old. Cause probably embryological remnant or degeneration of a lymph node)
  - carotid body tumour (firm, pulsatile, opposite thyroid cartilage)
  - carotid aneurysm
  - parotid (superior) or lateral thyroid (inferior) tumour.
- Posterior triangle
  - Cystic hygroma (arise from jugular lymph sac, transilluminate)
  - Developmental remnants
  - Bronchial sinuses and cysts
  - Pancoast tumour
  - Cervical rib
- Other
• Involving the SCM muscle - torticollis, SCM tumour
• Pharyngeal pouch - base of left neck, soft indefinite mass, history of dysphagia
• sebaceous cyst, lipomas

**Neck pain**
• Usually from facet joint dysfunction but may be intervertebral discs, muscles or ligaments
• Causes include trauma, arthritis or idiopathic
• Serious disorders not to be missed
  • Angina
  • Subarachnoid haemorrhage
  • Neoplasia
  • Severe infection
  • Vertebral fractures or dislocations
• Examination
  • Inspection - lateral flexion of neck, level of shoulders, contour of neck form the side.
  • Palpation - C2 spinous process is palpable as is C7. C6 is palpable but not in extension.
  • Movement - flexion, extension, lateral flexion and rotation.
  • Neurovascular exam for nerve root lesions (C5 to T1) looking for dermatomal pain, paresthesia, sensory loss, weakness and/or hyporeflexia.

• Ref: Murtagh General Practice 2nd Edition, p552-556; OHCS 5th Ed. p568

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### 75: Shock

**If BP falls so that vital organs are not perfused adequately, the patient is shocked**

**Causes**
• Hypovolaemia - burns, bleeding, d&v
• Anaphylaxis - Type 1 IgE mediated hypersensitivity eg to: Penicillin, contrast agents, NSAIDs, latex, stings, eggs, fish, peanuts, semen, etc
• Septic - Gram -ive (or +ive) endotoxins
• Cardiogenic - Arrhythmias, cardiac tamponade, tension pneumothorax, MI, myocarditis, myocardial depression, valve destruction, PE.
• Endocrine failure – Addison’s/hypothyroidism
• Iatrogenic - Drugs eg anaesthetics, antihypertensives

**Signs**
• Hypovolaemic
  • Inadequate tissue perfusion:
    • Skin - cold, pale, blue, slow capillary refill
    • Kidneys - oliguria, anuria
    • Brain - confusion, restlessness
  • Increased sympathetic tone:
    • Tachycardia, narrowed pulse pressure
    • Sweating
    • Blood pressure - may be maintained initially (despite up to a 25% reduction in circulating volume if the patient is young and fit), but later hypotension supervenes
  • Metabolic acidosis - compensatory tachypnoea

**Additional clinical features may occur in the following types of shock**
• Anaphylaxis
  • Signs of profound vasodilation: Warm peripheries, low BP
• Erythema, urticaria, angio-oedema, pallor, cyanosis
• Bronchospasm, rhinitis
• Oedema of the face, pharynx and larynx
• Pulmonary oedema
• Hypovolaemia due to capillary leak
• Nausea, vomiting, abdo cramps, diarrhoea
• Septic
- Pyrexia and rigors, or hypothermia (unusual)
- Nausea and vomiting
- Vasodilation, warm peripheries
- Bounding pulse
- Rapid cap refill
- Hypotension
- Occasionally signs of cutaneous vasoconstriction
- Other signs - jaundice, coma (rare), bleeding due to coagulopathy

- Cardiogenic
  - Raised JVP
  - Pulsus alternans, ‘gallop rhythm’
  - Basal crackles, pulmonary oedema

**Investigations**

- All cases - Hb, PCV, WCC, Blood glucose, Platelets (coagulation), U&Es & Cr, LFT, Blood gases

**Treatment**

- General: Maintain airway, oxygen, head down
- Hypovolaemia
  - Bleeding - Take blood for cross match, Stop bleeding, IV fluids
  - Fluid loss - IV saline until BP rises. Treat cause.
- Anaphylaxis
  - Secure airway, remove the cause
  - Adrenaline 0.5ml 1/1000 IM, repeat every few minutes if necessary. Adrenaline may be nebulised for laryngeal oedema
  - Corticosteroids - prevent late symptoms, hydrocortisone 250-500mg IV, oral prednisone 20-40mg daily, 3 days min
  - Antihistamines - Not a substitute for adrenaline. Phenergan 25mg IV slowly or oral. H2 antagonist eg cimetidine may be useful.
  - Oxygen, IV fluids, nebulised beta-2 agonist, aminophylline
- Septic
  - Take blood for culture, specific antimicrobial therapy or IV cefuroxime or gentamycin if pathogen unknown. Give colloid. Refer to ICU for intensive monitoring and inotropes
  - Every effort must be made to identify the source of infection and the causative organism eg x-rays, US, CT, urine, sputum, CSF, pus drained from abscesses etc.
- Cardiogenic
  - Diamorphine 2.5-5mg IV for pain and anxiety, Correct cause eg arrhythmias, tamponade etc, Positive inotropes eg dobutamine, ↑renal perfusion by low dose dopamine, If pulmonary wedge pressure < 15mmHg give plasma expander

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### 27: Dehydration

**Mild <6%**
- Head: thirsty, restless, dry mucous membranes
- Heart: no signs
- Skin: cool, slightly mottled limbs, slightly decreased turgor
- Urine: concentrated

**Moderate 6-9%**
- Head: moderate thirst, dry mucous membranes, lethargic but irritable
- Heart: tachycardia
- Skin: cool, mottled limbs, decreased turgor (1 - 2sec), dry axilla
- Urine: output decreased

**Severe >9%**
- Head: drowsy and hypotonic, very dry mucous membranes
- Heart: rapid thready pulse, hypotension (<70 mmHg in children), or postural hypotension.
- Skin: cold, clammy, mottled limbs and trunk, decreased turgor (> 2 sec)
- Urine: oliguric or anuric

- Applies to children and adults. mls lost = normal weight in kg x % lost x 10
- Treatment: replace losses orally when possible otherwise use IV fluids. Check for electrolyte disturbances and ½ replacement rate if hypernatraemic

### 110: Use the Ambu Bag

- Check valve is functioning properly by squeezing the bag a couple of times.
- Connect oxygen line either directly or to a reservoir bag.
- Check that the tube you have connected is connected to oxygen by following the tube the whole way.
- Turn on oxygen flow at tank and at tap. Ensure flow is adequate (10L/min) and that you can feel it coming through.
- Hold mask tightly over face supporting the jaw adequately. May need pharyngeal airway (size = reaches from ear lobe to angle of mouth).
- When ventilating give approx 500ml tidal volume. Watch chest for symmetrical movement. Expanding abdomen may indicate poor airway control or excessive tidal volume

- For ET intubation pre-oxygenate with at least three puffs before inserting tube.
- For intubation must have:
  - Appropriate experience or experienced person beside you especially if acute or in a child
  - Laryngoscope (that works) and appropriately sized blade.
  - Correct ET size tube (m=9, f=8, kids = age/4 + 4 or size of their little finger). (NB- LMA size m=4, f=3).
  - Inflate cuff to check for leaks then deflate before inserting.
  - Tape to secure ET tube in place
  - Correct connector between ET tube and valve on the bag.
  - Insert tube so that black line just enters through glottis (space between vocal chords).
  - After placing ET tube look for symmetrical chest movement and listen for breath sounds in both axilla.
  - Complications: dental damage, endobronchial intubation, oesophageal intubation, excessive pressure from the cuff leading to subglottic necrosis and stricture.
Chest and Vascular

109: Respiratory Exam

- Introduce yourself. Wash your hands. Sit the patient upright.

General appearance
- Consciousness, comfort colour, posture
- Rate, depth and pattern of breathing. Accessory muscles
- Cyanosis, cachexia, rheumatological disorders (ankylosing spondylitis)
- Cough (wet or dry), wheeze, hoarseness, stridor, gurgling
- Sputum, inhalers, oxygen, IV lines etc

Nails and Hands
- Clubbing (if present check for HPOA – wrist tenderness), peripheral cyanosis, staining from cigarettes
- Wasting (Pancoast tumour) – finger abduction and adduction.
- Flapping tremour: late and unreliable sign of severe CO2 retention

Pulse
- Rate and rhythm (bounding in CO2 narcosis). Count respiratory rate (at rest should be < 14 per minute). Sinus arrhythmia,

Blood Pressure
- Pulsus paradoxus - fall in sys BP > 10mmHg on inspiration

Face
- Eyes: lids, pupils, anaemia, jaundice (liver mets)
- Nose: block each nostril, polyps (associated with asthma), deviated septum (nasal obstruction), nasal flaring
- Mouth: pursed lip breathing, cyanosis, URTI
- Sinuses (and ears (otoscopy) when appropriate)

Neck
- Trachea: displacement, tug (inferiorly with inspiration). Accessory muscle use. JVP
- Nodes (incl supraclavicular). Subcutaneous emphysema

Inspection
- Scars, signs of radiotherapy
- Shape: funnel or pigeon chest, kyphosis and scoliosis
- Movement: symmetrical, intercostal indrawing, paradoxical breathing of the abdomen
- Prominent veins. Pemberton’s sign: SVC obstruction – hold arms over head → facial plethora, inspiratory stridor and ↑ JVP

Palpation
- Expansion: the affected side dose NOT expand – regardless of pathology
- Apex beat: not found then → ? hyper-expanded. Maybe displaced by pathology (pneumothorax, fibrosis, etc)
- Tactile fremitus: Feel with hand while patient says 99, each side font and back
- Compress sternum to spine → pain if fracture or bone tumour

Percussion
- Stony dull, dull, normal or hyper-resonant. (Ask patient to bring elbows together)

Auscultation
- Breath sounds
  - Symmetrical? Decreased? Length of inspiration vs expiration. Bronchial?
- Additional sounds
  - Crackles (inspiratory) - time, pitch, clear with cough. Early/medium - COPD, pneumonia. Late/pan fine - fibrosis, oedema. Late/pan coarse - bronchiectasis
• Wheeze (expiratory) - pitch, single or multiple, clear with cough. Heard on inspiration implies severe narrowing.
• Pleural rub (present in inspiration and expiration)
• Vocal resonance
• NB - beware the silent chest in asthma and COPD, the patient will be CO2 retaining - vasodilation, bounding pulse, flap, confusion, headache.

Other systems
• Heart palpation and auscultation. Listen to P2: louder in pulmonary hypertension
• Check liver for mets, and for ‘ptosis’ due to hyperinflation
• Legs: DVT, oedema (pulmonary hypertension), cyanosis, clubbing of the toes, ↓capillary refill
• Temperature chart, peak flow meter/FEV. Breast examination. Introduce yourself. Wash your hands. Sit the patient upright.

102: Cardiovascular Exam

• Introduce yourself. Wash your hands. Position the patient at 45 degrees.

General appearance
• Consciousness, comfort, colour, posture
• Cyanosis, dyspnoea, cachexia, rheumatological disorders, Marfan’s, Down’s, Turner’s
• GTN spray, oxygen, IV lines etc

Nails and Hands
• Clubbing, splinter haemorrhages, cigarette staining, capillary refill, Osler's nodes, Janeway lesions, peripheral cyanosis, xanthomata, peripheral cyanosis

Pulse
• Rate and rhythm. Collapsing, pulse deficit, radial-radial delay

Blood Pressure
• Cuff width = 2/3 arm length. Bladder should not quite wrap around whole arm. A too small bladder will over estimate BP.

Face
• Eyes: anaemia, jaundice, xanthelasma
• Mitral facies/Malar flush: rose cheeks and cyanosed tongue (mitral stenosis (pulmonary HT and decreased CO) pulmonary stenosis)
• Mouth: cyanosis (lips and under tongue), mucosa for petechiae, diseased teeth (cause of infective endocarditis), palate (Marfan's)

Neck
• Carotid arteries (info about aorta and LV function): Character and volume
• JVP (info about RA and RV function): More of a sucking in than bulging out pulse, double waveform, not palpable, can be obliterated, hepatojugular reflex. Usually ↓ with inspiration. Height (from lowest point) and waveform. NB if external can be filled and then empties JVP cannot be raised as external cannot be lower than internal.
• Giant a wave - (↑atrial contraction) - TS, pulmonary HT,
• Cannon a wave - AV dissociation (do not occur every a wave)
• Large v wave - TR
• ↑ with inspiration (Kussmaul's sign) -limited RV filling - tamponade, RV infarction.

Inspection
• Scars
• Deformity: funnel chest, pigeon chest) or kyphoscoliosis
• Pacemaker
• Pulsations: apex beat and others (eg over pulmonary artery in severe pulmonary hypertension)

Palpation
• Apex beat
- Pressure (systolic) loaded - forceful and sustained - AS, HT
- Tapping - MS
- Double or triple - HOCM
- Dyskinetic - anterior MI, LV aneurysm
- Volume (diastolic) loaded - displaced, large area, uncoordinated - AR, MR, MI
- Parasternal impulse
- Tap of pulmonary valve closure (P2) over pulmonary areas in pulmonary hypertension
- Thrills

**Auscultation**
- Mitral, tricuspid, pulmonary, aortic with diaphragm and bell
- Axilla (mitral regurg) with diaphragm
- Carotid arteries for bruits and if there is a suspected aortic murmur
- Left lateral position with bell at apex and normal breathing (palpate for thrills again)
- Leaning forward on expiration with diaphragm on left sternal edge (palpate for thrills again)
- Dynamic auscultation
- S1
  - Loud - MS, ↓PR, ↑HR
  - Soft - MR, prolonged filling time (LBBB, 1st block)
- S2 (A2+P2 - ↑inspiration. Fixed splitting - ASD equalises ventricular volumes per beat. No splitting or reversed (↑expiration) in AS)
  - Loud - aortic/pulmonary HT
  - Soft - AR or calcification
- S3 (gallop - ↓ventricular compliance)
- S4 strong atrial contraction (not heard in AF)

**Peripheral Exam**
- Percuss and auscultate the back for pulmonary oedema and pleural effusion. Check for sacral oedema
- Abdomen (lying flat with one pillow): tender or enlarged liver (heart failure), pulsatile liver (tricuspid regurg), ascites (heart failure), splenomegaly (infective endocarditis)
- Legs: femoral pulse (auscultate for bruits), radiofemoral, oedema, wasting, pallor, cool. Popliteal, posterior tibial, dorsalis pedis pulses. Cyanosis and clubbing of the toes, ↓capillary refill.

**Other**
- Temperature chart (endocarditis), weight chart (oedema), fundi (endocarditis).
17: Murmur

<table>
<thead>
<tr>
<th>Timing</th>
<th>Position where murmur is best heard</th>
<th>Lesion</th>
<th>Maneuvers to accentuate murmur</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic</td>
<td>Aortic area, clavicles &amp; carotids, also apex</td>
<td>Aortic Stenosis</td>
<td>Squatting, Leg raise, Sitting in full Exp</td>
</tr>
<tr>
<td></td>
<td>Left Sternal Edge (3-4th IC spaces)</td>
<td>Pulmonary Stenosis</td>
<td>Inspiration</td>
</tr>
<tr>
<td></td>
<td>Lower LSE (4-5th IC spaces) &amp; apex</td>
<td>Atrial Septal Defect</td>
<td>Accentuated on Standing, Valsalva</td>
</tr>
<tr>
<td></td>
<td>Aortic or Pulmonary areas</td>
<td>Hypertrophic Cardiomyopathy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Atrial Septal Defect</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anaemia, Thyrotoxicosis, Pregnancy</td>
<td></td>
</tr>
<tr>
<td>Late Systolic</td>
<td>Apex</td>
<td>Mitral Valve Prolapse</td>
<td>Earlier &amp; louder with Valsalva</td>
</tr>
<tr>
<td></td>
<td>Left Sternal edge</td>
<td>Coarctation of the Aorta</td>
<td></td>
</tr>
<tr>
<td>Pansystolic</td>
<td>Apex to Axilla</td>
<td>Mitral Regurg (blowing, high freq)</td>
<td>Squatting, leg raise, hand grip</td>
</tr>
<tr>
<td></td>
<td>Left Sternal edge</td>
<td>Tricuspid Regurg (low pitched)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ventricular Septal Defect</td>
<td></td>
</tr>
<tr>
<td>Early Diastolic</td>
<td>LSE (3-4th IC spaces) &amp; Apex</td>
<td>Aortic Regurg (blowing, high pitched)</td>
<td>Sitting forward in full expiration</td>
</tr>
<tr>
<td></td>
<td>Right (or Left) Sternal edge</td>
<td>Pulmonary Regurg</td>
<td>Inspiration</td>
</tr>
<tr>
<td>Mid Diastolic</td>
<td>Apex</td>
<td>Mitral Stenosis (low freq, rumbling)</td>
<td>Left lateral position</td>
</tr>
<tr>
<td></td>
<td>Left Sternal edge</td>
<td>Austin Flint (aortic regurg)</td>
<td></td>
</tr>
<tr>
<td>Late Diastolic</td>
<td>Apex</td>
<td>Tricuspid Stenosis</td>
<td>Left lateral position</td>
</tr>
<tr>
<td>(Presystolic)</td>
<td>Left Sternal edge</td>
<td>Mitral Stenosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tricuspid Stenosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Atrial Myxoma</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Left 1st or 2nd IC space Midclav line</td>
<td>Patent Ductus Arteriosus</td>
<td>Decreased by occluding vein or by</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A V Fistula</td>
<td>lying child flat</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Aorto-pulmonary Fistula</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Venous Hum in Children</td>
<td></td>
</tr>
</tbody>
</table>

Remember R.I.L.E.
Chest Pain

- Not a topic on the list – but rather important!

History
- Differential diagnosis
  - Cardiovascular
    - Angina/MI
      - Previous heart problems?
      - Risk factors: hypertension, diabetes, smoking, hyperlipidaemia, family history
    - Aortic aneurysm or dissection (tearing pain down back)
  - Respiratory (caused by pleuritic irritation which usually causes pleuritic pain (= sharp inspiratory pain)).
    - Infection (pneumonia)
      - Rigor and fevers, feeling unwell prior to pain, cough, infectious contacts
    - Pulmonary embolus – REMEMBER THIS!
      - Calf pain or swelling
      - Risk factors: Previous PE or DVT, family history, immobility, pregnant, recent surgery, contraceptive pill, smoker, malignancy or dehydration
  - Musculoskeletal
    - Costochondritis
    - Rib fracture
    - Shingles (esp before the rash has become apparent)
  - GI related:
    - Association with eating (and type of food – eg spicy)
    - Hoarseness, difficulty swallowing, vomit taste at back of mouth
    - Use of NSAIDs
- Symptom assessment: site, onset and progression, character, radiation
- ROS: especially breathlessness, nausea, anxiety
- Risk assessment: cardiac and PE risk factors

16: Breathlessness

History
- Symptom assessment: Duration, Acute, subacute or chronic onset, rest or exertion (How much exertion?), progressive or reversible, continuous or intermittent (asthma?), Orthopnoea or PND, ankle oedema (heart failure?), exposure to allergens (dust, pets, etc)
- ROS: Chest pain, wheeze, cough (sputum or haemoptysis), fever or night sweats.
- Risk assessment: Smoker, alcohol (aspiration?), asbestos exposure, bird keeper

Differential
- Sudden
  - Foreign body
  - Pneumothorax
    - PE – remember this one!
  - Psychiatric
- Over a few hours
  - Acute asthma
  - Acute pulmonary oedema
  - Pneumonia
  - Extrinsic allergic alveolitis
- Intermittent
  - Asthma
  - Pulmonary oedema
- Over days
  - Pleural effusions
  - Carcinoma of bronchus/trachea
- Over months or years
- COPD
- Cryptogenic fibrosing alveolitis
- Occupational fibrosing lung disease

**Examination**
- CVS and respiratory
- Peak flow

**Investigations**
- ECG
- Imaging: CXR, echo (if cardiac cause)
- Blood: FBC, U&E, ABG

- Ref: Murtagh General Practice 2nd Edition, Chapter 45, pp 448 - 462

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**25: Cough**

**History**
- Symptom assessment: onset, duration, progression, character (e.g. barking quality = epiglottitis), worse at a particular time of day, mucous (colour, volume, smell), haemoptysis
- ROS: Fever, night sweats, rigors, breathlessness, PND
- Risk assessment: infectious contacts, heart conditions, occupation, pets/allergies

**General Differential**
- Respiratory
- CV (including PE)
- Psychogenic
- Drugs

**Acute causes**
- Acute bronchitis or pneumonia
- cough of recent origin
- yellow or green sputum
- may be associated with fever or other symptoms of RTI
- Severe pneumonia:
  - RR > 30, diastolic BP < 60, urea > 7.0, confusion
- Lung abscess: foul smelling dark colour sputum
- PE
- Pulmonary oedema
- Tb – weight loss, night sweats, infectious contact

**Chronic causes**
- Asthma
  - INTERMITTENT
  - Chronic cough +/- wheezing worse at night
  - Check peak flow
- ACE inhibitors/oesophageal reflux/acid irritation of lung
- Chronic, irritation, dry cough
- Bronchiectasis
  - Produces large volumes of purulent sputum
- Cancer
- Interstitial lung disease
- Tb

**Description of cough**
- Epiglottitis - barking quality
- Tracheal compression - loud and brassy
- Recurrent laryngeal nerve palsy - bovine cough
- Worse at night - asthma or heart failure
- Immediately after eating/drinking - oesophageal reflux or tracheo-oesophageal
fistula

- Ref: Murtagh General Practice 2nd Edition, Chapter 38, pp 370 - 384

93: Wheeze

**Differentiating**
- Monophonic heard in one part of the chest – partial obstruction of one airway
- Polyphonic – widespread narrowing of airways of differing calibre
- Generally, the higher the pitch of the wheeze – the narrower the airway it is coming from.

**Causes**
- Localised
  - Partial bronchial obstruction
    - Impacted foreign body (tumour, peanut, etc)
    - Impacted mucous plug
    - Extrinsic pressure (tumour, lymph node, etc)
- Generalised
  - Asthma
  - Obstructive bronchitis
  - CORD
  - Cardiac asthma (wheezing sensation such as that experienced by PND)

**Using a spacer**
- Explain use of spacer: allows more of each dose to reach the lungs
- Shake the inhaler and fit it into the opening
- Press the inhaler once
- Sit upright, chin up, lips around mouthpiece
- Breathe in slowly through mouthpiece, hold breath for 10 seconds
- Breathe out slowly
- Wait a minute between doses and repeat as necessary
- Wash spacer with warm soapy water each week, air dry

**Asthma History**
- Symptoms during an episode
- Interval symptoms: cough, waking at night, breathlessness
- Severity: Previous hospital admissions, ICU admissions, days off school, effect on lifestyle
- Triggers: smoking, pets, exercise, season
- Risk assessment: eczema, hay-fever, family history

- Ref: Murtagh General Practice 2nd Edition, Chapter 45, pg 449

80: Stridor

- Inspiratory sound due to partial obstruction of the upper airways
- May be associated with dysphagia, cyanosis or pallor, accessory muscle use, and tracheal tug.

**Causes**
- Within the lumen
  - Foreign body
  - Tumour
- Within the wall
  - Epiglottitis
    - Soft inspiratory stridor, rumbling expiratory stridor may develop
    - Soft voice
    - Toxic appearance
    - Lack of harsh cough
    - Drooling
  - Croup
    - Usually parainfluenza virus
• URTI → stridor and barking cough
• Admit if cyanosis or respiratory distress
• Treatment: humidified O2; steroids, nebulized adrenaline (depending on severity)
• Tracheitis
• Laryngomalacia
  • Floppy airway cartilage → airway collapse
  • Stridor worse on lying on back
  • Symptoms improve by 2 years but may recur during infections.
• Laryngeal oedema, trauma, tumour or spasm
• Tonsillar oedema (eg EBV)
• Neurological abnormalities (vagal or recurrent laryngeal nerve palsy)
• Extrinsic
  • Retropharyngeal abscess
  • Goitre
  • Lymphadenopathy (tumour or infection)
  • Tumour

49: Hoarseness

Common causes
• Viral laryngitis
• Trauma (shouting, coughing, vomiting, inhaling noxious fumes)
• Nodules and polyps
• Intubation
• Gastroesophageal reflux

Exclude
• Imminent airway obstruction - croup, epiglottitis, allergy
• Malignancy
• Endocrine causes - myxoedema, goitre, acromegaly, Addison's
• Vocal cord paralysis - surgery, lung or neck tumour, central lesion
• Granulomatous disorders - Tb, syphilis, sarcoid, Wegener's
• Severe infections - laryngeal abscess, diphtheria

Investigations
• Laryngoscopy must be performed if it is chronic (> 3weeks) or a serious condition is suspected

Management
• Acute
  • Treat according to the cause
  • Vocal rest or minimal usage at quiet conversational level
  • Avoid irritants - smoke, alcohol
  • Consider steam inhalations and cough suppressants
• Chronic
  • Must establish the diagnosis. ENT referral likely to be necessary

Ref: Murtagh General Practice 2nd Edition, Chapter 123, pg. 1221

61: Nasal Obstruction

Causes in children
• Large adenoids
• Rhinitis
• Choanal atresia
• Postnasal space tumours
• Foreign body
Causes in adults
- Deviated nasal septum
- Rhinitis
- Polyps
- Sinusitis
- Granuloma (Tb, syphilis, Wegener’s)
- Drugs (topical vasoconstrictors, tricyclics, alpha blockers)

History
- Variability of symptoms
- Pattern of obstruction
- Effects on eating, speech and sleep (snoring)

Examination
- Assess any nostril deflection
- Is either nostril completely blocked (hold a mirror under each nostril: does it steam up?)
- Examine post-nasal space with a mirror

Most causes also present with at least some degree of nasal obstruction.

Infection (rhinitis or sinusitis) – mucosa looks red and swollen.
- Bacterial - purulent
- Viral – may also have a purulent discharge
- Allergy - bilateral and variable clear discharge. Mucosa looks pale and swollen.
  - Seasonal
  - Perennial
- Vasomotor rhinitis – similar presentation as allergic rhinitis but generally does not respond to antihistamines.
- Chemical
  - Excessive use of decongestants (rhinitis medicamentosa).
  - Smoking
  - Occupational exposure to specific irritants
- Other
  - CSF - due to fracture or tumour infiltration.
  - Foreign body - organic material usually presents early with unilateral purulent discharge.
  - Polyps - in children generally associated with CF. In adults associated with allergy and chronic ethmoid sinusitis.

Ref: OHCS 5th Ed. p548.

77: Sore Throat

The key questions to answer are:
- Is this due to infection or some other cause?
- If this is an infection should you give antibiotics or not?

Differentiating between a viral and bacterial cause (if all four of these features are present then the cause is likely to be bacterial)
- No cough
- Fever > 38 C
- Tender cervical lymphadenopathy
- Purulent tonsillar exudate

If the cause is likely to be bacterial or the patient is >/= 4yrs and is from a high-risk group eg South Auckland, Maori or Pacific Islander) then treat with penicillin V for 10 days. If allergic give erythromycin.
- Candida - Candida of the throat should be considered due to HIV until proven otherwise.
- EBV - consider in a teenager/young adult with posterior lymphadenopathy. The tonsillar exudate may be extensive. Do monospot /Paul-Bunnell or EBV serology.
Serious conditions to consider

- Cancer
- Angina/MI
- Peritonsillar (quinsy) or pharyngeal abscess – requires surgical drainage
- Acute epiglottitis
- Diphtheria
- Blood dyscrasias can cause a sore throat – take a drug history.

Investigations

- Swab if persistent (benefits of always taking a swab are debatable)
- EBV tests
- Endoscopy or pH probe if reflux oesophagitis is suspected
- FBC can show atypical lymphocytes, rule out anaemia, reveal blood dyscrasias

46: Haemoptysis

- Do not confuse with haematemesis. Haemoptysis is frothy, alkaline and bright red. It may cause melaena if enough blood is swallowed. The bleeding itself rarely needs treatment but the underlying cause needs to be determined. Take a careful history and do a full CVS and respiratory exam.

Respiratory causes

- Trauma - foreign body, post-intubation
- Infection - acute bronchitis, pneumonia, abscess, bronchiectasis, Tb, fungi
- Neoplasia - primary or secondary
- Vascular - lung infarction, vasculitis (Wegener's, RA, SLE), AV fistula, malformations
- Parenchymal - fibrosis, sarcoidosis, haemosiderosis, Goodpasture's syndrome, CF

Cardiovascular causes (pulmonary hypertension)

- Pulmonary oedema
- Mitral stenosis
- Aortic aneurysm
- Eisenmenger's syndrome - congenital heart defect which is originally a L to R shunt which causes pulmonary hypertension and then shunt reversal and cyanosis
- PE

70: Palpitations

History

- Can you tap out the rhythm?
- Do the palpitations start suddenly?
- How long do they last?
- What do you think brings them on?
  - Are they related to stress, worry, or excitement?
  - How much tea, coffee and Coke do you drink?
  - Do you smoke cigarettes, and how many?
- What symptoms do you notice during an attack?
  - Do you have chest pain or breathlessness during the attack?
  - Do you feel dizzy or faint during the attack?
- What medications do you take?
  - Any alcohol or social drugs?
  - Any medications?
- Ever had rheumatic fever?
- Loss weight recently or do you sweat a lot?

Differentiating causes

- Irregular, fast palpitations: Paroxysmal AF, or flutter with variable block
- Dropped or missed beats: Atrial or ventricular ectopics
- Regular pounding: Anxiety
- **Slow palpitations:**  *Drugs*
- Chest pain:  *Myocardial ischaemia or aortic stenosis*
- Breathlessness:
  - Anxiety with hyperventilation
  - Mitral stenosis
  - Cardiac failure
- Dizziness or syncope
  - Sick sinus syndrome
  - Complete heart block
  - Aortic stenosis
  - Cerebrovascular disease
- Tachycardia:
  - Anxiety
  - Catecholamine effect: hyperthyroidism, phaeochromocytoma
- Arrhythmia:
  - Cardiac
  - Metabolic: K, Ca
  - Drugs: digoxin

**Examination**
- Best if can do it during an attack
- Pulse rate, rhythm and character
- Evidence of fever and infection
- Evidence of anxiety or depressive illness
- Hyperventilate into bag to see if triggers palpitations
- Evidence of underlying:
  - Anaemia
  - Thyroid disease
  - Alcohol abuse
  - Cardiac disease

**Investigations**
- Blood: Hb and film; thyroid function tests; U&E, Ca
- CXR, ECG, ambulatory 24 ECG monitoring
- Ref: Murtagh General Practice 2\textsuperscript{nd} Edition, Chapter 64, pp 666 - 678

**19: Claudication**

**Differential**
- Vascular claudication
- Spinal claudication
- Trauma/arthritis
- Varicose veins

**History**
- Description of Pain: in addition to normal pain characteristics
  - Walking distance
  - Same distance for consistent symptoms
  - Relieved by rest – after how long
  - Pain at night – indicates need for immediate surgical assessment
- Associated changes:
  - Skin changes, ulceration
  - Fatigue, tiredness, malaise
- Enquiry about differentials (think vascular, neurological, arthritic, trauma):
  - Trauma to legs
  - Sensory changes
  - Weakness
- Stiffness of joints
- Swelling of legs or joints

Risk factors for claudication:
- Hypertension
- Hyperlipidaemia
- Diabetes mellitus
- Smoking
- Previous history of IHD, CVA
- Family History

- Medications
- Social History: impact on lifestyle and employment

Examination
- Inspection:
  - Colour
  - Presence of hairs
  - Varicose veins, ulcers
  - Shiny skin
  - Oedema
  - Swelling
  - Wasting
  - Toenails
- Ask patient about pain

Palpation:
- Compare size, tenderness and warmth of calves
- Temperature of feet
- Capillary return
- Sensation
- Oedema

Peripheral pulses:
- Femoral: palpate and auscultate for bruits
- Pulpate popliteal, posterior tibial, dorsalis pedis

Buerger’s test:
- Elevate to 45° → pallor is rapid if poor arterial supply
- Then hang over bed → cyanotic if arterial supply is impaired

Varicose Veins: Trendelenburg test: lie down, empty great saphenous vein, occlude by proximal pressure, stand up, if it refills the perforating veins are stuffed (blood should flow from outside to inside)

Vascular Claudication
- Due to atherosclerosis involving the aorta, iliac and/or any other peripheral vessels.
- Usually occurs over the age of 50, chiefly in men who are smokers

Symptoms:
- Pain felt in the leg or buttock on exercise (intermittent claudication). The calves are the most common site. After walking a fairly consistent distance (the claudication distance), cramping pain forces the patient to stop. Exercise can be resumed after rest.
- Ulceration, gangrene, and pain at rest, eg burning pain at night relieved by hanging legs over the side of the bed are 3 cardinal features of critical ischaemia.
- Buttock claudication and impotence imply Leriche’s syndrome
- Complications of varicose veins: ulcers, phlebitis, DVT, bleeding

Signs: (May be few with intermittent claudication):
- Absent pulses
- ↓Capillary refill
- Cold white leg/s
- Absent skin hairs
- Atrophic skin
- Punched out ulcers
• Postural colour change
• Bruits: femoral, popliteal

Tests:
• FBC (anaemia, infection), U&Es, ESR/CRP, Lipids, Syphilis serology, glucose, ECG.
• **Ankle-brachial pressure index** (Doppler): Normal = 1, Intermittent claudication = 0.9 – 0.6, Rest pain = 0.6 – 0.3, Impending gangrene <0.3 or ankle systolic pressure <50mmHg
• Arteriography - to assess extent and location of stenosis.

Management:
• Stop smoking, reduce weight, increase exercise (>3 30 min walks/week), treat diabetes, hypertension (avoid B blockers), and hyperlipidaemia. Vasodilators rarely help
• Percutaneous transluminal angioplasty
• Arterial reconstruction
• Sympathectomy
• Amputation – to relieve intractable pain or prevent sepsis from gangrenous leg.

**Spinal claudication**
• This is due to spinal stenosis – narrowing of the spinal canal (may be congenital eg Achondroplasia, or acquired eg OA of the facet joints).
• As the patient walks the nerve roots become hyperaemic and swell. This causes buttock and lower limb pain with associated numbness. This resolves with rest.
• As opposed to intermittent vascular claudication, the symptoms of patients with spinal claudication may improve upon walking up hill as this forces the patient to lean forward slightly, flexing the spine, which increases the AP diameter of the spinal canal.

Ref: Murtagh General Practice 2nd Edition, Chapter 60, pp 608 - 625

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**53: Ischaemic Foot**

**Acute**
• The six p’s: pain, pallor, pulseless, paralysed, paraesthesia (numb), perishing cold. Also look for collapsed superficial veins and no capillary return and pain on passive ankle movement
• Causes: embolus (peripheral arteries), thrombosis (major artery or popliteal aneurysm, traumatic contusion eg post-arterial puncture
• Management: Reversible if treated in 4 hrs. Irreversible after 6 hrs (fixed mottling of the skin). IV heparin 5000U and embolectomy or bypass. Lifetime anticoagulation with warfarin

**Chronic**
• Intermittent claudication - the level of obstruction is indicated by which muscle belly is affected.
• Pain at rest implies immediate threat to viability
• Same signs as in acute but less severe. Look for oedema. Listen for bruits in abdomen and femoral areas esp after exercise
• Postural colour changes (Buerger’s test) - raise both legs to 60 degrees for 1min then hang both legs over bed and watch colour return. Positive test is pallor on elevation, slow colour return (.10sec) and development of dusky redness
• Causes - as above. Also popliteal entrapment in those < 40yrs and vasculitis
• Tests: FBC, U&E, ESR/CRP, lipids, syphilis, glucose, ECG

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**90: Varicose Veins**

**Causes**
• Incompetent
• Venous hypertension from prolonged standing
• Occlusion by
  • Foetus
  • Fibroids
  • Ovarian tumour
  • Previous DVT
Risk factors
- Female
- Family history
- Pregnancy
- Multiparity
- Age
- Occupation
- Low fibre diet

Complications
- Pain
- Superficial thrombophlebitis
- Skin “eczema” (10%)
- Skin ulceration (20%)
- Bleeding
- Calcification
- Marjolin’s ulcer (SCC)

Examination
- Venous groin cough impulse (helps determine long saphenous vein incompetence)
- Trendelenburg test
- Short saphenous vein incompetence test
- Perthes test
- Refer to OHCM pg 162 or Murtaugh pg 621 for more about these tests

Management
- Prevention
  - Maintain ideal weight
  - Eat a high fibre diet
  - Test and wear supportive stockings if at risk (pregnancy, a standing occupation)
- Treatment
  - Keeps off legs as much as possible
  - Sit with legs on a foot stool
  - Use supportive stockings or tights
  - Avoid scratching itching skin over veins
- Compression sclerotherapy
  - Use a small volume of sclerosant (eg sodium tetradactyl sulphate)
  - It is ideal for smaller isolated veins particularly below the knee joint
- Surgical ligation and stripping
  - This is the best treatment when a clear association exists between symptoms and obvious varicose veins (ie long saphenous vein incompetence)
  - Remove obvious varicosities and ligate perforators.


Differentials
- Venous insufficiency:
  - History/presentation: later life, usually lower leg above ankles. Assoc with venous eczema, brown pigmentation, varicose veins, lipodermatosclerosis, atrophie blanche.
  - Investigations: duplex ultrasound
  - Treatment: high-compression bandaging with leg elevation, keep ulcer clean and moist (dress appropriately), analgesia, diuretics may help with oedema, surgery beneficial in some cases. Long term - lifelong support stockings.
- Arterial insufficiency:
  - History: claudication, hypertension, angina, smoking.
• Presentation: punched-out, painful ulcers higher up on the leg or on the feet. Leg may be cold/pallor, absent peripheral pulses, arterial bruits, loss of hair.
• Investigations: doppler ultrasound
• Treatment: keep ulcer clean and covered, analgesia, vascular reconstruction if appropriate
• Pressure sores (decubitus ulcers):
  • History: elderly, immobile, unconscious or paralysed. Majority occur in hospital. Factors that increase risk e.g. diabetes, RA, peripheral vascular disease. Precipitating factors e.g. surgery, urinary/faecal incontinance.
  • Presentation: early sign is red/blue discolouration of skin, rapidly → ulcer (1-2hrs), most commonly over heel/sacrum.
  • Treatment: immediate attention on noticing early signs. Regular turning, use of equipment to decrease pressure (pillows, roto cushions, fleece, special mattresses), treat general condition, keep ulcer clean and moist, pain relief, plastic surgery.
• Neuropathic:
  • History: diabetes, leprosy
  • Presentation: tend to be seen over pressure areas of the feet (metatarsal heads) owing to repeated trauma
  • Treatment: keep ulcer clean, remove pressure/trauma from affected area, diabetics regularly self check feet, correct fitting shoes
• Neoplastic e.g. squamous or basal cell carcinoma (rodent ulcer)
• Vasculitis e.g. RA, SLE
• Infection e.g. ecthyma, T, deep mycoses, tropical ulcer, syphilis, yaws
• Haematological e.g. sickle cell disease, spherocytosis
• Other e.g. trauma, necrobiosis lipoidica
• Ref: Kumar and Clark p.1185-186

50: Hypertension

• See No 95 for definitions, page 40
• 95% of hypertension is ‘essential’ but alcohol and obesity may play an important part
• 5% secondary: renal, endocrine, coarctation, drugs
• Several measurements should be made at each visit and unless extremely high BP is found, a minimum of 3 visits made before initial decisions on therapy are made.

Examination
• BP lying and standing:
  • Essential hypertension → ↑diastolic on standing
  • Secondary hypertension → ↓diastolic on standing
• Radio-femoral delay (⇒ coarctation of the aorta)
• Fundi for hypertensive retinopathy:
  • Grade 1: silver wiring (prominent central white line in arteries)
  • Grade 2: 1 + arteriovenous nicking
  • Grade 3: 3 + haemorrhage (flame) and exudates (soft or hard)
  • Grade 4: 3 + papilledema
• CVS
• Abdominal:
  • Renal/adrenal masses
  • Bruits
  • AAA
• CNS: Signs of previous CVAs
• Urinalysis: for renal disease

Treatment
• Lifestyle factors
  • Stop smoking - reduces arteriopathy, not BP
  • Decreased intake of animal fat - same as smoking cessation
  • If obese:
    • Explain about optimum weight

2003 OSCE Handbook
• Find good diet
• Encourage ‘sensible’ exercise
• Cut alcohol intake to < 2 units/day
• Avoid heavy salting of food
• Increase dietary Potassium (eg 5 pieces of fruit/day)
• Reduce stress

• The decision to treat hypertension with drugs is not made solely on a blood pressure value:
• BP between 150-170/90-100 should only be considered for treatment if their risk of cardiovascular disease in 10yrs is 20% or more. *(Use tables to assess this risk)*
• BP of 170/100 should be considered for treatment regardless of other risk factors for cardiovascular disease.

• Drug Treatment
  • 1st line agents:
    • Thiazide Diuretic - Contraindications: Gout, Diabetes, Hypokalaemia
      SE: ↑lipids, hyponatraemia, impotence
    • β Blocker - Contraindications: Airways disease, Heart failure, Vascular disease
      SE: Loss of energy, cold peripherals, nightmares
  • 2nd line
    • ACE inhibitors - SE: Steep fall in BP, cough
    • Angiotensin receptor blockers eg Losartan, no cough
    • Ca antagonists -SE: peripheral oedema, flushing, headache, decreased cardiac contraction
    • Alpha blockers - SE: postural hypotension, diarrhoea (note - improve lipids)
    • Direct-acting vasodilators eg Hydralazine or Minoxidil

*Co-existing Disease*
• Diabetes ACE inhibitor, Ca channel blocker
• Heart failure ACE inhibitor, Thiazide diuretic
• Angina β blocker, Ca channel blocker
• Hyperlipidaemia Alpha 1 blockers
• CORD ACE inhibitor, Ca channel blocker

*Management in pregnancy*
• Mild: monitor and reduce stress
• Intermediate: 1st labetalol, 2nd methyldopa, 3rd nifedipine
• Severe: aspirin, MgSO4 (prophylaxis and treatment of seizures), hospitalise, IV hydralazine, delivery

95: Measure Blood Pressure

*Measuring blood pressure points to remember*
• Patient seated and relaxed > 5mins
• Arm relaxed and supported at heart level
• Mercury Sphygmomanometer at eye level
• Cuff:
  • Width - 2/3 length of humerus
  • Bladder should almost completely encircle arm
  • Centre of bladder over artery
• Release pressure at 2-3 mmHg/heart beat
• Systolic = Kortokoff 1
• Diastolic = Kortokoff 5
• Pressure measured to nearest 2 mmHg
WHO definitions of Blood Pressure:

<table>
<thead>
<tr>
<th>Category</th>
<th>Systolic</th>
<th>Diastolic</th>
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<tbody>
<tr>
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<td>&lt;85</td>
</tr>
<tr>
<td>High Normal</td>
<td>130-139</td>
<td>85-90</td>
</tr>
<tr>
<td>Hypertension:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>140-159</td>
<td>90-99</td>
</tr>
<tr>
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<td>160-179</td>
<td>100-109</td>
</tr>
<tr>
<td>Severe</td>
<td>180-209</td>
<td>110-119</td>
</tr>
<tr>
<td>Very severe</td>
<td>&gt;210</td>
<td>&gt;120</td>
</tr>
</tbody>
</table>

Postural Hypotension

- A fall of at least: 15mmHg Systolic
  10mmHg Diastolic

Obtaining an ECG

- Have patient lying still. Place electrodes on soft tissue not on bone.
- Limb leads - right arm, left arm left leg

Interpretation

- Always ask ‘Is there a previous one for comparison’
- Name and date
- Calibration: 1cm high (= 1mV =2 big squares) with flat top.
- Rate and rhythm
  - 300/No big squares (each=0.2s) 2.5=120, 3=100, 3.5=86, 4=75, 4.5=67, 5=60, 6=50, 7=43
  - Rhythm - use marks on card method. AF, A flutter, VF, ectopics,
  - Initiation of depolarisation – sinus, nodal/junctional (absent P waves or P waves just before or in normal width QRS), ventricular (QRS>0.12)
  - 1st block -normal, MI, acute RF
  - 2nd block - MI, drugs (digoxin, B blocker, verapamil) - Mobitz 1 (Wenckebach) = progressive, Mobitz 2 = 2:1 = every 2nd p wave works.
  - 3rd block - more often due to fibrosis of bundle of His than ischaemia
- Axis
  - Left deviation (usually = conduction defect in anterior fascicle (L anterior hemiblock) but may be due to hypertrophy). II is negative, I is positive and aVF is negative
  - Right deviation (= L posterior hemiblock or R hypertrophy). I is negative, III more positive than usual.
- P wave (best seen in II and V2)
  - Shape:
    - LA hypertrophy = bifid, > 0.12s in II, V1 and V2 deeply negative.
    - RA hypertrophy = tall (> 2.5mm), P wave in II, V1 is positive.
    - PR interval - normal = 3-5 squares (0.12-0.21s) Increased in RF or normal
- QRS
  - Width - if > 0.12ms = 3 small squares implies conduction defect.
    - LBBB: always means heart disease (usually L) - aortic stenosis, IHD, MI. 'M' (RSR pattern) in V6
    - RBBB: pattern with normal QRS length can be seen in healthy people. Long QRS may indicate IHD, fibrosis, pulmonary HT, PE or congenital heart disease (eg ASD). 'M' (RSR pattern) in V1 and S waves in I.
  - QRS height
    - LV hypertrophy - S in V1 or 2 + R in V5 or 6 => 35mm
    - RV hypertrophy - SI, QIII, TIII, V1 positive, V6 has deep S wave. Usually also R axis deviation: PE, pulmonary hypertension
- Q waves (may be normal if in III, aVF and V1 if < 2mm): (> 1square wide and > 2mm deep). Diff dx = WPW (↓PR, slurred QRS upstroke, inverted T in ant chest leads), post-infarction (T wave is upright). Occur > 24hrs after MI
- QT interval (QRS start to end of T). QT cycle length = (Q-T)/(square root of (R-R)) n=0.35-0.43. Decreased with hypercalcaemia, increased with hypocalcaemia.

- ST segment and T waves (look in all leads)
  - Planar elevation (> 1mm) implies infarction
  - Planar depression (> 0.5) implies ischaemia
- T wave: Usually abnormal if negative in I, II, V4-6. Peaked in hyperkalaemia, flattened in hypokalaemia
- U wave: may be normal or due to hypokalaemia

Strain pattern - ST depression and inverted T waves in V1-3on R or V4-6 on L.
Posterior MI: Large R waves in V1 + V2 + ST depression
Digoxin effect - ST depression, T wave inversion in V5-V6 = reverse tick
Digoxin toxicity - any arrhythmia esp ventricular ectopics and nodal bradycardia
PE - deep S in I, Q waves in III, inverted or absent T in III
Pericarditis - saddle ST elevation in all leads. Unlike MI there are no reciprocal changes.
Rare.
Hyperkalaemia - Peaked T, ST depression, increase P-R/absent P and wide QRS
Hypokalaemia - flattened T, U waves, ventricular bigeminy (extrasystole with each sinus beat)

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98: Peak flow Measurement

PEFR
- The maximum rate of expiration maintained for 10 milliseconds, that occurs within about the first second of forced expiration.
- PEFR is closely related to FEV1
- Variation in PEFR of 15% is considered significant – non-asthmatics usually less than 5% variation.
- Best times to take readings - on rising, afternoon, before bed, during any nocturnal disturbance. If patients on an inhaled beta agonist - before and after inhalation.
- Although mean values are useful, within patient results are the most instructive - they show trends over time (often helpful to graph PEFRs on new/unstable asthmatics when trying to sort out triggers)

PEFR depends on
- Age
- Sex
- Height
- Weight
- Posture - standing gives the best PEFRs
- Patient effort - most common source of fallibility in lung function testing

Method
- Explain why measuring PEFR is useful
- Ensure that the patient is standing
- Check that the pointer of the PF meter is on 0
- Instruct the patient to take as deep a breath as is possible
- Put the mouth piece (new one for each patient) between the lips, which must be sealed tightly around it
- Holding the meter horizontally, with fingers not obstructing pointers track, the patient should blow “huff” out as hard as is possible
- Read the position of the pointer
- Take the best of 3 readings
Note: Some subjects will prepare themselves for the test by taking repeated deep breaths, this often aggravates the situation in unstable asthmatics - by the time the result is obtained, the PEFR may be lower than when the test procedure was started.

Sources of error
- Failure for the patient to take a full breath
- Failure to seal lips
- Patients concern over mouthpiece, loose false teeth etc
- Escape of air down the patients nose
- Faulty technique on the part of the patient
- Poor effort
- Protracted, low-force blow
- ‘Cheek blowing’ or ‘spitting air’ which may artificially raise the poor results of those with extensive pulmonary disease
- Rarely the piston may stick and yield no result, or it may suddenly release and shoot past the true value

Summary
- Name, Date, Projection
- Correct orientation
- Exposure - should be able to just see intervertebral spaces through the heart
- Centering - medial ends of clavicles against spinous processes
- Inspiration - count down ribs - want to be able to see 10th post rib
- Soft tissue and Bones
- Mediastinum
- Hila
- Heart
- Diaphragm
- Lung fields
- Review

Frontal Film
- Name, Date and projection

Centring
- Medial ends of clavicles should be equidistant from midline spinous processes

Exposure & Quality
- Look for:
  - Spine should just be seen through mediastinum
  - Film needs to be exposed on full inspiration (about 6 anterior ribs)

Correct Orientation
- Do not miss dextrocardia (heart apex on left & stomach bubble on right)

Systematic Film Interpretation
- My way of remembering it is: “My Hard Hat Does Look Bloody Ridiculous” (stupid I know! 😄)
- Mediastinum:
  - Trachea central in midline
    - May be deviated by goitre or mediastinal mass
    - May be deviated, with mediastinum, by large pleural effusion, tension pneumothorax, or pulmonary collapse
  - Whether is it enlarged or not
- Hila:
  - Left higher than right
  - More prominent if:
    - Patient rotated
- Lymphadenopathy
- Large pulmonary artery
- Mass/tumour

- **Heart**
  - Borders
    - Right heart border: Right atrium
    - Left heart border: Left ventricle
    - Enlarged LA: Double heart border
    - Enlarged LV: Tip apex outwards and downwards
    - Enlarged RV: Tip apex upwards
  - Aortico-pulmonary window: should be convex
  - Cardiothoracic diameter should be <50%
  - Valve calcification (best seen on lateral)

- **Diaphragm**
  - Right higher than left
  - Make sure costophrenic angles are sharp
  - If hemidiaphragms low and flat, chronic lung disease may be present
  - Hemidiaphragm raised:
    - Loss lung volume
    - Phrenic nerve palsy

- **Lung fields:**
  - Nodular shadows:
    - Septic emboli
    - Viral pneumonia
    - Granuloma
    - Malignancy
    - Pneumoconiosis (except asbestos – linear shadowing)
  - Reticular shadows:
    - Fibrosis of chronic disease
    - Sarcoïd, silicosis, asbestosis
    - Early LVF
    - Malignancy
    - Extrinsic allergic alveolitis
    - Autoimmune eg SLE, PAN
  - Alveolar Shadows
    - Pulmonary oedema, infection, or haemorrhage
    - Smoke inhalation
    - ARDS
    - Drugs (heroin)
    - DIC
    - Fat emboli

- **Bones and soft tissue:**
  - Breast shadows (mastectomy may explain mets)
  - Soft tissue gas (eg in pneumothorax)
  - Rib fractures or notching

- **Review**
  - Apices: Pancoast’s tumour or TB
  - Retrocardiac region: Collapsed left lower lobe may reveal itself as a triangular opacity behind the heart shadow.
  - Pneumothorax: Difference between translucency of two lungs
  - Costophrenic angles

**Lateral Film**
- Retrosternal and retrocardiac angles usually same density
- Vertebræ become less opaque lower down the spine, unless there is pulmonary or pleural disease
- Posterior costophrenic angle is sharp unless fluid or adjacent consolidation
• Hemidiaphragms well defined unless there is pleural or pulmonary disease

133: Pulmonary Function Report

• Note the patients age, sex, height and ethnicity

![Pulmonary Function Diagram]

• VC > FVC implies airway collapse causing air trapping.
• Reversibility with bronchodilator implies > 10% improvement

Alternative approach using FIF50 and FEF50 values

• FIF50/FEF50
  - >1 = obstructive
  - <1 = restrictive
  - =1 = normal
• If the FEV1 is low:
  - A low FIF50 implies airway narrowing
  - A normal FIF50 implies airway collapse

155: Lung V/Q Scan

Indications
• Clinical suspicion of PE

Complications
• Anaphylaxis?

Techniques
• Inhale radioactive xenon gas
• Inject with $^{99m}$Tc
• Limitations
  - A matched defect may arise with a pulmonary embolus with causes an infarct, or from emphysematous bullae
  - This test therefore must be interpreted in the context of history, examination, and other investigations

High probability scan (>90%)
• > Or equal to 2 big (>75% of a segment) segmental perfusion deficit OR
• > Or equal to 4 involving >26% of a segment OR
• One big & > or equal to 3 moderate deficits

Low probability (<15%)
• Any number of matched ventilation-perfusion defects (whatever their size) OR
• Small perfusion defects (<25% of a segment) OR
• Non-segmental deficits
Intermediate probability (about 50%)

- Lies between high a low probability

CPR

- **Stop** and take a breath (“the first pulse to check in any emergency is your own”)
- **Safety** (insure your own and others)
- **Responsiveness** (check by shake gently on shoulder and calling their name)
- **Help** (send someone else)
- **Airway** (open with head-tilt chin lift or jaw thrust if risk of neck injury)
- **Breathing/circulation**: look, listen and feel for no longer than 20 seconds
- Precordial thump (if arrest within last 90 secs)
- Go for help if there was no one to send for help
- 2 Effective Breaths – up to 5 attempts (In children give 5 breaths)
- Chest compression/ventilation: 2 breaths per 15 compressions even with two rescuers. (In children give 1 breath per 15 compressions)
- Check carotids every 4 cycles

Defibrillation

- ASAP: attach monitor Defibrillator and assess rhythm
- VF or VT or AED ‘shock advised’:
  - Defibrillate immediately: 200, 200, 360J (3 * 360J thereafter). (In children: 2, 2, 4J/kg)
  - Adrenaline every 3 minutes
  - 1 minute CPR
- Not VF or VT or No Shock Advised
  - If bradycardia or asystole:
    - Adrenaline and atropine
    - 3 minutes CPR
  - 3rd degree heart block: no treatment required
- Reassess rhythm or circulation

Defibrillation/EEG: source, settings and electrodes

Adjunct Priorities:

- Defibrillation
- Advanced airway/ventilation adjuncts
- Oxygen
- IV access

Advanced Adjuncts:

- Endotracheal Intubation
- Laryngeal Mask airway
- Adrenaline: 1mg/3 minutes
- VF/VT: Defibrillation, Lignocaine 1mg/kg after 3 loops
- Asystole: atropine 3 mg
- Consider: pacemaker, buffers

Check:

- Defibrillator/EEG: source, settings and electrodes
- Endotracheal/IV placement

Consider and Correct:

- Hypoxia (O2)
- Hypovolaemia (colloids, saline)
- Hyper/hypokalaemia (Ca/K)
- Hyper/hypoglycaemia (insulin & K/glucose)
- Hyper/hypothermia (cool/warm)
- Tension Pneumothorax (chest drain)
- Tamponade (pericardiocentesis)
- Toxicity
- Thromboembolus

History

- Previous lumps
- LMP
- Change in size in relation to period
- Any nipple discharge
- Redness
- Heat or pain
- Medications (eg HRT)
- Family history
- Systemic symptoms

Examination

- Inspect

15: Breast lump
• Compare breasts for: nipples, veins (unilateral suggest Ca), colour, dimpling.
• Raise arms above head (tethering?), hands pressed against hips (deep attachment?)
• Palpate
  • Four quadrants + nipple + axilla + supraclavicular nodes
  • Cancer = FISH (Fixed, Irregular, Single, Hard)

Management
• All solid lumps need histological or cytological assessment - FNA, "Tru-cut" (core biopsy) or open (incision) biopsy. Ultrasound helpful for cysts in those < 35. Mammography can be diagnostic in those > 50.

Causes
• Premenopausal
  • Mammary hyperplasia
  • Cysts (fibrocystic change)
  • Fibroadenoma
  • Breast abscess/periareolar inflammation
  • Hormonal changes
  • Malignancy
• Postmenopausal
  • Any new discrete mass = cancer until proven otherwise
  • Inflammatory lesion
  • Cysts unlikely
  • > 50 and on HRT
  • Any new mass treat with suspicion
  • Cysts and hormonal changes may occur

Causes by pathology
• Physiological: OCs or just before period
• Inflammatory
  • Mastitis, abscess
  • Fat necrosis
  • Mammary duct ectasia
• Fibrocystic change
• Tumour
  • Fibroadenoma
  • Cancer (in situ or invasive)
  • “Non-breast” lumps – lipoma, sebaceous cyst

Nipple disease
• Skin disease differential
  • Eczema
  • Duct ectasia
  • Infection
  • Paget’s
  • Intraductal papilloma
• Nipple discharge history
  • Serous or bloody?
  • One nipple or bilateral?
  • From one duct or many?
  • Associated lymph?
• Nipple discharge differential
  • Intraductal papilloma (most common)
  • Duct ectasia
  • Cancer (discharge without a mass is very unlikely to be cancer)
  • Physiological (may be blood stained pregnancy)

• Ref: OHCM p138; General Practice, Murtagh p824
Neuro-sensory

103: Cranial Nerve Exam

1: Olfactory
- Not if doing general screen. Close eyes. Check each nostril patent then test.
- Poor smell common (smoking, allergies, ageing). Also in frontal lobe tumour, trauma, Alzheimer’s, Parkinson’s, MS, chemotherapy, Kallman’s syndrome.

2: Ophthalmic nerve
- Check if they normally wear glasses.
- Acuity (test each eye separately and use pinhole if they’ve forgotten their glasses).
- Visual fields (red pinhead test good for vague hemianopia)
- Fundoscopy

3, 4 and 6: Occulomotor, Trochlear and Abducens
- Look for ptosis
- Pupils: shape and symmetry, corneal light reflex, swinging light test and accommodation
- Eye movement: draw an H > 45cm away, ask for report of diplopia, watch for one eye lagging or nystagmus. If diplopia found, find field where it is maximal. Hold finger perpendicular to line of movement (easier to follow) and only do test once.
- Cover test: look at target, cover one eye, does other eye move? Shows which is fixing eye.
- May wish to test saccadic movement (overshoot and come back → ipsilateral cerebellar lesion, MLF or contralateral parietal lobe)

5: Trigeminal
- Light touch and pinprick in all 3 divisions on both sides.
- Corneal reflex (early sign of lesion)
- Motor: temporal wasting, jaw deviation and opening in midline (tests pterygoids). Bit stick on each side separately and should not be able to pull out and/or clench jaw and palpate masseters. Jaw jerk only if indicated

7: Facial
- Wrinkle forehead, close eyes tightly, ‘show your teeth’ (not smile), puff up cheeks.
- Taste anterior 2/3 tongue - not tested routinely

8: Vestibulocochlear
- Whispered voice at arms length, with patient’s eyes close and distracting opposite.
- Rinne test and Weber tests. (Otoscopy if hearing is decreased)
- Test of vestibular function not routine unless gait or balance complaint. Romberg test - tests vestibular and position sense.

9 and 10: Glossopharyngeal and Vagus
- Uvula in midline and moves up symmetrically
- Check swallowing and speech (for hoarseness). Say ”eeee” to test vocal chord apposition.
- Not done - Gag reflex/pharyngeal sensation. Unilateral absence abnormal, bilateral absence may be normal.

12: Hypoglossal
- Observe in mouth: fasciculation (?motor neuron disease), wasting
- Protrude tongue: deviates to weak side. Move tongue rapidly from side to side or say 'la la la'

11: Accessory
- Observe sternomastoid and trapezius at rest for wasting, fasciculation, or dystonia.
- Shrug shoulders: observe then test strength
- Look sideways, try to return head against resistance (use fist against lateral forehead and feel contralateral side to head turn)
- Always test neck extension if diffuse muscle weakness – if abnormal indicates lesion above C1/C2
108: Peripheral Nervous System Examination

- **NB - ask if the patient has any pain/sore joints before beginning the examination**
- **Sit patient on side of bed. Do cranial nerve exam first.**

**Upper limb motor exam**

**Observe**
- Wasting (1st dorsal interosseus and abductor pollicis), fasciculations, abnormal movements.
- Hands outstretched with eyes closed and palms up (check for drift – non-specific test).
- Tone: wrist, supination, elbow - slow (rigidity) and rapid (spasticity) fall range movements

**Power**
- Shoulder abduction and flexion (deltoid, C5-6 axillary nerve).
- Shoulder adduction and extension (C6, 7, 8)
- Elbow flexion (biceps, brachialis, C5-6, musculocutaneous nerve)
- Elbow extension (triceps, C7-8, radial)
- Wrist extension (extensor carpi ulnaris and radialis, C6-7, radial nerve)
- Wrist flexion (flexor carpi ulnaris and radialis, C6-7, ulnar and median nerves)
- Finger extension (extensor digitorum, C7-8).
- Finger flexion (flexor digitorum, C7-8).
- Finger abduction (ulnar nerve, T1, dorsal interosseus). Look for thenar wasting
- Thumb abduction (median nerve, T1, abductor pollicis). 
- Thumb adduction (ulnar nerve, T1 adductor pollicis) test with Froman's paper grip test.

**Reflexes - SJ, BJ, TJ**
- Supinator (C5/C6), biceps (C5/C6), triceps (C7)
- Finger reflex if suspect C8 lesion: tap your fingers while placed over outstretched fingers of pronated hand. Often normally absent

**Coordination: rapid alternating movements, finger-nose test.**

**Gait**
- ALWAYS observe posture and gait: movement of arms, stride length, broadness, smoothness.
- Heel-toe walking: tests midline cerebellar vermis
- Romberg: tests dorsal column sensory loss (proprioception- rare in clinical practice) and vestibular function. Also test one leg standing for balance (with eyes closed if necessary)

**Rapid leg tests:**
- If they can walk on their heels, then no foot drop (L5 or common peroneal)
- If they can walk on their tiptoes, then no S1 lesion (plantar-flexion)
- To test proximal leg function, crouch and stand up or rising from a seat without using hands (up and go test) and

**Lower limb motor exam**

**Observe: Look for wasting (esp tibialis anterior and small muscles of feet) and fasciculations**

**Tone: knees, ankle clonus (2-3 beats may be normal if symmetrical)**

**Power**
- Hip flexion and adduction (ilio-psoas, L2-3, lumbar plexus).
- Hip extension and abduction (gluteus maximus, sciatic nerve, L5-S1).
- Knee Extension (quadrecteps, femoral n, L3-4).
- Knee Flexion (hamstrings, sciatic nerve, L5 – S1)
- Ankle dorsiflexion (tibialis ant peroneal n, L4 – 5)
- Ankle plantarflexion (gastrocnemius, sciatic nerve, S1 – 2)
- Ankle inversion (tibialis ant & Post, peroneal and tibial n, L4 – 5)
- Ankle eversion (peronei, peroneal nerve, L5 – S1)

**Leg (if no response, interlock fingers of both hands and pull just before tap)**
- Patella (hold knees up) (L3/L4), ankle (passively dorsiflex ankle) (S1).
- Plantar responses (Not positive if withdrawal response (hip and knee flexion))
- Superficial Abdominal reflexes: Not tested routinely. Stroke lightly with sharp object in each quadrant towards midline. Normal reflex is contraction. Tires quickly (T7-T11)
- Coordination: heel-shin test, tapping foot rapidly with heel on the ground
Cerebellum (lesion on ipsilateral side to symptoms)
- Flocculonodular (vestibulo-) cerebellum - truncal ataxia, vertigo, nystagmus
- Lateral (cerebro-) cerebellum - distal limb ataxia, intention tremor, heel-shin, rapid movements
- Midline (spino-) cerebellum - truncal ataxia (broad based drunk gait), broad-based gait, dysarthria, heel-toe problems

Sensory test
- Avoid suggestion. Test from area of least sensation outwards (better discrimination this way)
- Get patient to close eyes. Stimulate at irregular internals so patient can’t anticipate them. Test from abnormal to normal. Don’t try to completely map – just test key boundaries.
- Guide extent and focus of testing according to history and earlier examination findings.
- Common scenarios: Hemisensory (stroke, nerve root or peripheral nerve). Glove and/or stocking (spinal chord or peripheral neuropathy)
- Dermatomes:
  - Stand on S1,
  - Sit on S3,
  - Groin: L1,
  - Umbilicus T10,
  - Nipple T4,
  - T2 meets C4 on line connecting axillae,
  - Middle finger C7.
- Light touch (cotton wool)
- Pinprick: Use large safety pin and discard after use. Toes, fingers, face (no more unless suspicious, eg ↓ reflexes). Is it sharp or blunt? Can alternate sharp and blunt end to see if they can tell the difference. More reliable than light touch if both damaged.
- Position: Big toe and thumb. Hold digit by the sides, explain which way is up and down, then test. Has low yield in practice. If absent test next joint proximally.
- Vibration: 128 Hz fork. First sensation to go in progressive deterioration. On bony prominences (what do you feel?). Move up until positive. Bunion → medial melleolus → tibial tuberosity → anterior iliac spine. Test fingers for completeness.
- Temperature (Rarely done. Same pathways as pinprick)
- Others:
  - Two point discrimination
  - Stereognosis: recognising objects by their feel (coin, key, etc). Normal hand first
  - Graphaesthesia: write numbers on the hand
  - Sensory inattention: touch sides separately and together – which is being touched?

8: Abnormal Sensation
- Where is the lesion?
- What is the lesion?

Differential
- Dermatology: infection (eg Shingles before eruption), etc, pruritis (eg from cholestasis)
- Oedema
- Pain from musculo-skeletal injury or disease
- Peripheral neuropathy: diabetic, alcoholic, radiculopathy
- Central neuropathy: MS, stroke, spinal lesion (eg infection) etc
- Polyneuropathy:
  - Mostly motor: Guillain-Barre Syndrome, Lead poisoning, Charcot-Marie Tooth syndrome
  - Mostly sensory: Diabetes, Uraemia, Leprosy
- Vascular insufficiency (eg from claudication)
- Fibromyalgia
- Psychogenic
- Referred from viscera
**History**
- Time course, character, aggravating and alleviating factors, etc, other symptoms (weight loss in cancer, arthralgia in connective tissue disease), travel, sexual history, alcohol use, medications, family history

**Examination**
- Neuro exam:
  - Establish distribution
  - Distinguish peripheral distribution from spinal root
- Musculo-skeletal exam

**Investigations**
- Bloods: FBC, ESR, glucose, U&E, LFT, Thyroid, plasma B12, syphilis, ANA
- Nerve conduction studies

**Treatment**
- Involve physios and OTs. Care of feet.

---

### 14: Blackouts

**History**
- Before attack
  - Any warning: aura, etc?
  - What circumstances do attack occur?
  - Can patient prevent attack?
- During (any witnesses)
  - How long was the episode
  - LOC?
  - Did they injure themselves?
  - Patient move?
  - Incontinence (faecal or urinary)?
  - Bite tongue?
  - Colour of patient?
  - What is pulse like?
  - Assoc symptoms (palpitations, chest pain, dyspnoea)?
- After
  - Patient confused or sleepy, can they remember?
  - How much does patient remember afterwards?
- Past medical history: especially heart disease, head injury
- Medications
- Social history: alcohol and smoking

**Causes**
- Head:
  - Epilepsy:
    - Most likely to be grand mal if presenting as blackout
    - Suggestive of epilepsy: Urinary and faecal incontinence, tongue biting, aura (preceding seizure or part of the attack), post attack drowsiness or coma, amnesia, attacks when asleep or lying down, identifiable precipitant, altered breathing, cyanosis, typical movements,
  - TIA, stroke, increased ICP (eg haemorrhage, tumour)
- Heart:
  - Vaso-vagal
    - Onset over seconds
    - Preceded by nausea, pallor, and closing in of visual fields
    - Can’t occur lying down
    - Incontinence of urine rare, “never” faecal incontinence
    - May jerk limbs, but never tonic → clonic sequence.
  - Stokes Adams Attack:
• Transient arrhythmias (normally secondary to complete heart block) causing decreased CO and LOC
• A few clonic jerks may occur if attack is prolonged
• Patient falls to ground (often no warning except palpitation) → pale with slow or absent pulse
• Recovery in seconds → patient flushes, pulse speeds up, consciousness regained
• Can happen several times a day and in any posture
• Postural hypotension:
  - Unsteadiness or LOC on standing from lying due to inadequate vasomotor responses
  - Causes: Elderly, autonomic neuropathy (eg diabetes), anti-hypertensives, dehydration, over diuresis
• Situational syncope
  - Cough syncope: Weakness and LOC after coughing
  - Effort syncope: Syncope on exercise. Causes: HOCM, aortic stenosis
  - Micturition syncope: Mostly men, at night, at end of micturition
  - Carotid sinus syncope: Carotid sinus hypersensitivity in the elderly. Often caused by turning head or shaving.

• Other
  - Hypoglycaemia
  - Tremor, hunger and perspiration herald light-headedness or LOC
  - Rare in non-diabetics
  - Anxiety, Meniere’s, Fictitious, choking

Examination
• CVS, Neuro, BP lying and standing

Investigations
• ECG (heart block, arrhythmia, long Q-T)
• Blood: U&E, FBC, glucose
• Imaging: CT, echo, CT, MRI

• Ref: OHCM 4th Edition, pp 418 - 419

20: Coma

• ABC
  - Vitals, pupils and LOC (either GCS or ACVPU)
  - Stabilise C spine
  - O2 saturation and finger prick glucose
  - Insert IV and take blood: FBC, U&Es, LFTs, ESR, glucose, alcohol, drug screen, blood cultures, ABG
  - Have low threshold for giving O2, thiamine, glucose and/or naloxone

• The five levels of consciousness:

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Consciousness</td>
<td>Awake</td>
</tr>
<tr>
<td>2) Clouded consciousness</td>
<td>Confused (drowsiness / reduced awareness)</td>
</tr>
<tr>
<td>3) Stupor</td>
<td>Responds to Voice / shake and shout (unconscious)</td>
</tr>
<tr>
<td>4) Semicomatose</td>
<td>Responds to Pain</td>
</tr>
<tr>
<td>5) Coma</td>
<td>Unresponsive</td>
</tr>
</tbody>
</table>

• Arousal is controlled by the Ascending Reticular Activating System (from brainstem to thalamus). Decreased levels of consciousness are due to:
  1) Diffuse, bilateral, cortical dysfunction (ie METABOLIC cause = COM) or
  2) Direct physical effect on the ARAS (ie NEUROLOGICAL cause = A)

Differential
  - C = CO2 narcosis: respiratory failure
  - O = Overdose (or withdrawal)
The document contains information about causes of coma, examination, and treatment. The text is divided into sections titled "History," "Examination," and "Treatment," providing a comprehensive guide to assessing and managing coma cases. The sections are structured to guide the reader through understanding and managing various factors contributing to coma, such as opioids, alcohol, hypnosedatives, and metabolic causes. The document also emphasizes the importance of gathering information from witnesses and relatives, performing a thorough examination, and providing necessary treatments.

### History
- Gather information from witnesses and relatives. Is there a suicide note?

### Examination
- **Pupils**
  - Normal response = intact midbrain, mid-position (3-5mm)
  - Unreactive +/- irregular = midbrain lesion
  - Small reactive = pontine lesion
  - Unilateral dilated and unreactive = 3rd nerve
  - Horner's = ipsilateral medulla or hypothalamus
- **Breath**
  - Alcohol, uraemia, hepatic failure, ketoacidosis
- **Breathing**
  - Progressively deeper then shallower (Cheyne-Stokes) = cerebral or brainstem dysfunction
  - Shallow irregular = brainstem
  - Deep rapid hyperventilation (Kussmaul) = metabolic acidosis.
- **Skin**
  - Injection sites (diabetes, drug addict), medic alert bracelet or necklace, colour, texture, (snake/spider bites!!)
- **Hydration**
  - CVS, respiratory, abdo exam

### Treatment
- Supportive: ventilation, fluids
- Specific: eg dextrose, naloxone (half life may be shorter than opiate half life), thiamine (should always precede glucose infusion if there is any suspicion of Wernicke's).

### Memory Loss
- Explore patients complaints about their memory eg onset, associated symptoms etc
- ROS - neuro, thyroid, depression
- ADLs
- Medical history - past history of head trauma, heart/vascular disease
- Medications
- Alcohol use
- Family history, eg Alzheimer’s
- Home living arrangements and support systems

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2003 OSCE Handbook
Investigations (Depend on history)

- Head CT
- ECG
- Thyroid function

Possible causes

- Medication
- Head trauma
- Hypothyroidism
- Dementia / Alzheimer’s
- CVS problems eg several small strokes from AF
- Normal pressure hydrocephalus

Refer to 14: Blackouts on page 51

History Questions

- When do the seizures occur
- What do they remember BEFORE the turn? Aura = simple partial
- Do they remember DURING the turn? No = generalised or complex partial
- Did anyone WITNESS the turn?
  - Automatisms = non-specific, dystonic posturing = localising.
  - Are they aware – will they respond
  - Are their automatisms
  - Is there dystonic posturing
  - How long did it last
- What happened AFTER the turn? Todd’s paresis = partial, Post-ictal confusion = not absence
- Key differential:
  - Seizure
  - Cardiac cause: Arrhythmia, Stokes-Adam attack
  - Faint: vaso-vagal, postural hypotension
  - Any history of head trauma

Is it a seizure?

- Always consider other ‘spells’: syncope, pseudo-seizure (conversion disorder), TIAs, daydreaming, delirium, psychosis, drug abuse…

Generalised

- Bilaterally symmetrical without local onset
- Tonic-clonic (Grand mal) seizures: Tonic phase: 10 – 20 secs – extension phase then tremor begins – repetitive relaxation of tonic contraction. Clonic phase: usually 30 seconds, random movements, tongue often bitten
- Absence (Petit Mal) Seizures: Characteristic type of absence attack. Childhood or adolescent onset, associated with 3/sec spike and wave on the EEG. Blank stare and unresponsive for 5 – 15 seconds. No post-ictal confusion or sleepiness. May also have automatisms and mild clonic motion (usually eyelids at 3 Hz). May be induced by hyperventilation. 80% have no further seizures after 20 years old.
- Atonic: complete, sudden loss of tone – completely collapse, may injure themselves
- Tonic: sustained contraction, maybe with fine tremor. Usually brief.
- Myoclonic: Sudden, very brief jerk but still generalised. May be so brief that they are not aware they have had a seizure.
- Clonic: rhythmic jerking
- Infantile spasms: Sudden bilateral symmetrical jerk, extensor or flexor. Often subtle, occur in clusters. Usually around 3 – 6 months, boys > girls. Grow out of the spasms. Bad prognosis: cerebral palsy, retardation, etc. Medical emergency: try to urgently get them under control

Partial

- Begin locally
- In simple partial seizures consciousness is preserved.
• **Complex partial seizures** are focal seizures in which consciousness is altered (eg blank unresponsiveness followed by automatisms, eg lip smacking, other semi-purposeful activity) – usually temporal lobe but may be frontal. Can go on for minutes. Aware it is coming (cf absence which is sudden)

• **Partial seizure secondarily generalised**: they have an awareness first

• Localising it:
  - Preceding aura: olfactory, visceral, auditory, visual, déjà vu
  - Dystonic posturing: contraction of agonist and antagonist muscles
  - Post-ictal Todd’s Syndrome: if they have one area of weakness after a seizure (ie one hand weaker than the other) then it started locally

• Automatic behaviours are usually seen in complex partial seizures: but can be in absence (petit mal) seizures. Eg Oral or manual automatisms

**Differential in a Child**

• **Acute symptomatic**: any person in that situation would seize eg hypoglycaemia, heatstroke, meningitis, hyponatraemia. Seizure will stop when cause goes away (unless scarring – when it becomes a ‘remote symptomatic’ seizure)

• **Single Seizure** (may progress to Epilepsy)

• **Benign Febrile Convulsion**: Age 6 months – 5 years, temp > 38.5, occur only with a fever, family history common

• **Epilepsy**: Repeated *unprovoked* seizures

• **Anoxic** Seizures:
  - White breath-holding attacks: Vaso-vagal events due to stimulus: eg anger, pain, vomiting, etc. [Big sympathetic drive → parasympathetic overcompensation??]. Reflex bradycardia or brief asystole or peripheral vasodilation
  - Blue breath-holding attacks:1 – 5 years. Follow a stimulus, eg crying. Get worked up, don’t breath in, run out of breath and don’t breath in (actually stop breathing). Cyanosis with *retained heart rate*

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**47: Head Injury**

**Assessment**

• ABC: Assume C-spine injury until cleared by x-ray
• GCS - motor, verbal, eye. <9 is severe, >12 is minor
• Check pupils - unequal pupils likely to be due to raised ICP. Lesion is usually on the side of the fixed pupil.
• Brief neurological exam for spinal injury

**Treatment**

• 100% oxygen
• Maintain airway and ventilation. Intubate and ventilate if GCS < 9. If breathing satisfactorily place in lateral position. Beware of spinal injuries (present in 10%)
• Maintain BP at 120-160 systolic but do not over hydrate as this may worsen cerebral oedema in infants
• Clean and close scalp wounds to prevent further blood loss and infection
• Treat seizures - clonazepam 0.25mg/min up to 1 mg (4 mins)
• To reduce cerebral oedema want to maintain 70mmHg difference between MABP and ICP.
  - Sit at 30 degrees
  - Hypoventilate to reduce CO2 to 30-35mmHg
  - Avoid hypotension
  - Diuretic - frusemide 40mg IV and possibly acetazolamide to ↓CSF volume
  - Mannitol 0.5-1g/kg over 20mins IV
  - Other - cooling, barbiturates, surgical decompression

**CT scan if:**

• GCS < 15 at 4hrs
• GCS < 9 at any time
• Compound head injury
- Seizures
- Focal neurological signs

**Long term management**
- Help return to work
- Watch for and treat depression
- Support family

---

**48: Headache**

**Differential**

- **Acute single episode:**
  - Meningitis: Fever; photophobia; stiff neck; rash; coma
  - Encephalitis: Fever; odd behaviour; decreased consciousness
  - Sub arachnoid haemorrhage: Sudden headache; stiff neck; decreased consciousness
  - Sinusitis: Respiratory infection; tender face/sinuses
  - Head injury: Cuts/bruises; decreased consciousness; lucid interval; amnesia
  - Glaucoma: Constant aching pain develops over 1 eye + radiates to forehead; red eye; see haloes; fixed big pupil; decreased acuity

- **Acute recurrent attacks:**
  - Tension Headaches: bilateral, non-throbbing, every day, gradual onset, feels like a tight band around the head
  - Migraine: Pre-attack aura; seeing spots; vomiting; severe + unilateral + pounding; precipitants: diet (chocolate, cheese), stress, period, ↓ sleep, hunger, alcohol, sex, exercise
  - Cluster: Severe pain around one eye, facial flushing and swollen, watery eye; strictly unilateral; clusters of 20-60mins once or twice/day for 8wks, then pain free for months to 1-2 years. Non-throbbing. Rare <10y. (0% males.

- **Drugs**
- Adolescent headache - related to exertion. Common and usually benign

- **Chronic headache:**
  - GCA: Giant Cell Arteritis. Temporal headache; scalp tenderness; jaw claudication; >55 years old; increased ESR
  - Tension: “Tight band around head”; stress at work/home; decreased mood
  - Increased ICP: Worse on wakening, sneezing or coughing; vomiting; focal neuro signs; papilledema; increased BP; decreased pulse. Causes: tumour, pseudotumor cerebri (normal MRI, impaired CSF resorption), chronic onset hydrocephalus
  - Analgesic: Rebound headache on stopping taking analgesics
  - Paget’s: >40years old; bowed tibia; increased ALP.

**Examination**
- BP, neurology; ophthalmoscopy + pupils, MSE.

**Investigations**
- Blood: FBC; ESR; CRP
- Imaging: X-rays; CT

**Management**

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**26: Cranial Nerve Lesions**

- See 103: Cranial Nerve Exam, page 48, and 20: Coma, page 52
Differential Diagnosis

- Structural problem with speech production (⇒ dysarthria):
  - Foreign body
  - Vocal chord lesion: nodule, carcinoma, etc
  - Hypothyroidism – deepens voice
  - Infection – URTI
  - Laryngeal nerve palsy (secondary to lung tumor, thyroid surgery, etc)
  - Ineffective bellows function of the lung (eg severe asthma, CORD, etc)
- Neurological problem (⇒ dysphasia or dysarthria)
  - Causes: stroke, tumor, dementia, head injury
  - Dysphonia: problems with the mechanics, not ideas, of speech production (eg nerves involved in motor control, connective tissue disease, etc)
  - Aphasia
    - Broca’s (expressive) dysphagia: Non-fluent speech with malformed words. Reading and writing are impaired but comprehension intact. Patients understand questions. Inferior-lateral frontal lesion
    - Wernicke’s (receptive) dysphasia: Empty fluent speech. Maybe mistaken for psychotic speech. Reading writing and comprehension are impaired. Posterior Superior temporal lobe lesion.
- Dysarthria
  - Cerebellar disease: ataxia of the muscles of speech → slurred and irregular speech
  - Pseudo-bulbar palsy: UMN, spastic tongue with no wasting. Often labile emotions eg Motor neuron disease or severe MS
  - Bulbar palsy: LMN eg facial nerve, Guillian-Barre, radiotherapy etc

Assessment

- If speech fluent, grammatical and meaningful or patient can repeat a sentence ⇒ Broca’s unlikely
- If the patient comprehends simple instructions with several steps ⇒ Wernicke’s unlikely

Tremor

- Causes:
  - Physiological (ie they’re cold!)
  - Benign (essential) tremor
  - Anxiety (including hyperventilation)
  - Endocrine: Hyperthyroidism or Phaeochromocytoma
  - Toxic: alcohol, liver failure
  - Drugs: lithium, narcotic withdrawal
  - Parkinson’s disease
  - Drug induced Parkinsonism
  - Cerebellar disease
  - Alzheimer’s dementia
  - Lesions of the red nucleus and rarely with frontal lobe lesions.
- Differentiating:
  - Resting tremor: Parkinson’s: Pill-rolling tremor (4-7 Hz) associated with bradykinesia and rigidity. The tremor disappears on movement (eg finger nose test). Evoke tremor by distracting patient (ie pretend to examine left hand while looking at right). Also alcohol withdrawal
  - Action or postural tremor: Arms out-stretched and fingers apart (with a piece of paper on them). Everyone has a physiological tremor of the outstretched hands (~8-12Hz). Increased by anxiety, hyperthyroidism, sodium valproate, and lithium. Alcohol and Beta blockers may reduce it. It is abolished at rest.
• Intention tremor: Absent at rest, accentuated on finger nose test. Ipsilateral cerebellar disease. Associated with slowness and incoordination of rapid alternating movements (dysdiadochokinesis).
• Flapping (metabolic tremor): Slow coarse movements of extended wrists with arms outstretched: Wilson’s disease, hepatic encephalopathy, uraemia, respiratory failure, lesions of the red nucleus of the mid-brain
• Essential tremor: Positive family history + tremor with little disability + normal gait. Faster than Parkinson’s (8 – 13 Hz cf 4 – 6 Hz). Treatment: reassurance +/- propranolol
• Parkinson’s: Classic triad: tremor, rigidity, bradykinesia (poverty of movement). Mean age of onset = 60 years. Often starts unilaterally. Power, reflexes and sensation usually normal. ↓arm swing, start hesitation, slow turning, micrographia, masked facies, difficulty turning over in bed. Also early depression, progressive dementia after 10 years in 30%

**Dystonia**
• = sustained or intermittent abnormal repetitive movements or postures resulting from alterations in muscle tone
• ? disorder of the basal ganglia
• Blepharospasm: focal dystonia around the eye → uncontrolled blinking
• Writers cramp – focal dystonia of the hand
• Cervical dystonia: involuntary jerking or twisting of the head
• Lots of others
• Treatment: ? localised injection of botulinum toxin

**Chorea**
• Non rhythmic, jerky, purposeless movements (esp hands).
• Causes; Huntington’s chorea (Autosomal dominant. Reduction in GABA nergic and cholinergic neurones in the corpus striatum. Personality change -> dementia and death, epilepsy common. No treatment prevents progression.) and Sydenhams chorea. L dopa worsens chorea, stroke

**Tics**
• = sudden, rapid, stereotypical and involuntary movements of circumscribed muscle groups for no apparent purpose. Able to be suppressed for a while. In children, most resolve spontaneously. Consider clonazepam or clonidine if tics are severe.
• Motor and vocal tics can be a feature of Tourette’s syndrome. First appears when a child, duration > 1 year with no tic free period. Can be treated with haloperidol

**Other**
• Decreased movement of limb: Due to stroke leading to hemiplegia or left inattention.
• Fasciculations: Due to a L.M.N lesion. Decreased or absent voluntary movement of affected limb.
• Restlessness/Excessive movement: Anxiety, Hyperthyroidism, Opiate withdrawal

• NB: Multiple Sclerosis is characterised by weakness, visual disturbances and sensory impairment – rarely movement disorders. But have a high index of suspicion. Always UMN lesions.
• OHCM pg 428, Kumar and Clarke pg 1031. See OHCM pg 428 for more, less common abnormal involuntary movements

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**57: Limb Weakness**

**History**
• Onset, what happened (was there trauma)
• Distribution of weakness
• Associated symptoms e.g. fatigability, sensory loss/alteration, pain, bladder involvement
• Past medical Hx: predisposing conditions e.g. hypertension, previous stroke, malignancy
Examination
- Which areas are objectively weak?
- Power: Grading system:
  - Grade 0: No contraction
  - Grade 1: Flicker of contraction
  - Grade 2: Some active movement
  - Grade 3: Active movement against gravity
  - Grade 4: Active movement against resistance
  - Grade 5: Normal power

- Tone
- Reflexes
- Muscle wasting, fasciculations
- Sensory examination (if loss find the level)

Establish if upper motor neurone or lower motor neurone weakness

<table>
<thead>
<tr>
<th></th>
<th>Upper motor neurone</th>
<th>Lower motor neurone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tone</td>
<td>Spasticity</td>
<td>Hypotonia</td>
</tr>
<tr>
<td>Power</td>
<td>Reduced, effects muscle groups (not single muscles)</td>
<td>Reduced, flaccidity</td>
</tr>
<tr>
<td>Muscle wasting</td>
<td>Little or none</td>
<td>Marked muscle wasting/atrophy (may also see fasciculations of denervated muscle)</td>
</tr>
<tr>
<td>Reflexes</td>
<td>Hyper-reflexic, upgoing plantars, clonus, Hoffman’s reflex</td>
<td>Reduce or absent</td>
</tr>
</tbody>
</table>

Investigations
- Imaging: CT, MRI, spine x ray (as appropriate)
- Nerve conduction studies
- Electrolytes
- Tests for Myasthenia Gravis: Tensilon, anti-acetylcholine receptor antibody, repetitive nerve stimulation test, CT of thymus gland
- Others??

Differentials
- Stroke
- Cord compression
- Electrolyte disturbances: hyperkalaemia, hypokalaemia, hypophosphataemia
- Myasthenia Gravis
- Multiple sclerosis
- Motor neurone disease
- Myopathies

Management
- Treat the cause, symptomatic relief
- In non reversible weakness rehabilitation in important (physio, OT)
- Take care of associated needs e.g. if immobile prevention of pressure sores, addressing urinary retention or faecal incontinence

CT (Computed tomography)
- CT scans are described using density eg Hyper, Iso, or Hypodense.
- Hyperdense areas/structures are the brightest/whitest.
- The difference in X-ray attenuation between bone, brain, and CSF makes it possible to distinguish between normal and infarcted tissue, tumour, extravasated blood, or oedema.
- IV contrast can be used to show areas of increased blood supply and oedema more clearly.
- Intrathecal injection of water-soluble contrast media can be used for contrast imaging of the spinal cord and ventricles.
- CT is safe apart from occasional reaction to contrast media. The irradiation involved is small.
Limitations of CT:
- Lesions under 1cm diameter may be missed
- Lesions with attenuation close to that of bone may be missed if near the skull
- Lesions with attenuation similar to that of brain are poorly imaged e.g. MS plaques
- Lesions in the posterior fossa are sometimes missed
- The spinal cord is not imaged directly by CT (contrast is necessary)
- The results are poor when a patient cannot cooperate – A GA is occasionally required

Thrombotic Infarction – appearances on CT
- Ictus-24hrs:
  - Normal
  - Subtle loss of grey-white matter interface definition due to grey matter hypodensity
  - Hypodensity confined to the vascular territory involved
  - Little or no mass effect
- 24hrs-7days:
  - Hypodense wedge confined to a vascular territory
  - Homogeneous
  - Sharp margins
  - +/- mass effect depending on size
- 7-21days:
  - Iso to slightly hyperdense serpiginous bands and nodular regions developed within the hypodense infarct; these changes are located mainly in the grey matter
  - Infarcted white matter does not show density change
- 21days +
  - Changes approach the density of CSF
  - The isodense cortical bands are no longer seen
  - The infarct shrinks because of absorption of necrotic tissue and contraction from gliosis
  - There is dilatation of the adjacent ventricle
  - The overlying cortex is atrophic with enlargement of the adjacent sulci
  - The changes are usually stable at the end of the third month

MRI (Magnetic resonance imaging)
- Use the words “signal intensity” when describing a MRI scan e.g. the brightest (whitest) area has a higher signal intensity than a darker area.
- Which structure/fluid has the highest signal intensity depends on the weighting of the images.
- T1 weighted images:
  - Provide good anatomical planes and better separation of cystic and solid structures due to the wide variation of T1 values among normal tissues.
  - Fat has the highest signal intensity (appears brightest).
  - The remaining tissues appear as varying degrees of lower signal intensity.
  - Flowing blood appears black.
- T2 weighted images:
  - These provide the best detection of pathology and a decreased visualisation of normal anatomy.
  - Tumour surrounded by fat may not be seen on T2.
  - Fat and fluid have the highest signal intensity.
Advantages of MRI
- Non-ionising radiation
- Shows vasculature without contrast
- Images can be produced in any plane eg sagittal and coronal (good for spinal cord, aorta, vena cava)
- Visualisation of posterior fossa and other areas prone to bony artefact on CT eg cranio-cervical junction
- High inherent soft tissue contrast
- Precise staging of malignancy

Disadvantages of MRI
- High cost of equipment
- Long imaging time causes increased motion artefact
- Claustrophobia (in magnet tunnel for 30-120mins – hypnosis, music, or sedation may be needed)
- Unsuitable for those with metal foreign bodies eg pacemakers, CNS vascular clips, cochlear implants, valves etc
- Unable to scan very ill patients requiring monitoring equipment
- Unable to image calcium

**Thrombotic Infarction – appearances on MRI**
- **Ictus – 24hrs**
  - T2: Increased signal intensity & Increased thickness of involved cortex
  - T1: Decreased signal intensity & Cortical thickening and loss of sulci
  - Note: MRI is more sensitive than CT in the first 24 hours
- **24hrs – 7days: T2 - Hyperintensity in the region of the infarct**
- **7 – 21days**
  - T2: Loss of hyperintensity that had previously arisen from oedema and of the cortex
  - T1: Isodense bands
- **21days +: Changes become more homogenous and approach the density of CNS**
Visual Acuity
- Ask:
  - Do you normally wear glasses or contacts?
  - Why and when do you wear them?
  - When was the last time you had your eyes checked
- One eye at a time
- Cover eye with palm of hand (not fingers)
- Snellen chart (use glasses if normally worn) use pinhole if < 6/6 (the smaller the fraction the worse the vision):
  - Clearly state where on the chart to start reading
  - Tell the patient to read above or below as appropriate
- Record:
  - Which eye
  - Distance from the chart in metres on top, chart number on bottom
  - Add/subtract extra/missed letters as appropriate
  - Record if wearing spectacles, etc
- If they can’t read the top line then:
  - Count fingers ½ metre distance
  - Hand movement ¼ metre distance
  - Light projection - can they tell if a light is shone on dark wall
  - Light perception - can they tell if a light is shone in their face
- Near vision (in patients > 40yrs complaining of near vision blurring – probably presbyopia)
- Test with pinhole

Visual Fields
- Hat pin confrontation (red or white tipped). One eye at a time while patient sits one metre in front of examiner focusing on their nose.

Extraocular Movements
- Both eyes together. Move finger in H pattern while patient keeps head still and follows finger with their eyes. Ask patient which movement provokes the most diplopia.
- Corneal light reflex
- Cover test

External examination
- Lids
- Conjunctiva
- Cornea using fluorescein drops
- Anterior chamber
- Pupils – PEARL (Pupils Equal And Responsive to Light)
- Lens

Ophthalmoscopy
- Hypertensive retinopathy (see 50: hypertension, page 39)
- Diabetic retinopathy
  - Non-proliferative - microaneurysms, blot haemorrhages, hard exudates (+ cotton wool spots = advanced)
  - Proliferative - new vessel growth
  - Maculopathy - hard exudates around macula
- Cotton wool spot (soft exudate) = retinal infarction
- Hard exudates = protein/lipid exudate
- Flame haemorrhage = arteriole bleed into nerve layer
- Blot haemorrhage = subretinal bleed
- Dot haemorrhage = capillary bleed
• Microaneurysm = swollen capillary
• Roth’s spot = central white infarct on red background due to infective emboli.

Slit lamp
• OHCS pg 476

7: Abnormal Pupils

Differential diagnosis
• Unilateral (local pathology)
  • Trauma (eg foreign body)
  • Inflammation of the eye (eg anterior uveitis)
  • 3rd nerve lesion (may be due to ↑ICP, cerebral oedema)
  • sympathetic nerve damage (eg Horner’s syndrome)
• Bilateral (systemic pathology)
  • Neurological lesions:
    • Midbrain = mid-position and fixed
    • Pons = pinpoint
    • Tectal = large and fixed
  • Metabolic eg hypoglycaemia (dilated), opioid OD (small and reactive)
  • Drugs
  • Hypothermia
• Irregular shaped pupils: iritis, syphilis, trauma, globe rupture

History
• Symptom assessment: Bilateral/unilateral, time course, associated pain or change in vision
• ROS: neurological
• Risk assessment: eg Ankylosing Spondylitis → iritis
• Drugs (eg anticholinergics, overdose)

Examination
• Are they equal, central, circular, reactive to light (directly and consensually)
• If unequal – which is constricted and which is dilated
• Fundoscopy and fluorescein drops
• Neuro-exam

Eponymous pupils
• Hutchinson pupil: rapidly ↑ unilateral ICP. Pupil on affected side first constricts then dilates then the other pupil goes through the same process.
• Argyl Robinson pupil: small and irregular and unequal. React to accommodation but not to light = syphilis or DM
• Holmes-Aide (myotic) pupil: unilateral in 80%. Benign and usually in females. Moderately dilated pupil poorly reactive to light and slow to accommodate. This with decreased or absent ankle and knee reflexes = Holmes-Aide syndrome.

10: Abnormal Vision

Symptoms
• Decrease in visual acuity or visual fields:
  • refractive error
  • ↓transparency (cornea, lens, vitreous)
  • retinal lesion (vein, artery, inflammation/infection, drusen, glaucoma)
  • neural lesion (optic n, chiasm, tract, LGN, radiation, occipital cortex, agnosia)

• Superimposed visual phenomena:
  • Floaters: opacities in vitreous
  • Haloes: diffraction causing rainbow rings = corneal oedema
  • photopsia - lights/patterns - retinal detachment, migraine, vascular insufficiency
• metamorphopia - abnormal appearance of objects eg squiggly lines when they should be straight = macula oedema or degeneration

• Diplopia:
  • cranial n. palsies
  • extraocular muscle lesions eg myasthenia gravis
  • increased orbital pressure eg orbital cellulitis, thyroid eye disease
  • (cataract may cause monocular diplopia)

**History**
• What do they mean by ↓ vision. Eg blurring, complete loss or blind spot
• Central (↓ acuity - can they still read/watch TV?) or peripheral (blind spots = scitoma).
• Unilateral or bilateral
• Sudden onset or gradual
• Other eye symptoms:
  • Abnormal sensation - pain/discomfort, itching, photophobia, dry, watery
  • Abnormal appearance - red eye, abnormal pupils

**Examination**
• Visual function - acuity and fields
• Extraocular muscle function - eye movements
• Pupils - size, reaction to light and accommodation
• Ophthalmoscopy
  • cornea (fluorescein stain) + corneal light reflex
  • lens and vitreous (red reflex)
  • retina (optic disc and cup, vessels, retina)

• Ref: OHCS 5th Ed p474

**Differential**
• **Cornea**
  • Inflammation (keratitis): white spot on cornea = collection of white cells
  • Abrasion/ulceration: epithelial breach with/without keratitis. Causes include: contact lenses, dry eyes (age, Sjogren's), inability to oppose eyelids (ectropion, thyroid eye disease, VII n. lesion, trachoma = chlamydial infection),
  • Infection: eg herpes simplex or zoster, pseudomonas
  • Acute (closed angle) glaucoma: blurs vision due to corneal oedema
• **Iris**: acute iritis (anterior uveitis)
• **Lens**
  • Refractive error: myopia, astigmatism, hypermetropia, presbyopia
  • Cataract: lens opacity causes blurred vision. Glare is troublesome. Changing specs only helps for a little while. Measure blood glucose and Ca to exclude DM and hypocalcaemia
• **Vitreous**: haemorrhage: more common in diabetics, loss of red reflex
• **Retina**:
  • Detachment: premonitory symptoms in 50%. Peripheral +/- central visual loss. Due to traction (DM), exudate behind retina (tumour, leaky vessels) or tear in retina (myopia)
  • Central retinal artery occlusion: loss in seconds, RAPD, pale retina, sparing of tract from macula to disc.
  • Retinal vein occlusion: branch or central vein. “Blood storm” appearance.
  • Macular degeneration: central visual loss. "wet" = rapid and treatable, "dry (drusen atrophy)" = slow and progressive.
  • Retinitis pigmentosa: various genetic abnormalities: black spots on retina
• **Diabetic retinopathy**
  • background = hard exudates, microaneurysms, blot haemorrhages
  • proliferative = neovascularization, cotton wool spots, haemorrhages
• **Hypertensive retinopathy**: cotton wool spots, flame haemorrhages, rarely papilloedema
• **Choroid**: chorioretinitis (choroiditis): toxoplasmosis, toxicaria (nematode = roundworm), Tb, sarcoidosis (- these all cause granulomatous inflammation). AIDS CMV.

• **Neural**:
  - Open angle glaucoma: visual fields impaired first so bump into objects. Family hx. Changing specs doesn't help.
  - Optic neuritis – hours to days, unilateral decrease in acuity, colour desaturation, causes include MS (develops in 45-80% in 15yrs) and diabetes. RAPD. Slight ocular discomfort.
  - Ischaemic optic neuropathy: occlusion of posterior ciliary a. due to giant cell arteritis or atherosclerotic plaque. Fundoscopy shows pale, swollen disc. Check ESR in all cases of sudden painless visual loss.
  - CVA/TIA
  - Amblyopia

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**34: Disturbance of Eye Movement**

- For disturbance of eye movements re *extraocular muscle lesions* see 10: Abnormal Vision page 63
- For disturbance of eye movement re *squint* see 79: Strabismus, page 65
- For more on the causes of *nystagmus* see 35: Dizziness or vertigo, page 68

**Nystagmus differential**

- Horizontal and rotary nystagmus
  - Peripheral lesion (nystagmus away from lesion, ie toward the side that is tonically firing)
    - Nystagmus usually acute and transient
    - Assoc. with severe prostrating vertigo
  - Central lesion:
    - Cerebellar: nystagmus in both directions but worse on looking to side of lesion.
    - Parietal lesion: ipsilateral pursuit and contralateral saccade defects
    - Acute frontal lobe = gaze deviation towards side of lesion
    - Long lasting nystagmus
    - Vertigo tends to wane after days or weeks (nystagmus outlasts it)
- Vertical nystagmus (only caused by central lesions)
  - Up-beat nystagmus = non-specific central lesion
  - Down-beat nystagmus = medulla/ponto-cerebellar lesion. (eg meningioma)
- Pendula nystagmus
  - Almost always binocular, horizontal and present in all directions of gaze
  - Cause is invariably ocular, when there is poor visual fixation due to loss of central vision early in life or a congenital lesion, when it is associated with head-nodding

**Investigations**

- Calorimetry
- Imaging: MRI


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**79: Strabismus**

- Always refer children with strabismus to exclude ocular pathology such as retinoblastoma, congenital cataract or glaucoma, which would require urgent surgery
- Children with strabismus (even if the ocular exam is normal) need specialist management because the deviating eye will become amblyopic (a lazy eye with reduced vision). The younger the child, the easier it is to treat amblyopia; it may be irreversible if first detected later than school age.

- Exotropia = divergent squint
- Enotropia = convergent squint - commonest type in childhood, may have no cause or be due to hypermetropia
- Prominent epicanthic folds may produce pseudo-squint
Non-paralytic squint

- Usually start in childhood
- Normal range of eye movement
- Diagnosis - difficult in uncooperative children
- Screening tests:
  - Corneal reflection: reflection from a bright light falls centrally and symmetrically on each cornea if no squint, asymmetrical if squint present.
  - Cover test: movement of the uncovered eye to take up fixation as the other eye is covered demonstrates manifest squint; latent squint is revealed by movement of the covered eye as the cover is removed.
- Management
  - The 3 O’s: Optical; Orthoptic; Operation
  - Treatment should start as soon as the squint is noticed
  - Optical Assess the refractive state of the eyes after cyclopentolate 1% eyedrops: cycloplegia allows objective determination of the refractive state, glasses may be required; mydriasis allows a good view into the eye to exclude abnormality, eg cataract, macular scarring, retinoblastoma, optic atrophy
  - Orthoptic The good eye may be patched to encourage use of the one tending to squint.
  - Operations These help alignment and give good cosmetic results

Paralytic squint

- Diplopia is most marked when trying to look in the direction of pull of the paralysed muscle.
- When the separation between the two images is greatest the image from the paralysed eye is furthest from the midline and faintest.
  - 3rd nerve palsy. Eye looking down and out, ptosis, proptosis, fixed pupil dilatation.
    Causes: Cavernous sinus lesions, superior orbital fissure syn, diabetes, post communicating artery aneurysm
  - 4th nerve palsy: Eye is elevated in adduction and cannot look down and in (superior oblique paralysed). Head may be held tilted (ocular torticollis) Causes: Trauma, diabetes, tumour, idiopathic
  - 6th nerve palsy Eye is medially deviated and cannot move laterally from the midline.
    Causes: Tumour causing ↑ICP, trauma to base of skull, vascular

History

- Essentials of history:
  - History of trauma
  - Vision
  - Degree and type of discomfort
  - Presence of discharge
  - Presence of photophobia
  - Social and occupational history
  - Objects in or near eye (contacts, drops or ointments)
- Identifying the dangerous eye:
  - Is acuity affected?
  - Is the globe painful?
  - Does the pupil respond to light
  - Is the cornea intact? (use fluorescein eye drops)
- Unilateral red eye always should consider:
  - Trauma
  - Foreign body (including IOFB)
  - Corneal ulcer
  - Iritis (uveitis)
  - Viral conjunctivitis (commonest type)
  - Acute glaucoma
Examination

- Testing and recording vision
- Meticulous inspection under magnification
  - Foreign body
  - Irregular pupil
  - Episcleral vessels
  - Mucopurulent discharge (bacterial conjunctivitis)
  - Dendritic ulcer
  - Conjunctival vessels
  - Papillae of allergic conjunctivitis (on everted eyelid)
- Testing the pupils
- Testing ocular tension
- Local anaesthetic test
- Fluorescein test
- Subtarsal examination

Major causes

<table>
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<tr>
<th></th>
<th>Pain</th>
<th>Discharge</th>
<th>Vision</th>
<th>Photophobia</th>
<th>Pupil</th>
<th>Cornea</th>
<th>Ocular Tension</th>
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<tr>
<td>Conjunctivitis (Bacterial)</td>
<td>Gritty</td>
<td>Purulent</td>
<td>Normal</td>
<td>No</td>
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<td>Sub-conjunctival Haemorrhage</td>
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<td>Normal</td>
<td>No</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
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<tr>
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<td>No, reflex lacrimation</td>
<td>Blurred</td>
<td>Yes</td>
<td>Normal</td>
<td>Abnormal</td>
<td>Normal</td>
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<td>Normal</td>
<td>No</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
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<td>No, reflex lacrimation</td>
<td>Blurred</td>
<td>Yes</td>
<td>Constricted + irregular</td>
<td>Normal</td>
<td>Normal or low</td>
</tr>
<tr>
<td>Acute glaucoma</td>
<td>YES</td>
<td>No, reflex lacrimation</td>
<td>Blurred + haloes</td>
<td>Yes</td>
<td>Dilated + fixed</td>
<td>Hazy</td>
<td>Elevated + hard</td>
</tr>
</tbody>
</table>

- Also consider: FOREIGN BODY, Stye, dry eyes, flash burns, problems with contact lenses.
- In children also consider: periorbital/orbital cellulitis

Treatment

- Acute glaucoma
  - Pilocarpine 2-4% hourly (miosis opens drainage angle)
  - Acetazolamide 500mg PO (decreases formation of aqueous)
  - Surgery

- Acute iritis
  - Steroids (prednisolone 0.5% drops every 2 hours)
  - Cylcopenolate 0.5% 1-2 drops/6hr to keep pupil dilated

- Episcleritis/scleritis
  - Must identify if underlying cause (eg autoimmune condition) is present
  - Refer, especially for scleritis
  - Corticosteroids or NSAIDS may be used
Subconjuctival haemorrhage: Nothing, unless recurrent, in which case look for bleeding diatheses and check BP

Corneal ulceration: Prophylactic antibiotics (eg gentamicin 0.3%)

Ulcerative keratitis
  Initial: Urgent gram stain then…
  - G -ve: cefuroxime 50mg/ml; G +ve: genatmicin 15mg/ml
  - Fungal only: miconazole 1 %
  - No bacteria:gentamicin 15mg/ml + cefuroxime 50mg/ml
  - Change ot most suitable antibiotic when organism is known

Ref: Murtagh, General Practice 2nd Edition, Chapter 47, pp 475 - 486

35: Dizziness or vertigo

See also 34: Disturbance of eye movement, page 65

You must first determine if the problem is due to light-headedness (eg arrhythmia, postural hypotension) or vertigo. Does it fell like he/she is going to faint or does it feel like the room is spinning around?

Vertigo falls like you are spinning in the same direction as the direction of nystagmus

Differential of Vertigo

Peripheral (vestibular end-organs)
  - BPPV (posterior canals affected)
    - sudden-onset rotational vertigo
      - symptoms elicited with Hallpike test → latency phase then vertigo with rotational nystagmus (in one direction) for 10-20sec; diminished response on repeated tests.
  - Vestibular neuronitis: following febrile illness → abrupt onset of severe vertigo, nausea and vomiting exacerbated by head movement (horizontal canals affected). Nystagmus away from the side of the lesion.
    - Head impulse test → saccadic movement on turning head quickly to affected side.
    - NO tinnitus or deafness
    - Patient prostate and immobile
    - Vertigo subsides in days, complete recovery in 3-4 wks
  - Meniere’s
    - Triad of deafness, tinnitus, vertigo
    - Recurrent, spontaneous attacks
  - Trauma
  - Ototoxicity
  - Motions sickness
  - VIII nerve: acoustic neuroma
    - Present with hearing loss, vertigo is a late (or absent) sign
    - With progression, ipsilateral cranial nerves V, VII, IX, X may be affected
    - Signs of raised ICP late
  - Central
    - Brainstem and cerebellum: MS, Infarction, Haemorrhage, Migraine
      - Test for vertical nystagmus by dropping head straight back over the bed.
      - Alcohol: intoxication, Wernicke’s

History

- Duration of vertigo, triggers, associated symptoms eg tinnitus
- ROS: neurological
- Risk assessment
  - Recent viral illness
  - Past use of ototoxic drugs (eg gentamicin),
  - Stroke risk factors
- Migraine risk factors: unilateral headaches, family history.

**Examination and Investigations**
- Test hearing, cranial nerves, cerebellar function, and reflexes
- Romberg's test (+ive if balance worse with eyes shut)
- Do provocation test (Hallpike test)
- Calorimetry (cold water in ear drum)
- CT, MRI, EEG, LP

Ref: Murtagh, General Practice 2nd Edition, Chapter 42, pp 426 434.

**33: Balance**
- See also 35: Dizziness or vertigo, page 68

**Differential of neurological causes of poor balance (also consider cardiac and vestibula systems)**
- Peripheral nerves: DM, alcoholism, connective tissue disorders eg SLE
- Posterior columns (light touch, vibration and proprioception)
  - Clumsiness
  - Numbness
  - Band-like sensation
  - Tingling of limb and electric-shock like sensations
- Brainstem
  - Pontine lesions: affect all forms of sensation on side opposite lesion. III, IV, V, VI and VII cranial nerve nuclei often involved.
- Cerebellar lesions
  - Lateral lobes
    - Distal limb ataxia
    - Finger-nose/heel-shin difficulties
    - Dysdiadochokinesia
    - Dysarthria
  - Vermis (midline cerebellar)
    - Truncal ataxia → difficulty standing and sitting unsupported
    - Broad-based ataxic gait.
  - Flocculonodular: Vomiting, vertigo and ataxia of gait if it extends into roof of fourth ventricle. Tilted head posture.
- Basal ganglia: Parkinson's, Huntington's
  - Develop bradykinesia or akinesia, with muscle rigidity + involuntary movement.,
  - Lesion of corticospinal tract (UMN lesion)

**68: Painful Ear**
- Pinna – haematoma – evacuate to prevent ischaemic necrosis of cartilage ('cauliflower ear')
- TMJ dysfunction – articular disc, arthritis or myofascial (muscle clenching)
- Ear canal
  - Otitis externa - pain on pulling pinna and pressing tragus. Purulent discharge
  - Boils
  - Impacted wax
- Middle ear
  - Otitis media- bulging drum, deafness, may have discharge if perforated
  - Mastoiditis - foul-smelling discharge present for more than ten days. Look for swelling behind the ear and downward displacement of pinna
  - Barotrauma - history of air travel or diving
  - Cholesteatoma – foul-smelling discharge, attic perforations or retractions
- Referred pain
  - Trigeminal – sphenoidal sinus, teeth eg dental abscess
  - Greater auricular nerve (C2, 3) – neck and cervical vertebrae
  - Facial nerve – geniculate herpes (followed by facial n. palsy = Ramsay Hunt syndrome)
  - Glossopharyngeal and vagus nerves – pharynx and larynx (cancer, tonsillitis, pharyngitis, quinsy)
Causes

- Outer or middle ear
  - Ear wax
  - Otosclerosis – 50% have family history
  - Otitis media
- Inner ear or neural
  - Noise
  - Viral
  - Hearing loss including presbycusis – tend to be elderly
  - Meniere’s disease – tends to be high-pitched, sudden onset with vertigo
  - Drugs – aspirin, loop diuretics/aminoglycosides, marijuana
- Other
  - Idiopathic
  - Anaemia
  - Hypertension
  - Psychological
  - Head injury

Important points

- Investigate unilateral tinnitus fully (eg MRI) to exclude acoustic neuroma.
- Early morning headache and visual disturbance suggest a space-occupying lesion.
- Depression in tinnitus has been severe enough to cause suicide. Make a psychological assessment
- Roaring tinnitus on nose breathing, absent on mouth breathing, suggests a patulous Eustachian tube. The eardrum may be seen to move with breathing

Investigations

- FBC and U&E if anaemia or renal failure is suspected
- Salicylate levels if aspirin overdose possible
- Tymanogram for middle-ear function and stapedial reflex threshold. Audiogram to objectively assess hearing loss
- Cerebral angiography if vascular pathology suspected
- MRI scan is the most sensitive way to examine the inner ear and skull for structural lesions

Management

- Try gencobalobar (a herbal remedy, doses recommended on packet are homeopathic)
- Educate and reassure
- Relaxation techniques
- Treat any depression which arises
- Background noise – eg music
- Drugs usually unhelpful except beta-histine in Meniere’s disease.

38: Ear Discharge

- Furunculosis - staph infection of hair follicles in outer third of canal. Pain on movement of pinna. Rx = heat, analgesia and topical or systemic ABs depending on systemic symptoms/cellulitis
- Otitis externa - Discharge is offensive if coliforms. Also causes pain on movement of the pinna, itching (esp if secondary to eczema or psoriasis), fullness, hearing loss. Causes: bacteria - Pseudomonas, E coli, S aureus, Proteus, Klebsiella. Fungi - Candida (pale cream debris), Aspergillus (black spores). Rx = swab, aural toilet (dry cleaning of canal) and keep dry (eg vasoline, blu-tak), topical ABs and steroids (eg sofradex), wicks
- Cholesteatoma - discharge usually scant and never perfuse, foul odour, purulent. Hearing loss. Perforated or retracted pars flaccida. Rx= referral for surgery
- Acute suppurative otitis media - Pain, fever, malaise, hearing loss. Pain relieved by perforation causing profuse purulent discharge that usually settles after a few days. Rx = amoxicillin
Mastoiditis - as for acute otitis media but discharge continues for days. Subperiosteal abscess may cause swelling behind ear and displace pinna downwards. Rx = surgery. (NB - discharge may also continue after AOM if grommets are present.)

Chronic suppurative otitis media - discharge and hearing loss but no pain. Rx = aural toilet, ABs, steroids and surgical repair of drum

Other rarer causes
- Barotrauma (divers and plain travellers) - due to rapid change in atmospheric pressure and an occluded eustachian tube. Occasionally causes discharge. Rx = analgesics and reassurance and advice about prevention
- Foreign bodies - discharge secondary to infection or bleeding due to penetration of ear drum
- Neoplasia
- Rinnohroea secondary to skull fracture

4: Abnormal Hearing

Degrees of hearing impairment:
- Loss of 20 – 40 dB: mild (loss of soft spoken voice)
- Loss of 40 – 70 dB: moderate (40 dB is normal spoken voice)
- Loss of 70 – 90 dB: sever (shout)
- Loss of over 90 dB: Profound

Children
- Conductive loss: Middle ear effusion is common and temporary
- Sensorineural loss (Permanent deafness in infancy is rare. Incidence 1-2/1000)
  - Hereditary
  - Acquired
    - In Utero – infections (TORCH), ototoxic drugs, congenital abnormality
    - Perinatal – anoxia, birth trauma, cerebral palsy, kernicterus
    - Postnatal – meningitis chiefly, ototoxic drugs, lead, skull fractures, tumour, hypothyroidism

Testing:
- Parents will know – take them seriously.
- Look for absence of response to loud sounds, speech delay, inability to follow simple commands, etc. Screen at 8 – 9 months and at school entry.
- Hearing should be tested before 8 months old using Auditory Brain Stem Response (ABR).
- 12 months – 2 years (objective) Otoacoustic emissions. Evoked response audiometry
- > 3 years – pure tone audiometry.

Treatment
- Hearing aids
- Encourage parents to talk as much as possible to their deaf children.
- Schooling. Ordinary schools for the partially deaf. Special schools for the completely deaf.
- Sign language
- Cochlear implant suitable for those with sensorineural deafness too severe to be helped by hearing aids.

Differential Diagnosis in Adult
- Unilateral
  - Conductive:
    - Ear canal: impacted cerumen (wax), otitis externa
    - Ear drum: perforation
    - Middle ear: serous otitis media, otosclerosis
  - Sensorineural: Meniere’s, acoustic neuroma, herpes zoster, infarction/vascular disease

Bilateral
- Noise induced
- Presbycusis
- Ototoxicity
- Psychiatric: Auditory hallucinations present in 74% of Schizophrenics.
- Tinnitus: Almost everyone has experienced tinnitus but only 0.5-2% are severely affected. Peak age of onset 50 – 60 years. Investigate unilateral tinnitus fully (eg MRI) to exclude acoustic neuroma

**History**
- Difficulty hearing. If mild may only be in certain situations (eg crowded rooms) or certain sounds (eg s, f or th)
- Onset and progression
- Other ear symptoms: pain, discharge, tinnitus, vertigo (in Meniere’s disease, MS, acoustic neuroma or syphilis)
- Risk assessment: recent infections (eg mumps), drugs (alcohol, aminoglycosides, frusemide, quinine, salicylates), occupation (past and present), use of firearms, diving/air travel, head injury, family history

**Exam**
- Inspect face, skull, ears
- Otoscope for obstruction, intact membrane, signs of inflammation
- Hearing tests:
  - Hair rubbing
  - Tuning fork (512 hz):
    - Weber: Tuning fork on midpoint of skull. If unilateral sensorineural heard in good ear, if unilateral conduction loss then heard better in bad ear
    - Rinne’s test: Held outside ear and on mastoid process. Normally air conduction is better than bone. Bone louder in conductive loss
  - Pure tone audiometry, tympanometry and Auditory brain stem response (ABR)
- Cranial nerve exam

**Treatment**
- Find cause
- Syringing: only for wax in people with intact TM and a known healthy middle ear
- Children with sensorineural loss or sudden onset in age: specialist referral
- Elderly: usually presbycusis – greater loss of high frequency. Also associated with mental illness (anxiety, depression, agitation, etc)
- Otosclerosis: stapedectomy (90% effective), hearing aid (less effective)
- Hearing aids: for conductive loss
- Advice for families: speak directly to them, clearly at a uniform pitch, be patient
Psychological Medicine

105: Higher Mental Function

- Mini-Mental State Examination: (this is not a mental state exam – it is a limited test of cognitive function, which is only one component of mental state)
- Level of consciousness
  - Alert, drowsy/lethargic (needs continual stimulus to be kept awake), obtunded (can be kept awake by painful stimulus), stuporose (responds to pain), comatose (unconscious and unresponsive)
- Orientation
  - Time 5/5
  - Person
  - Place 5/5
- Memory
  - Short 3/3
  - Recent
  - Remote
  - Recall 3/3
- Attention and concentration
  - Spell "world" backwards 5/5
  - Serial 7’s or say five digits in reverse order
- Language
  - Name two objects 2/2
  - Say "no ifs, ands or buts" 1/1
  - Follow three step command - "take paper, fold in half, put on table" 3/3
  - Read and obey "close your eyes" 1/1
  - Write a sentence 1/1
  - Copy a design 1/1
- Other
  - Clock face
  - Draw five point star (good for hepatic encephalopathy)
  - Abstract thinking - interpret a proverb

Bullet points with a score indicate questions form the MMSE. Total score is 30.

23: Confusion

- Sudden onset = delirium (acute confusional state). A disturbance of consciousness, attention, perception, thinking, memory, behaviour, emotion and sleep-wake cycle. This condition is frequently missed especially if the patient becomes withdrawn
- Causes: Almost anything so go through your sieve

- Compares with Gradual onset = dementia differential. Impairment of memory plus some other aspects of brain function but NOT impairment of consciousness. NOT a feature of normal aging.

Differential

- Acute:
  - Delirium (often secondary to a medical condition)
  - Psychiatric disorder: eg anxiety disorder, psychotic disorder
  - Drugs or alcohol (use of withdrawal)
  - Medical condition eg metabolic disturbance, neurological (eg CVA, post-ictal)
- Chronic:
  - Dementia: Alzheimer’s, Vascular, Lewy Body
  - Psychiatric illness: eg depression
  - Drugs or alcohol
  - Medical conditions eg hypothyroidism, hepatic encephalopathy
**Examination**
- Neurological and gait
- CVS
- MSE

**The Delirious Patient**
- Consciousness:
  - Impaired
  - Onset over hours or days
  - Fluctuates throughout day (worsens late afternoon and evening)
  - Disorientation in time and place
- Behaviour:
  - Inactivity, quietness, reduced speech or preservation **OR**
  - Hyperactivity, noisiness and irritability
- Thinking:
  - Slow and muddled
  - Ideas of reference or delusions
- Perception:
  - Disturbed
  - Illusions and hallucinations (especially visual)
- Mood
  - Lability, anxiety, perplexion
  - Fear, agitation, or depression

**Management**
- Identify and treat the underlying cause (most often a UTI, pneumonia or post-op)
- Supportive:
  - Reduce stress and prevent accidents and give reassurance
  - Nurse in quiet, moderately lit room with the same staff
  - Repeated reassurance and orientation to time and place
  - Attention to hydration and nutrition
  - Minimise medication
- If agitated:
  - Haloperidol 0.5mg bd up to 5mg 1-
  - Lorazepam 0.5-2 mg q 15-20 min


### 136: Abnormal Grief

#### Characteristics of Grief
- Reassure bereaved person that these are normal. If overwhelming, seek help
- Emotional: bewildering and intense range or emotions without warning - shock, numbness, relief, anxiety, anger, blame, guilt, loneliness, helplessness, hopelessness
- Physical: hollow stomach, tight chest, breathlessness, weakness, lack of energy, ↓sexual desire, sleep disturbances, symptoms similar to person who died (can be pathological)
- Cognitive responses: disbelief, confusion, ↓concentration, going crazy, preoccupation
- Behaviours: searching, crying, sighing, absent minded, restless, ↓socialising, visiting/avoiding places that are reminders
- Symptoms usually resolve in 6-12 months.
- Kubler-Ross stages of grief: 1) denial and disbelief, 2) anger and guilt, 3) bargaining, 4) depression, 5) acceptance and recalling the good times.

#### Abnormal grief
- Extreme vegetative symptoms eg profoundly depressed mood, major sleep disturbance.
- Feeling worthless and hopeless (what does the person think about **themselves** not the person who has died)
- Marked and enduring impact on social or occupational functioning
Factors Complicating Grief – Risk Factors for Pathological Grief

- Dependent family members (children, handicapped, elderly)
- Loss of primary care giver/constant companion
- Loss of financial provision
- Loss of home (feared or actual)
- Anxiety about decisions
- Unable to share feelings
- Family discord
- Uncontrolled pain/emotional distress before death
- Concurrent life crisis
- Prolonged reaction/suicidal thoughts
- Lack of community support

Signals for attention from a grieving child

- Marked change in behaviour: illegal behaviour, persistent aggression (> 6 months), tantrums, withdrawal, drug abuse
- Inability to cope with problems and daily activities
- Many complaints of physical ailments
- Persistent depressions, panic attacks
- Change in school performance
- Fearfulness for self, or for loved ones

Talking with a dying patient

- Talk about death - feelings and fears
- Manage symptoms or refer to someone who can
- Checking supports
- How and where do they want to die?
- Discuss enduring power of attorney and getting affairs in order

11: Alcohol - Assessment

Background

- Excess consumption:
  - Hazardous drinking = prolonged excessive consumption with no problems (but high risk).
  - Problem drinking = problems caused by alcohol regardless of the level of consumption.
  - Alcohol abuse = drinking is causing problems (either medical or social) but no signs of dependence
  - Alcohol dependence = features of alcohol addiction:
    - Tolerance and withdrawal
    - Physical: signs and symptoms related to specific organ pathology
    - Psychosocial: accidents, assault, neglecting work or family, trouble with the law.
  - Difference between abuse and dependence: dependence shows signs of tolerance and withdrawal
- Intoxication
  - Effects depend on level of tolerance, but a rough guide for a normal person is:
    - talkative, emotional lability >0-50mg/dL
    - judgment affected, dysarthria, ataxia, nystagmus, N+V >100mg/dL
    - drowsiness, coma >200mg/dL
    - respiratory depression, death >350mg/dL
  - Withdrawal: ↑pulse, ↓BP, tremor, seizures, hallucinations (visual or tactile)

History

- How much do you drink (make a suggestion that’s higher than the amount they’re likely to drink)
- Drinking situation (looking for stereotypical drinking):
  - When
  - Who with
  - What do you drink
• CAGE Screening questionnaire (positive response to 2 questions is suggestive, 3 or more
>99% specificity for alcoholism):
  • Ever tried to cut down
  • Ever been annoyed by others telling you you drink too much
  • Ever felt guilty about the amount you drink
  • Do you need an eye-opener to get going
• Looking differentiating abuse from dependence:
  • Can you drink more than you used to without getting drunk
  • What happens if you stop drinking (any withdrawal symptoms)
• Check for comorbidity (very common):
  • Do you use any other drugs, smoke, etc
  • Any psychiatric illnesses:
    • How have you been feeling in yourself (ie mood)
    • Do you get anxious a lot
    • Ever hear voices that others don’t hear
    • Do you gamble – how much?
• Check for medical consequences of alcohol:
  • Liver disease: Jaundice, itching
  • Heart problems
  • Anaemia (due to alcohol toxicity and poor nutrition)
  • Injury
• Family history
• Social history:
  • Impact on function: relationships, occupation, encounters with the law, finances
  • Living situation, social supports, etc

Investigations
• Blood alcohol (legal limit = 80mg/dL (17.4mmol/L) or 400ug/L of breath). Levels fall by 15-20mg/100ml(dL)/hr (3-4mmol/L/hr))
• FBC – macrocytic anaemia, ↓platelets
• Electrolytes and creatinine
• LFTs – raised GGT, AST and ALT (AST often > ALT), also glucose, INR, albumin
• INR and albumin
• Glucose
• B12 and folate,
• Urine drug screen for other drugs (esp benzos)

12: Alcohol - Intervention

Background to Education
• Making the change:
  • Success of long-term treatment depends on patient's willingness to change.
  • Stages: precontemplation → contemplation → action → maintenance → relapse
• Aims for different drinkers:
  • Hazardous drinkers:
    • Point out excessive consumption
    • Individualise future risks
    • Give clear advice on limits of consumption per week and/or day and ways to cut down
  • Problem drinker:
    • Motivate patient to change
    • Get patient to express pros and cons of cutting down
    • Agree on limits of consumption and ways to cut down.
  • Dependent drinkers - abstinence is the goal, which usually requires referral.

Management plan for early alcohol problems
• Inform patient about risks of drinking, and the damage they’ve already done to their body
- **Listen** carefully to their reactions. They will need to ventilate their feelings and may respond defensively.
- **Outline benefits** for reducing drinking.
- **Set goals** for alcohol consumption that you both agree on.
- **Set strategies** to keep below upper safe limit:
  - Quench thirst with non-alcoholic drinks before having alcohol.
  - Have first alcoholic drink after meal.
  - Low alcohol beer.
  - Have a physical workout when bored or stressed.
  - Explore new interests.
  - Avoid situations where alcohol consumed (eg parties).
- **Evaluate progress** by having patients monitor drinking using a diary.

**Long term intervention/Education**

- Explain:
  - Drinking too much is common, but causes lots of ill health and other consequences (jobs, relationship, law, etc), (20-30% of Kiwis misuse alcohol, but 40% of working males do not drink anything)
  - ALAC guidelines:
    - Men: 21 standard drinks per week/6 in one sitting
    - Women: 14/4
    - Standard drink: small wine, single nip of spirits, 1 can of beer
  - How much do they drink compared to the above?
  - Invite their comment.
  - Advise to drink within ALAC guidelines.
  - Suggest drinking behaviour changes:
    - drinking at slowest persons pace,
    - not buying a drink when it is your round,
    - taking small sips, put glass down when not drinking,
    - non-alcoholic drinks/drink more water,
    - find other activities to enjoy not involving alcohol.
  - Alcohol problems are family problems - how has the family being affected? Do some of them also have a problem? Can you enlist their support to help with treatment?
  - Medical treatment:
    - Be alert for co-existing drug dependence and psych disorders esp depression (may be primary or resolve with treatment of dependence)
    - Drugs: naltrexone to ↓ craving. Disulfiram (antabuse)
  - Always set an immediate goal with at patient at the end of an OSCE station and arrange follow-up.


**137: Alcohol Withdrawal**

**Immediate emergency intervention**

- Not all problems in alcoholics are a direct result of alcohol.
- If unconscious, remember ABC (ie put in recovery position).
- Thiamine (B1) should always be given to those suspected of suffering from the affects of alcohol or withdrawal. This should also always precede glucose infusion.
- Immediate treatment of organ pathology eg. hepatic encephalopathy, GI bleed.

**Withdrawal symptoms**

- Agitation.
- Prominent tremor.
- Sweating.
- Insomnia.
- Seizures.
- Delirium tremens

**Acute withdrawal**
- Diazepam 10-20mg orally every 2 hours (up to 120mg daily)
- Haloperidol 2.5-10mg PO or IM for hallucinations
- Na valproate (or carbamazepine) for seizures
- B-blockers to improve vital signs and tremor
- Thiamine 100mg IM or IV daily for 2-5 days then 100mg daily (oral)
- Vit B supplement (oral or IM) daily

**Delirium tremens**
- Clinical
  - May be precipitated by intercurrent illness or trauma
  - 1-5 days after withdrawal
  - Disorientation, agitation
  - Clouding of consciousness
  - Marked tremor
  - Visual hallucinations (eg pink elephants)
  - Sweating, tachycardia, pyrexia
  - Signs of dehydration
- Treatment
  - Correct fluid and electrolyte imbalance
  - Treat any systemic infection
  - Thiamine 100mg IM or IV daily for 2-5 days then 100mg daily (oral)
  - Diazepam 5mg by slow IV injection (over several minutes every 1/2 hour until symptoms subside OR Diazepam 10-20mg orally every 2 hours (up to 120mg daily)
  - If psychotic symptoms: haloperidol 2.5mg –5 mg (oral) bd
  - Phenytoin for seizures

- Ref: Murtagh, General Practice 2nd Edition, Chapter 106, pp 1029 - 1035

**138, 141, 142 & 143: Overview of Anxiety Disorders**

**General Screening Questions**
- Somatic symptoms: palpitations, fast breathing, tremour, etc
- Irritability
- Panic
- Worries
- Concentration
- Sleep – typically hard to get off to sleep (cf depression – wake early)

**Agoraphobia**
- Anxiety about being in places where escape may be embarrassing, or help is not available, in the event of having an unexpected or situationally predisposed panic attack
- Questions: Do you avoid going out? Any places you avoid for fear of a panic attack
- Situations are
  - Avoided
  - Endured with marked distress, or anxiety about having a Panic Attack
  - Require the presence of a companion
- Anxiety or phobic avoid avoidance not better accounted for by another mental disorder (eg Social Phobia, Specific phobia, OCD, PTSD, separation anxiety)
- Treatment
  - Behavioural and cognitive behavioural therapy
  - Pharmacological: propranolol, fluoxetine, etc

**Panic Attack**
- Sudden onset of intense apprehension, fearfulness, or terror, often associated with apprehension, fearfulness or terror.
• Associated symptoms: SOB, palpitations, chest pain/discomfort, choking or smothering sensations, etc…
• Classified as: unexpected; situationally bound (cued); situationally predisposed

Panic disorder with agoraphobia; Panic Disorder without agoraphobia
• Recurrent unexpected panic attacks
• Questions: Ever had a panic attack? Do you avoid situations?
• Persistent worry about having additional attacks AND/OR worry about the implications or consequences of an attack AND/OR a significant change in behaviour related to attacks
• Not due to substance or a general medical condition.
• Not accounted for by another mental disorder (eg social phobia)
• +/- agoraphobia
• Rx: CBT, TCAs, breathing exercises

Phobias
• Specific: significant anxiety provoked by exposure to a specific feared object or situation, often leading to avoidance behaviour
• Social: significant anxiety provoked by exposure to certain types of social or performance situations, often leading to avoidance behaviour
• Patient must realise these fears are unreasonable or excessive, and must interfere significantly with patient’s life
• Rx: Behavioural and CBT (gradually exposure them to phobic stimulus)

OCD
• Questions: Any rituals which you must do often? Even had a thought in your head that you can’t get rid of?
• Obsessions (which cause marked anxiety and/or distress)
• Compulsions (which serve to neutralise anxiety)
• Person must realise that the obsessional thoughts are a product of their own mind
• Person must realise that the obsessions and compulsions are excessive or unreasonable (NB this does not apply to children)
• Rx: TCAs or SSRIs

PTSD
• Questions: Since it happened, have you been troubled by intrusive memories, nightmares, or have you avoided things connected with it?
• Re-experiencing an extremely traumatic event
• Symptoms of increased arousal and numbing of general responsiveness
• Avoidance of stimuli associated with trauma

GAD
• Questions: do you worry about things most other people don’t worry about?
• At least 6 months of persistent and excessive anxiety and worry
• Finds it difficult to control worry
• Associated symptoms: restlessness, easily fatigued, difficulty concentrating or mind going blank, irritability, muscle tension, sleep disturbance

Also
• Acute stress disorder
• Substance induced anxiety disorder
• Anxiety disorder due to general medication condition
• Anxiety disorder NOS

General Treatment
• ALWAYS TRY NON-PHARMACOLOGICAL TREATMENT FIRST (eg CBT, relaxation techniques, meditations, etc)
• Drugs: anxiolytics (eg benzo’s) BEWARE ADDICTION. Antidepressants for OCD and GAD and panic disorder if severe
144: Hallucinations

Differential Hierarchy
- Delirium or Substance induced psychosis
- Schizophrenia, Mania, Depression

Schizophrenia
- History questions:
  - Do you hear people speaking when there is no one around?
  - Do you have thoughts or beliefs that other might find strange?
  - Do you feel if someone is trying to hurt you?
- 1% prevalence
- +ive symptoms
  - Hallucinations
  - Delusions
  - Thought disorder
  - Bizarre and disorganised behaviour
- -ive symptoms
  - Deficiency in mental function
  - Alogia
  - Affective flattening
  - Anhedonia/asociality
  - Attentional impairment
- >6mths
- Associated symptoms: suicide, lack of insight, substance abuse, depression, neurological soft signs, EPS
- Subtypes: Paranoid; Disorganised; Catatonic; Undifferentiated; Residual

Schizophr Protect
- Same as schizophrenia expect symptoms <6mths

Schizoaffective Disorder
- Mood episode and active-phase form of schizophrenia occur together
- These were preceded or were followed by at least 2 wks of delusions or hallucinations without prominent mood disorder

Delusional Disorder
- Non-bizarre delusions of at least 1 month
- Criteria for schizophrenia has never been met.
- Apart from impact of delusion, functioning is not markedly impaired and behaviour is not obviously odd or bizarre
- If mood episodes have occurred with delusions, their relative period has been brief

139 & 145: Mood Disorders

Mood swings differential diagnosis
- Mood disorder
- Other psych disorders: substance abuse, anxiety, eating disorder, adjustment disorder, personality disorder, PTSD
- Dementia (memory loss a key differentiating feature)
- PMS
- Hypothyroidism
- Drugs: steroids, β blockers
- Grief
- Tiredness - any cause

Major Depressive Episode
- History Questions
  - How have you been feeling in yourself? Dad, blue, down, black?
- Do you still enjoy things you used to enjoy?
- 2wk period with depressed mood or loss of interested or pleasure
- Five or more of
  - Weight loss
  - Insomnia or hypersomnia
  - Decreased concentrated
  - Fatigue
  - Feelings of worthlessness or guilt
  - Recurrent thoughts of death
- Symptoms not better accounted for by bereavement
- Not due to substance or general medical condition.

**Manic Episode**
- History questions:
  - Have you felt especially good about yourself or felt you were special?
  - Have you been speaking more than usual, spending more than usual
  - Have you needed less sleep
- Distinct period of abnormally and persistently elevated mood of >1 week
- Three of more of
  - Inflated self-esteem or grandiosity
  - Decreased sleep
  - More talkative
  - Flight of ideas
  - Easily distracted
  - Increased goal directed activity
  - Increased involvement in pleasurable activities that have potential for painful consequences (eg unrestrained buying sprees)
- Causes marked social, occupational and relationship impairment.

**Bipolar I**
- **One or more Manic Episodes or Mixed Episodes**
- Often have also had one or more Major Depressive Episodes
- Not due to substance or general medical condition
- Not better accounted for by another medical condition (eg Psychotic disorders)

**Bipolar II**
- One or more Major Depressive Episodes, which are…
- Accompanied by at least one Hypomanic episode
- **Never been a Manic or Mixed Episode**
- Not due to substance or general medical condition
- Not better accounted for by another medical condition (eg Psychotic disorders)

**Treatment**
- Depression
  - Education and reassurance (most people do get better)
  - Lifestyle: regular sleep, reduce alcohol, plenty of exercise
  - Psychological treatment (eg CBT)
  - Anti-depressants: SSRI’s best
  - ECT
  - Admit if: social circumstances, high suicide risk, isolation
- Mania
  - May need sedation (eg benzodiazepine)
  - Check compliance, U&E, ECG, and T4, then….
  - Give Lithium carbonate (MUST TITRATE DOSE & BEWARE TOXICITY)

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**Suicide Assessment**
- Not a formal topic – but a given!
- History of Suicide attempt:
  - Time sequence
• What had they tried to do (get details, get dose of drugs, did they take them all)
• Drinking/drugs before hand
• Was attempt impulsive or planned
• Did you make any final arrangements (eg leave a note, make a will)
• Precipitation events (‘What was the final straw’) eg employment, relationships, bereavement
• Did you want to die – or just want people to know how bad it is for you
• Do you feel remorse, glad to be alive now
• Are you hopeful
• How do you feel about accepting help
• Previous suicide attempts:
  • Same questions as above for each attempt
  • What stopped you going through with it
  • What happened afterwards
• Psychiatric assessment:
  • Depression: mood, appetite, sleep, concentration
  • Drug and alcohol use
  • Past psychiatric history
• Predisposing factors:
  • Family history: suicide or psychiatric illness
  • Suicide exposure
  • Other illness, especially pain
  • Social situation: living alone, isolated, unemployed
• Protective factors:
  • Social supports
  • Responsibility for family
  • Hopefulness

32: Discuss/start mood stabilisers

General principles
• Check other medical conditions and medications (incl. eg St John’s Wort)
• Have they had antidepressants before: if so how did they find them?
• Information about side effects. Don’t make assumptions about what people want – ask them
• SE’s begin when drug is started - therapeutic effect takes about 3-4 weeks
• Make sure adjunct treatments (eg psychological treatments) are considered
• Monitor closely to ensure that SE’s are tolerable and to check compliance
• Adjust dose if necessary: want maximum effect for minimum side-effects
• Continue for 9mths for first depressive episode, 3yrs for subsequent episodes
• Taper off treatment

Tricyclic Antidepressants - still initial drug of choice. 60-70% response rate.
• Use in pregnancy, severe or melancholic features
• Build up dose over 10 days; maintain this if SE’s allow for at least six weeks month before deciding if effective or not.
• SE’s
  • Convulsions (dose related), arrhythmias, heart block
  • Anticholinergic - dry mouth, blurred vision, constipation, urinary retention, drowsiness, sweating (explain that these effects will diminish with time, until then do advise against driving, operating machinery etc.)
  • Hepatitis and agranulocytosis - can occur up to 3 months after TCA’s are started. (Do monthly FBC’s during this period)
  • Sedation - take at night
  • Sexual dysfunction
• Interactions
  • The pill may ↓ effect of TCA’s
  • SE’s may be worse if phenothiazines are used concurrently
  • The effect of some anti hypertensives may be reduced
Contraindications

- When there is substantial risk of suicide by overdose
- Coronary artery disease, serious arrhythmias. Ideally someone aged > 45 should have an ECG before starting TCA’s
- Glaucoma or prostatism
- Elderly patients at ↑ risk of postural hypotension
- Where the person needs to drive or operate machinery
- Inability to moderate alcohol or other drug intake.
- When person cannot tolerate the side effects

SSRIs

- Use in adolescents
- No convincing evidence that SSRIs are more effective than TCA’s - just different SE’s
- Take 3-4 weeks to start working
- Less toxic in over dose than TCA’s
- Less sedating than TCA’s
- 30 times the price of TCA’s
- SE’s
  - Nausea and GI disturbances
  - Sexual dysfunction (anorgasmia)
  - Insomnia/ increased arousal especially in first weeks - take in the morning
  - Dry mouth, blurred vision
  - Seizures
- Contraindications
  - Uncertainty about teratogenicity means pregnant or breastfeeding women shouldn’t take SSRIs
  - Insomnia
  - Previous intolerance of SSRIs
- NB - always make patients aware that antidepressants should be stopped gradually

Lithium treatment

- Takes 7-14 days to take effect
- SE’s: upset stomach, diarrhoea (usually settle after 2-3 weeks), weight gain, mild difficulty concentrating, ↑thirst, metallic taste in mouth
- Lithium toxicity, serum lithium levels need to be checked every 5-7 days at the beginning of treatment. Can be caused by dehydration, UTI’s, gastroenteritis. Early symptoms: nausea, vomiting, diarrhoea, unsteadiness, forgetfulness/mild confusion.

- Be cautious of starting antidepressants in the depressive phase of bipolar - may cause mania
- Lithium takes 7-14 days to take affect so is no good in treating acute mania. Antipsychotic medication may be more appropriate
Abdominal and Gastro-Intestinal

100: Abdominal Exam

- Introduce yourself. Wash your hands. Lay the patient flat with one pillow under their head.

*General appearance*
- Consciousness, comfort colour, posture
- Jaundice, pigmentation (eg haemochromatosis), xanthomata.
- Weight and wasting (weigh them). Mental state: hepatic encephalopathy.
- TPN/special drinks, IV lines, oxygen etc

*Hands:*
- Nails: Leuconychia, clubbing. Fingers: Dupuytren’s contracture. Xanthomata
- Palms: Palmar erythema, anaemia.
- Arthropathy. Hepatic flap (extend wrists and separate fingers for 15secs)

*Arms:*
- Brusing, scratch marks, spider naevi, muscle wasting
- Blood pressure
- Axillae: acanthosis nigrans (GI Ca, lymphoma, endocrinopathies), lymphadenopathy.

*Face:*
- Eyes: xanthomata, jaundice, anaemia, episcleritis/iritis, Kayser-Fleischer rings (occur very late).
- Mouth: breath (alcohol, fetor hepaticus), angular stomatitis (B6, B12, folate and Fe def), Peutz-Jeghers Syndrome, hereditary haemorrhagic telangiectasia, ulceration or candida.
- Tongue: coating (esp smokers), leucoplasia, glossitis, macroglossia.
- Parotids (swell due to fatty infiltration with ↑ alcohol, or tumour)

*Neck and Chest:*
- Cervical lymph nodes and supraclavicular (esp on left in gastric ca).
- Gynaecomastia (liver disease, alcohol effect on Leydig cells, drugs (spironolactone, digoxin)), spider naevi (>1 is likely to be abnormal), body hair (loss of in men)

*Inspection*
- Colour, scars, swelling, wasting deformity, movement (pulsation)
- Scars, veins (check flow direction), striae (ascites, preg, weight loss, Cushing’s) Skin lesions/pigmentation (eg Shingles causes strange pains until it erupts)
- Distension
- Hernias (umbilical, inguinal, femoral). Visible lumps/organs
- Abdomen moves with respiration (look from side on to view asymmetry ⇒ ? mass)
- Pulsations: abdominal aorta, pulsatile liver.

*Palpation and Percussion*
- Warm hands. Ask if patient has abdo pain. Bend knees up to relax muscles if necessary.
- Light palpation: look at face. Feel for tenderness/peritonism, lumps. If tense can use their hand.
- Deep palpation for masses. Aorta, uterus, bladder (dullness on suprapubic percussion if enlarged).
- Percuss then palpate liver (span, hard/firm/soft, regular/irregular, tender/non-tender, pulsatile/non-pulsatile). Know Murphy’s sign (cholecystitis) and Courvoisier’s law (enlarged GB).
- Percuss then palpate spleen: enlarged 3-4X to palpate. Start inf to the umbilicus. Use both hands.
- Ballot kidneys (tumour or obstructed). Be able to distinguish between spleen and kidney.
- Percuss for shifting dullness in suspected ascites.

*Auscultation*
- Bowel sounds: just below umbilicus - present, absent or tinkling
• Liver, spleen, renal, epigastrium (superior mesenteric a.) areas for rubs and bruits

Other
• Inguinal lymph nodes, hernial orifices, testicular exam, rectal exam.
• Legs: bruising, muscle wasting, oedema (check sacral as well)

30: Difficulty Swallowing

• = Dysphagia

History & Differential diagnosis
• Did you have difficulty swallowing fluids and liquids from the start?
  • Yes: Think motility disorders (achalasia, neuro. causes)
  • No: Suspect a stricture (benign or malignant)
• Where does they food get stuck?
• Is it difficult to make the swallowing movement?
  • Yes: Suspect bulbar palsy, especially if he coughs on swallowing
• Is swallowing painful?
  • Yes: suspect a malignant stricture or oesophagitis
• Is the dysphagia intermittent or is it constant and getting worse?
  • Intermittent: Suspect oesophageal spasm
  • Constant and getting worse: Suspect a malignant stricture
• Does the neck bulge or gurgle on drinking?
  • Yes: Suspect a pharyngeal pouch (food may be regurgitated)
• Other history questions:
  • Change in diet
  • Weight loss
  • Past medical and family history
  • Smoking and alcohol

Red Flags
• Hoarseness
• Pain waking at night
• Weight loss
• Early Satiety
• Abrupt onset
• ↑Severity
• Symptoms for 1st time over 45 years or with sudden onset
• Vomiting blood

Causes
• Mechanical block
  • Malignant stricture: Gastric, Oesophageal or Pharyngeal cancer
  • Benign stricture
    • Oesophageal web or ring
    • Peptic stricture
  • Extrinsic pressure
    • Lung cancer
    • Retrosternal goitre
    • Mediastinal cancers
• Motility disorders
  • Achalasia
  • Diffuse oesophageal spasm
  • Systemic sclerosis/ CREST syndrome
  • Bulbar palsy
  • Pseudobulbar palsy
  • Myasthenia gravis
  • Syringomyelia
• Others: Oesophagitis – infection or reflux
**Investigations**
- Imaging: Barium swallow, endoscopy with biopsy, oesophageal manometry
- Blood: FBC, ESR
- ENT opinion

Ref: OHCM 4th Edition, pg 492

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<td>Mallory-Weiss tear</td>
<td>Epistaxis</td>
</tr>
<tr>
<td>Oesophageal varices</td>
<td>Oesophagitis</td>
</tr>
<tr>
<td>Portal hypertensive gastropathy</td>
<td>Oesophageal or gastric malignancy</td>
</tr>
<tr>
<td>Gastritis</td>
<td>Aorticoduodenal fistula</td>
</tr>
<tr>
<td>Gastric or duodenal ulcer</td>
<td>Haemobilia</td>
</tr>
<tr>
<td>Drugs - anticoagulants, NSAIDs, steroids, thrombolytics</td>
<td>Angiodysplasia, Peutz-Jeghers syndrome</td>
</tr>
<tr>
<td></td>
<td>Bleeding disorders</td>
</tr>
</tbody>
</table>

**History**
- Vomit
  - How much
  - What did it look like
  - Any black dots like coffee grounds or any blood clots
  - Were you vomiting normal vomit before the blood appeared.
- Bowel
  - Have you passed a motion and if so what colour
  - Motions black or unusual in any way
- Drugs/alcohol
  - What drugs have you been taking
  - Have you been taking aspirin or tablets for arthritis or back pain
  - How much alcohol do you drink
  - Illicit drugs, needle sharing
- Have you had any indigestion, heartburn or stomach pains recently
- Have you had any previous operations on your stomach for a peptic ulcer
- Risk factors for Hep B and C: Sexually transmitted infections, needle sharing, tattoos, body piercing, health care worker, transfusions or organ transplants

**Assessment**
- Assess severity of blood loss, check for shock and begin resuscitation
- Notify rostered specialist team
- Take history
- Examination: signs of liver disease, telangiectasia; or purpurae; jaundice
- Investigations: FBC, U&Es, LFTs, coags, ABG, ECG, CXR

**Resuscitation**
- Normal saline up to 2 litres
- Colloids - 4% normal serum albumin (NSA) or dextrose
- Blood - indications = postural hypotension, Hb<100g/L or shock (=clinically profoundly hypovolaemic and systolic BP<100mmHg)

**Suspected variceal bleeding**
- Consider Octreotide 25-50ug SC stat then IV at 25ug/hr and/or balloon tamponade.
- Note: rebleed carries significant mortality

**Admit to ICU for urgent endoscopy if:**
- Major haemorrhage = P>100 or systolic BP<100 or postural hypotension (↓15mmHg)
- >60 years old
- Known or suspected liver disease
- Rebleed
Management

- Refer to OHCM pg 495 for a good management flow diagram, and Rockall risk-scoring system for GI bleeds.


Chronic or recurrent pain or discomfort centred in the upper abdomen
- Need to exclude gastro-oesophageal reflux, oesophageal or gastric cancer, peptic ulcer
- Ask about NSAID use
- Refer for endoscopy if:
  - Onset of symptoms at age > 45y
  - Any alarm symptoms
- Alarm symptoms:
  1. Nausea and vomiting
  2. Weight loss
  3. Dysphagia
  4. Hoarseness
  5. Nocturnal cough or choking
  6. Asthma
  7. Bleeding or anaemia (do a FBC)
  8. Non-cardiac chest pain
  9. Early satiety
  10. Wakes patient at night

Functional (non-ulcer) dyspepsia is the cause in up to 60%. Patients tend to have delayed gastric emptying and increased sensitivity however prokinetic agents, acid reducing agents and H pylori eradication usually have limited results. Compared with healthy subjects these people have higher scores for anxiety and depression but the significance of this is uncertain

Differential:
- Reflux
- Ulcer (gastric usually worse with food, duodenal usually better with food and wakes patient at night)
- Gall-bladder
- Chronic pancreatitis
- Pancreatic cancer
- Cancer

Management:
- Educate patient about the problem
- Lifestyle changes: avoid trigger foods and large meals, lose weight, stop smoking, avoid noxious medicines (NSAIDs, calcium antagonists, anticholinergics TCAs), reduce stress
- Medications
  - Antacids: after every meal and before bed
  - Prokinetics (eg cisapride): improve oesophageal clearance and gastric emptying
  - H2 receptor antagonists and proton pump inhibitors

Abdominal Distension

Specific Causes
- Air:
  - GI obstruction (including faecal)
  - Aerography (air swallowing)
- Ascites
  - Malignancy (of any intra-abdominal organ)
  - Hypo-proteinenaemia (eg nephrotic)
  - Right heart failure
  - Portal hypertension
- Solid Mass
  - Malignancy (any intra-abdominal organ)
  - Lymph nodes
  - AAA
- Cysts (renal, pancreatic)
- Pelvic Masses
  - Bladder: full of cancer
  - Fibroids
  - Foetus
  - Ovarian cyst, ovarian cancer
  - Uterine cancer

Differentiating between causes
- Air: Resonant on percussion
- Ascites: Shifting dullness, Fluid thrill
- Pelvic Masses: Can’t get below it (lower margin can’t be defined)

Investigations
- Ultrasound
- Aspirate any fluid for cytology, culture, and protein estimation using 18G needles in RIF
- AXR


2: Abdominal Mass

Descriptive features
- Site, size, surface
- Tenderness
- Edge
- Consistency
- Mobility, movement, able to get above mass
- Pulsatile

Sites
- RIF
  - Appendix mass, appendix abscess
  - Caecal carcinoma, caecal distension due to obstruction
  - Pelvic mass (foetus, fibroids, bladder, ovarian cyst/malignancy)
  - Crohn’s disease
  - Hernia
  - Carcinoid tumour
  - Intussusception
  - TB mass (iliocaecal TB)
  - Amoebic abscess
  - Antimycosis (refer to pg 223 OHCM)
  - Kidney malformation, kidney transplant
  - Tumour in undescended testes
- LIF
  - Faeces (NB can be indented)
  - Carcinoma of sigmoid or descending colon
  - Diverticular abscess
  - Ovarian tumour or cyst
  - Psous abscess
  - Hernia
  - Transplanted kidney
- Upper Abdomen
  - Retroperitoneal lymphadenopathy (eg lymphoma, teratoma)
  - Hepatomegaly:
    - smooth (hepatitis, early ETOH cirrhosis, tricuspid incompetence
    - irregular (1st hepatic cancer or secondaries)
  - AAA
  - Spleen (broad causes: infective, haematological, neoplastic)
- Carcinoma of transverse colon
- Gastric dilatation (eg pyloric stenosis)
- Omental mass (eg metastatic tumour)
- Stomach carcinoma
- Kidney
- Pancreas (pseudocyst or tumour)

**Investigation of lump**
- Imaging: Ultrasound, CT, CXR, AXR, IVU.
- Bloods: FBC, U&E, LFT, ESR, proteins, Ca
- Mantoux test
- Tissue biopsy


## 24: Constipation

### History
- What do they mean by constipation (e.g. harder to pass, pass less often)?
- Acute or chronic?
- Bowel history: changes, colour, blood, mucus, pain, etc
- Diet
- General: weight loss, appetite, sleep, energy, fever, night sweats
- Signs of hypothyroidism (cold intolerance, etc)

### Causes
- Poor habit, poor diet, dehydration, lack of privacy (NB hospital)
- More serious common causes:
  - Extrinsic/intrinsic cancer
  - Kids: Hirschsprung’s
  - Drugs: morphine, anticholinergic, TCAs
  - Hypothyroidism
  - Calcium metabolism disturbances

### Investigation
- In older patients it is important to rule out colorectal carcinoma (especially if recent onset or associated with rectal bleeding, mucous discharge, or tenesmus)
- Rectal examination
- AXR

### Treatment
- Treat cause (see above) and exclude bowel obstruction
- Encourage mobilization
- Advise diet rich in fibre with adequate fluid intake
- Bulk producers (e.g. Bran. Isogel, Metamucil)
- Short term Rx: Stool softeners (e.g. liquid paraffin – don’t use regularly as can cause seepage), coloxyl
- Osmotic agents (e.g. Epsom salts – take with plenty of water, lactulose)
- Stimulants (e.g. Senna – should not be on stimulants long term as bowel becomes habituated by them, dulcolax = bisacodyl)
- Phosphate enemas and glycerine suppositories

## 29: Diarrhoea

### History (main areas to focus on)
- What does the patient mean when by diarrhoea? How often?
- Acute or chronic?
  - Acute:
- INFECTION + ask about travel abroad, anyone else infected, unusual diet
- UC may present acutely
- Chronic:
  - IBS: chronic, intermittent diarrhoea alternating constipation
  - Cancer: Weight loss, anorexia, anaemia, diarrhoea at night
- Blood, mucous or pus?
  - Bloody diarrhoea see below
  - IBS = mucous but “never” blood
  - Polyps = blood or mucous or both
  - Pus suggested IBD or diverticulitis
- Large or small bowel?
  - Watery stool +/- mucous or blood or both, pelvic pain relieved by defecation +/- tenesmus, urgency \(ightarrow\) LARGE BOWEL
  - Periumbilical pain not relieved by defecation, watery or pale, fatty, smelly stools (steatorrhea) \(\rightarrow\) SMALL BOWEL
- Non GI causes: Thyrotoxicosis, autonomic neuropathy, drugs…
- Non: GI symptoms:
  - Also ask about liver pain, malabsorption (floating stools)
  - Associated with IBD: arthritis, oral lesions, weight loss, eyes, rashes

**Bloody diarrhoea**
- Infection (dysentery): Campy, Shigella, Salmonella, E. coli
- IBD
- Other: Colorectal cancer, pseudomembranous and ischaemic colitis, diverticulitis.

**Investigations**
- PR (rectal mass, impacted faeces)
- Stool: FOB, FBC (anaemia), stool microscopy, (faecal fats),
- Imaging: Rigid sigmoidoscopy and biopsies, barium enema, colonoscopy

**Management**
- Treat cause
- Give clear fluids PO or 0.9% saline
- If dehydrated or elderly check U&E
- Refer to OHCM pg 487 for algorithm of treatment of infectious diarrhoea.


---

**58: Melena and Rectal Bleeding**


**Melena**
- Implies bleeding above proximal to distal ileum. Typical of upper GI bleeds. Refer to Question 44, page 86

**History**
- Describe bowel motion: black, bright red blood, pus
- Time: onset, frequency, progression
- Where is the blood seen: paper, bowel, stool
- Associated symptoms
  - Pain
  - Diarrhoea or constipation
  - Presence of lumps
  - Pruritis
  - Sensation of urgency or unsatisfied defecation
  - Tenesmus
  - Weight loss
  - Extra-intestinal manifestations of IBD eg arthritis, iritis, oral ulcers, rashes, jaundice
• Past history of haemorrhoid, polyp, cancer
• Drug history: eg warfarin, NSAIDs, antibiotics
• Family history: polyps, bowel cancer, IBD

Differentiating

• Sensation of urgency or unsatisfied defecation
• Pain on passing motion
• Change in bowel habit
• Occult anaemia

Rectal Cause

Haemorrhoid, fissure
Colonic or Rectal Cancer, IBD, IBS
Right Colon Cancer

Examination

• Abdominal
• Anal inspection and PR
• Sigmoidoscopy

### Appearance | Cause
---|---
Bright red blood in toilet separate from faeces | Internal haemorrhoids
Bright red blood on toilet paper | Internal haemorrhoids
Fissure
Anal carcinoma
Pruritis
Anal warts and condylomata
Blood and mucus on underwear | 3rd degree haemorrhoids
4th degree haemorrhoids
Prolapsed rectum
Mucosal prolapse
Prolapsed mucosal polyp
Blood on underwear (no mucus) | Ulcerated perianal haematoma
Anal carcinoma
Blood and mucous mixed with faeces | Colorectal carcinoma
 Proctitis
Colitis, ulcerative colitis
Blood mixed with faeces (no mucus) | Small colorectal polyps
Small colorectal carcinoma
Melaena (black tarry stools) | GI bleeding with long transit time to anus
Torrential haemorrhage (rare) | Diverticular disease
Angiodysplasia
Large volumes of mucus in faeces (little blood) | Villous papilloma or rectum
Villous papilloma of colon
Blood in faeces with menstruation (rare) | Rectal endometriosis
### 128: Abnormal Liver Function

<table>
<thead>
<tr>
<th>LFT</th>
<th>Hepatocellular hepatitis</th>
<th>Haemolytic jaundice</th>
<th>Obstruction</th>
<th>Gilbert’s disease</th>
<th>Liver mets/abscess</th>
<th>Alcoholic liver</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bilirubin</td>
<td>↑ to ↑↑↑</td>
<td>↑ (unconj)</td>
<td>↑ to ↑↑↑</td>
<td>↑ up to 50 unconj</td>
<td>↑ to N</td>
<td>↑ to N</td>
</tr>
<tr>
<td>ALP</td>
<td>↑ &lt; 2N</td>
<td>N</td>
<td>↑↑↑ &lt;2N</td>
<td>N</td>
<td>↑↑ to ↑↑↑</td>
<td>↑</td>
</tr>
<tr>
<td>AST</td>
<td>↑↑↑ &gt; 5N</td>
<td>N</td>
<td>N or ↑</td>
<td>N</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>GGT</td>
<td>N or ↑</td>
<td>N</td>
<td>↑↑</td>
<td>N</td>
<td>↑</td>
<td>↑↑</td>
</tr>
<tr>
<td>Albumin</td>
<td>N or ↓</td>
<td>N</td>
<td>N</td>
<td>N to ↓</td>
<td>N to ↓↓</td>
<td>N to ↑</td>
</tr>
<tr>
<td>Globulin</td>
<td>N or ↑</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N to ↓↓</td>
<td>N to ↑</td>
</tr>
</tbody>
</table>

Ref: Murtagh, General Practice 2nd Edition, Chapter 53, pg 537.

### 147: Abdominal X-ray

**GAS, STRIPES, STONE AND BONES**

- **Demographics:** Name, date view, orientation
- **Quality:** Any contrast used? Large enough field of view?

**Gas**
- Bowel gas
  - Gastric, caecal and rectal gas should always be seen
  - Caecum<9cm, colon<5.5cm, small bowel<3cm
- Ectopic gas
  - Bowel wall (pneumotosis) = necrotising enterocolitis or Infective colitis)
  - Peritoneal cavity – perforation
  - Biliary system – ERCP, fistula
  - GU system/retroperitoneal

**Stripes**
- Psoas stripes: May be obscured by retroperitoneal haemorrhage, inflammatory etc
- Flank stripes: Obscured by inflammation
- Visceral outlines (liver, kidney, spleen (in 50%))

**Stones**
- Renal, gall, bowel
- Calcification of aorta, renal artery, pancreas

**Bones:**
- Examine ribs, spine, pelvis and femoral heads for:
  - Trauma
  - Metastases
  - Metabolic bone disease Bones
- Localised peritoneal inflammation may cause local ileus = sentinel loop:

<table>
<thead>
<tr>
<th>Cholecystitis</th>
<th>Appendicitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pancreatitis</td>
<td>Diverticulitis</td>
</tr>
</tbody>
</table>

### 48: Barium enema

**Indications**
- Suspected bowel pathology or obstruction (?change in bowel habit, rectal bleeding)

**Complications**
- Perforation
Techniques
- Cleansing colon with low fibre diet for three days and well prepared with laxatives and washouts
- Warn patient it is unpleasant
- Rotating the patient
- Double contrast technique: superior for examining mucosa in greater detail (i.e. polyps, inflammation)
- Single contrast enema accurate for all other problems (i.e. diverticulitis, appendicitis, extrinsic abdominal mass
- If fistula suspected, use Gastrogaffin as contrast of choice to prevent contaminating operating field with barium


Indications
- Barium swallow: Investigate dysphagia (if endoscopy not available, or motility needs to be observed)
- Barium meal: Outline oesophageal, gastric and duodenal pathology

Techniques
- Barium swallowed in upright and prone positions
- Reflux of barium from stomach into oesophagus is demonstrated with the patient tipped head down.
- Swallowing bread with the barium (to add bulk) is sometimes useful in a case of dysphagia
Musculo-Skeletal

101: Back Examination

- WALK, LOOK, FEEL, MOVE, MEASURE, COMPARE, X-RAY

Walk
- Quick way to check neurological status of legs.
- Walk normally
- Walk on toes (S1, S2)
- Walk on heels (L4, L5)
- ?Need do full neurological examination of lower limbs.

Look
- Skin: Scars, pigmentation, abnormal hair, unusual skin creases
- Shape and posture: scoliosis, kyphosis, lumbar lordosis

Feel
- Palpate spinous processes and interspinous ligaments for any tenderness, temperature prominence, or a “step”

Move
- Flexion and extension (standing)
- Lateral flexion (standing)
- Rotation (sitting on chair to fixate pelvis)
- Straight leg raising

Joint above and below

Neuro exam of legs and abdo exam

106: Joint Examination - Hip

Hip examination
- Walking: normal, on toes (tests S1), on heels (test L5)
  - Antalgic: bad limb has a short stance phase
  - Broad based: cerebellar, drunk
  - Short leg
  - Foot drop
  - Trendelenburg: weak adductor causes opposite side to sad (look at hip and shoulder alignment)
- Observe from front and do Trendelenberg’s test
- Look (need joint above and below)
  - Skin: Scars, redness
  - Soft tissue: Swelling
  - Muscle: Wasting of quads, hamstrings, abductors, adductors
  - Bone: Deformity
- Feel
  - Exclude other pathology (eg groin – hernias, lymph nodes, or femoral artery aneurysm)
  - Look for tenderness over greater trochanter
- Move and Measure
  - Thomas’ test for fixed flexion deformity
  - Flexion and extension
  - Abduction (0-45°) and adduction (0-30°)
  - Internal rotation (0-25°) and external rotation (0-30°)
- Examine Joint Above And Below
  - Check foot pulses
  - X-ray
106: Joint Examination - Knee

**KNEE**

**Walk**
- Stiff knee gait
- Varus
- Lateral thrust: Posterolateral insufficiency, knee goes posterolaterally, a result of Medial Compartment OA.
- Squat on their haunches:
  - Stimulates pain in the front then it is an anterior problem
  - In the popliteal fossa → could be a medial meniscal tear.
  - Inspect the popliteal fossa (then you don’t have to get them to roll over on the bed)

**Look**
- Swelling
- Muscle wasting
- Bony deformity
- Arthroscopy scars
- Push their knee down into the bed to test extension and look for muscle wasting in VM

**Feel**
- Feel for effusion (*Meniscal pathology often produces an effusion*)
  - Patellar tap
  - Stroke test
  - Palpate joint line: Tenderness here may indicate a meniscal tear, above or below the joint line meniscus not causing it.

**Move**
- Raise their leg straight as high as they can. (checks extensor mechanism)
- Flex their knee and still keep their thigh pulled into their chest (testing knee flexion). Bring the other leg up with the knee in flexion to compare.

**PCL**
- Feet back down on the bed leaving both their knees in 90° flexion.
  - Look across the two knees for posterior sag, which could indicate a PCL rupture.
  - Stabilise the tibia (sit on their foot)

**ACL**
- Anterior draw to test the ACL + compare with the other side
- Lachman’s test
- McMurray’s test
- Pivot shift test
  - With the knee still in 15° steady it as you pull the leg into valgus, this tests the medial collateral ligament and the ACL. Now Push it into Varus, this tests the lateral collateral ligament
  - Lay the leg flat and repeat with the knee in full extension.

**Collateral Ligaments**
- Valgus stress test
- Verus stress test

**The Patellar-femoral joint**
- Palpate
  - Border
  - Anterior surface
  - Tendon and ligament insertions
  - Posterior surface (by pushing it to one side and then the other)
- Grind or Friction test:
  - Move the patella up and down while pressing it lightly against the femur
• Will cause painful grating if the central portion of the articular cartilage is damaged.
• Patella apprehension test:
  • Press the patella laterally and hold it slightly subluxed → Watch the person’s face and ask them to flex their knee → If they grimace or show signs of pain then the test is positive and is diagnostic of recurrent patellar subluxation or dislocation.

• Look at the direction that the patellar points in
• Have the patient flex and extend at the knee → should follow an inverted J course.

Joint Above and Below
• Check the Hip, the Ankle and the foot pulses.

106: Joint Examination - Shoulder

• LOOK, FEEL, MOVE, MEASURE, COMPARE, X-RAY

Look
• Skin: scars, sinuses (don’t forget to look in axilla)
• Shape
  • Asymmetry of shoulder
  • Winging of scapula
  • Wasting of deltid or short rotators
  • AC dislocation (look from behind)
  • Joint swelling
  • Wasting of pectoral muscles (from front)
• Position: if arm held internally rotated, think posterior dislocation of shoulder

Feel
• Skin: temperature
• Soft tissues: swelling
• Palpation: start with sterno-clavicular joint → follow clavicle laterally to AC joint → onto anterior edge of acromion → around acromion to back of the joint
• Tenderness and crepitus

Move
• Best done actively, with both arms at same to compare
• Abduction and adduction
• Flexion and extension
• External and internal rotation
  • Arms close to body and elbows flexed to 90 degrees, the hands are separated as widely as possible (external) and brought together across the body (internal)
  • External rotation in abduction: clasp fingers behind neck
  • Internal rotation in adduction: reach up back with their fingers
• Test passive movements if active movements are limited
• Note arc of pain

Test power of upper limb
• Remember to test serratus anterior (push up against wall)

X-ray

3: Abnormal Gait

Abnormal gait is due to
• Pain
• Deformity
• Weakness
• Neurological abnormality (eg balance problems, Parkinson’s)

Testing Gait
• Walk normally, then turn around quickly and walk back
• Walk heel-to-toe to exclude midline cerebellar lesion
• Walk on toes (S1 lesion makes this difficult)
• Walk on heels (L4 or L5 lesion causing footdrop makes this difficult)
• Squat then stand up, or sit in low chair and then stand (tests for proximal myopathy)
• Stand erect with feet together and eyes closed

Specific Causes
• Cerebellar: wide base, unstable, can’t walk heel-to-toe steps, eg: posterior fossa tumour, alcohol or phenytoin toxicity
• Extra-pyramidal: Flexed posture, shuffling feet, slow to start, postural instability, eg: Parkinson’s
• Hemiplegia: Foot is plantar flexed and leg is swung in lateral arc
  • Frontal (Apraxic): Shuffling, difficultly getting feet off floor (“magnetic”), eg: Normal pressure hydrocephalus
  • Sensory: Wide base, falls, worse in poor light, decreased joint position and vibration, eg: Tabes dorsalis, cervical spondylosis
  • Spastic: Stiff, narrow base, short paces, circumduction
  • Psychogenic: Wild flinging of arms/legs, usually no falls, or overcautious, “like walking on ice”, eg: Depression
• Proximal myopathy: Waddling gait
• Foot drop: High stepping gait
• Antalgic: Short stance phase to avoid pain
• Trendelenburg: Pelvis sags on affected, non-weight bearing side; trunk moves to opposite side to compensate

Investigations
• Imaging: Spinal X-ray, CT, MRI, CXR (Tb, Ca bronchus)
• Bloods: FBC, ESR, syphilis serology, serum B12, U&E, LFT, PSA, PTT/INR
• Other: LP, EMG, muscle biopsy, serum electrophoresis (myeloma)


5: Abnormal Movements

• See 83: Tremour, page 57

6: Abnormal Posture

Psychiatric
• Retarded depressives: Sit with shoulders hunched, immobile, and with the gaze directed towards the floor.
• Agitated depressives: Tremulous and restless, adjusting their clothing and pacing up and down.

Musculoskeletal
• Depends on bones or muscle groups affected
  • Ie Broken clavicle – patient will sit with arm on affected side supported in the sling position by the other arm.
  • Kyphosis – Patient sits (and stands) with a hunched back.
• Pay particular attention to patient’s hands. Always shake hands with patient and carefully observe their hands for signs of rheumatoid/osteo arthritis etc

Endocrine
• Hypothyroidism – Similar to retarded depressives – No twinkle in their eye.
• Hyperthyroidism – Similar to agitated depressives – frenetic activity, itch, tremor.

Neurological
• Stroke – motionless on one side or one part of the body eg arm hanging limply off the side of the bed.
- Parkinsons Disease – Stoop is characteristic, shuffling gait with poor arm swing. Posture sometimes called Simian to describe the ape like forward flexion, immobility of the arms and lack of facial expression. The patient sits with the trunk bent forward and motionless, without gesture or animation, while the limbs are tremulous.
- Dystonia – May occur after starting neuroleptic medication. Head pulled back, eyes drawn upwards. Treat with anticholinergics (procyclidine 5-10mg iv) (OHCM pg 428)
- See 83: Tremour, page 57

### History

### Swollen ankles:
- Use general pain screen.
- Is it better in the morning

### Symptoms of heart failure:
- SOB on exertion
- SOB on lying down
- Paroxysmal Nocturnal Dyspnoea
- How many pillows
- Cough, sputum
- Chest pain, palpitations

### Symptoms of liver failure:
- Ascites, jaundice, pruritis, fatigue, malaise
- Risk factors for Hep B and Hep C, alcohol
- Symptoms of hypothyroidism: Cold intolerance, weight gain, depression, sleepy, dry hair and skin
- Symptoms of GI malabsorption (→ hypoalbuminaemia): diarrhoea, weight loss, steatorrhoea
- Medical History: include heart risk factors (angina, hypertension, cholesterol, previous MI, stroke, etc)
- Medications
- Family History
- Social History: Smoking and alcohol

### Pitting Bilateral Lower Oedema
- Cardiac: CHF, constrictive pericarditis
- Drugs: Calcium antagonists
- Hepatic: Cirrhosis (due to increased venous pressure or hypoalbuminaemia)
- Renal: Nephrotic syndrome (causing hypo-albuminaemia)
- GI: Malabsorption, starvation, protein-losing enteropathy, causing hypoalbuminaemia
- Beriberi
- Cyclical Oedema

### Pitting Unilateral Oedema
- DVT
- Compression of large vein caused by tumour or lymph node

### Non-pitting Lower Limb Oedema (Implies poor lymphatic drainage)
- Hypothyroidism
- Lymphoedema: Infectious, Malignancy, Congenital, Allergy

Ref: Murtagh, General Practice 2nd Edition, Chapter 70, pp 730 -731

### 13: Ankle Swelling

### 55 & 81: Stiffness & Swelling of joints

- See No 106 Examination of joints

### History
- History of presenting complaint:
  - Use pain screen
• Associated symptoms: morning stiffness, warmth/swelling, locking/giving way, other joints
• Signs of inflammatory disease: fatigue, malaise, night sweats, fever
• Previous injury
• Functional aspects: how far can you walk, run, bend, dressing, etc
• Past medical history
• Medication
• Family History
• Social History: especially activities of daily living, supports, etc

**Bed side differential diagnosis of locomotor system disease:**

- Inflammatory Joint Disease
  - Peripheral, symmetrical, small joint polyarthritis
    - RA
    - Lupus like connective diseases, PMR
  - Asymmetrical, large joint, oligo-arthritis, possibly with inflammatory spinal disease; “seronegative spondarthritides”
    - Psoriatic arthritis
    - Enteropathic arthritis of inflammatory bowel disease
    - Ankylosing Spondylitis
    - Reactive arthritis and Reiter’s disease
  - Acute inflammatory mono or oligo-arthritis
    - Septic arthritis
    - Gout
- Non-inflammatory musculoskeletal disease
  - Osteoarthritis - Weight bearing joints and hands
  - Soft tissue rheumatism or locomotor pain syndromes

**General points**
- Temperature - hot = inflammation/infection
- Texture - hard = subluxation or osteophytes
  - soft = synovitis or synovial effusion
- Osteophytes, loose bodies in joint space, a tense effusion, or pain will limit movement
- Torn ligaments will decrease stability
- Joint swelling after injury
  - Immediately - Haemarthrosis
  - Hours later - Effusion

**Xray appearances**

<table>
<thead>
<tr>
<th>Signs</th>
<th>Rheumatoid</th>
<th>Osteoarthritis</th>
<th>Gout</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uniform joint space narrowing</td>
<td>Localised joint space narrowing</td>
<td>Relative preservation of the joint space and bone density</td>
<td></td>
</tr>
<tr>
<td>Erosions</td>
<td>Subchondral sclerosis</td>
<td>Para-articular erosions</td>
<td></td>
</tr>
<tr>
<td>Periarticular osteoporosis</td>
<td>Subchondral cysts</td>
<td>Asymmetric soft tissue swelling</td>
<td></td>
</tr>
<tr>
<td>Fusiform soft tissue swelling</td>
<td>Osteophytes</td>
<td>Small joints of the feet &amp; hands</td>
<td></td>
</tr>
<tr>
<td>Distribution</td>
<td>Symmetric</td>
<td>Asymmetric</td>
<td></td>
</tr>
<tr>
<td>MCP &amp; PIP</td>
<td>Weight bearing</td>
<td>Weight bearing</td>
<td></td>
</tr>
<tr>
<td>Differential Diagnosis</td>
<td>SLE</td>
<td>CIP &amp; 1st MCP</td>
<td></td>
</tr>
<tr>
<td>Scleroderma</td>
<td>Neuropathic</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2003 OSCE Handbook
Falls

30% of >65yrs fall per year
25% brought to med attention
<5% cause #

Causes (usually multifactorial, < 20% are due to a single medical event)
- Head (stroke)
- Heart (arrhythmia, postural hypotension)
- Vestibula disturbance
- Other eg hypoglycaemia

History
- Determine if the fall was due to a major medical problem: Neuro and CVS ROS
- Determine if any serious injuries were caused by the fall: fractures, head injury
- Assess risk for future falls

Risk Factors
- Medications
  - >= 4 meds
  - psychotropics
- Alcohol
- Medical conditions
  - Cognitive impairment
  - Parkinson’s
  - CVA
  - Arrhythmias → faints
- Functional ability
  - Muscle weakness
  - Arthritis
  - Hx of falls
  - Abnormal balance or gait

Examination
- Postural blood pressure
- Vision and hearing
- Cardiovascular exam - to rule out AS or arrhythmia
- Neurologic exam - looking for evidence of dementia, Parkinson’s, stroke, pay particular attention to strength, co-ordination, evidence of peripheral neuropathy, joint disease and the state of the feet

Management
- Is the fall part of a catastrophic illness that requires diagnosis and management? eg stroke, MI etc
- Has the fall caused any injury that requires active management or further investigation? eg #
- Identification of risk factors eg polypharmacy
- Rehabilitation - this involves provision for active mobilisation of the person after a fall
Prevention
- Number and dose of meds
- OT assessment and therapy
- Physio-gait training
- Transfer skills
- Exercise program

21: Common Fractures (Radiology) and 152: Skeletal Radiographs

- When describing an X-ray in an OSCE stations pretend you are talking to someone over the phone.
- Always check that the film belongs to the right patient.
- The rules of two:
  - 2 views at 90 degrees to each other
  - 2 joints - joint above and below
  - 2 times - if no abnormality detected initially, re-xray 14 days later
  - 2 sides - ie left and right humerus. (esp in children where you aren’t sure where the normal epiphyses are)

Describing a fracture
- Site: Left or right? Which bone is fractured? Where in the bone is it?
- Type: What kind of fracture?
  - Open or Closed?
  - Greenstick - involves only one cortex. Buckle, Bowing.
  - Transverse - night stick #. Force applied at right angles to the long axis of the bone
  - Oblique - eg being hit by a car while leg is in stance phase
  - Spiral - twist on long axis of bone
  - Comminuted - 3 or more fragments of bone
- What are the bone ends doing relative to each other
  - L = Change in Length
  - A = Angulation - distal part in relation to proximal part (medial = varus, laterally = valgus)
  - R = Rotation - distal part in relation to proximal part
  - D = Displacement

153: Isotope Bone Scan

- 99Tcm-MDP (methylene diphosphonate) is the most commonly used radiotracer
- It is given intravenously and is retained in areas with increased osteoblastic activity
- The uptake of the radionucleotide is predominantly dependent on blood flow
- Imaging is done with a gamma camera 3 hours post injection
- Common indications:
  - Paget's
  - Fractures: scaphoid, stress fractures
  - Tumours: metastatic disease, osteoid osteoma
  - Inflammation and infection: extent of arthritis
  - Characterisation of metabolic bone disease
- A bone scan is usually best done in conjunction with plain film x-rays, CT, or MRI. These modalities image anatomy whereas a bone scan tells of physiology/function. Hence doing both types of imaging together provides complementary information of greater clinical relevance than either type of test individually.
Genito-Urinary

107: Pelvic Exam

- See also 175: speculum examination, page 133
- Explanation while dressed. Check experiences with past exams
- Ensure chaperon if male
- Have available: light, additional light source and mirror for the patient
- Check bladder is empty
- Clear instructions to patient on what clothes to remove and position. Cover with sheet
- Position: flat on back on firm surface, unless prolapsed, obese or soft bed, in which case left lateral position (like recovery position)
- Pulse: indicator of anxiety
- BP
- General physical exam as indicated
- Vaginal Exam:
  - Bivalve Speculum: warm and check temperature. Introduce at 45 degrees then rotate. Use narrow speculum for nulliparous, wider speculum for multiparous, and paediatric for child or sometimes post menopausal. Use Sim’s Speculum for prolapsed. Warm blade, little (preferably no) lubricant if doing a smear.
  - Check size, shape, position and appearance of cervix, view transformation zone and os. Nulliparous or multiparous cervix
  - Bimanual:
    - Check uterus for size, shape, consistency, tenderness and mobility.
    - Check adnexae for abnormal swelling or tenderness
    - Normal tube and ovaries are not palpable.
  - Explain results when fully dressed

37: Dysuria

History
- Symptom assessment: describe this discomfort? Discharge?
- ROS: general and gentio-urinary
  - Fever, sweats or chills?
  - Abdo, testicular or flank pain
  - Joint or eye problems (Reiter's syndrome)
  - Do you find intercourse painful or uncomfortable (women)?
- Risk assessment: sexual history, previous UTIs, renal tract abnormalities

Differential:
- Urethritis: Usually causes pain at onset of micturition
- Cystitis: Usually causes pain at end of micturition; suprapubic discomfort.
- Strangury: Difficult and painful micturition associated with spasm
- PMS: Dysuria and discomfort common, due to atrophic urethritis (oestrogen dependent)
- Vaginitis: Dysuria described as burning “on the outside” with the discomfort usually felt at beginning or end of micturition
- Acute pyelonephritis: Chills; rigors; flank pain and tenderness
- Chronic pyelonephritis: Presents as chronic renal failure or one of its complications
- Other: Neoplasia; sexual abuse; trauma/foreign bodies; allergy or chemical irritant

Examination
- BP; temp; pulse
- Look for: enlarged bladder, large prostate, renal mass, meatal ulcer, vaginal discharge, loin tenderness, hypertension, signs of chronic renal failure
Investigations

- Micro: MSU
- Consider: Creatinine, blood culture, US, IVU/cytoscopy, PSA if:
  - Recurrent UTI (or first in men)
  - Overt haematuria
  - Unusual organism or persistent fever
  - Pyelonephritis

Treatment

- Drink plenty (2 cups/hour)
- Antibiotics (eg trimethoprim 200mg/12hr PO for 3-5 days {may need longer if complicated UTI)

Ref: Murtagh, General Practice 2nd Edition, Chapter 68, pp 703 - 711

<table>
<thead>
<tr>
<th>135: Test and Interpret Urinalysis</th>
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<tbody>
<tr>
<td>If you suspect renal disease always obtain some fresh midstream urine for:</td>
</tr>
<tr>
<td>- ‘Dipstick’</td>
</tr>
<tr>
<td>- Microscopy</td>
</tr>
<tr>
<td>- Culture</td>
</tr>
</tbody>
</table>

MSU collection

- Female
  - The patients bladder should be full (desperate to go)
  - The patient removes underpants and stands over the toilet pan
  - The labia are separated using the left hand
  - The vulva is cleansed front to back with sterile swabs
  - The patient voids downward into the toilet and continues until half done
  - Without stopping the urine flow, the sterile container is plunged into the stream of urine with the right hand. Only a small volume is required.
  - The patient then completes voiding into the toilet
- Male
  - The patients bladder should be full
  - The foreskin, if present, is retracted
  - The glans penis is cleaned with a sterile swab
  - The patient voids into the toilet until half done
  - Without stopping the flow, the sterile container is plunged into the urine stream
  - The patient then completes voiding into the toilet

Testing the urine specimen

- Put dipstick in urine
- Make sure all test squares are covered by urine
- Remove from urine and insert into machine
- Wait until it prints out the results
- If machine not available: Read appropriate square of dipstick at the appropriate time (indicated on dipstick container) against the references on the dipstick container

Causes of abnormalities on ward test (eg Multistix)

- Haematuria:
  - Renal:
    - GN; vasculitis; interstitial nephritis
    - Neoplasia; cystic disease
    - Trauma; vascular malformations; emboli
  - Extra-renal: Calculi; infection; catheterisation; trauma; neoplasia
  - Coagulation disorders
- NOTE: Dipsticks are very sensitive for blood and sometimes give positive results in normal people. Free haemoglobin and myoglobin will also return a positive dipstick test. Discoloured urine is also seen with porphyria, rifampicin, and beetroot ingestion.
- Work up: Microscopy, FBC, ESR, U&E, creatinine clearance, plain films, US etc
- Glycosuria:
  - Causes: Diabetes Mellitus, pregnancy, sepsis, tubular damage, low renal threshold
  - Work up: Blood glucose, pregnancy test, etc
- Proteinuria:
  - UTI, vaginal mucus, DM, GN, nephrosis, pyrexia, CCF, pregnancy, postural proteinuria
  - Rarer causes: Haemolytic uraemic syndrome, incr BP, SLE, myeloma, amyloid
  - Must be quantified with 24 hour collection
- Microalbuminuria: DM 20-200 micrograms/min = 30-300mg/day. Urine/albunin > 2.5
- Nitrites:
  - Nitrates in urine converted to nitrites by gram-ve (and other) bacteria that cause UTI's
  - May also be present after a high protein meal
- Leucocytes: A positive reaction on the dipstick for nitrites and leucocytes has a high predictive value for a UTI.

_Urine microscopy_
- RBC:
  - Uniform shape and size → bleeding from lower urinary tract
  - Variations in size or shape of cells → glomerular bleeding (GN)
- WBC: inflammation; renal Tb; renal stones; papillary necrosis
- Casts:
  - Hyaline casts: mucoprotein matrix without cellular elements → may be seen in normal urine
  - Granular casts: degenerative cellular elements (tubular cells) → renal disease esp ATN
  - RBC casts → glomerular bleeding (GN)
  - WBC casts → pyelonephritis; interstitial nephritis; glomerulonephritis

_Assessment of glomerular filtration_
- Urea
  - Raised with GI bleeding, high protein diet or corticosteroids
  - Reduced by low protein diet, malnutrition or liver disease
- Creatinine
  - Affected by muscle mass, protein intake and age
- Creatine clearance
  - 24 urine collection (creatinine clearance = UV/P)
  - OR ESTIMATE
    - \[ \text{GFR} (\text{ml/sec}) = \left(\frac{140-\text{age[yrs]}}{\text{lean body weight (kg)}}\right) \times 0.85 \times \text{serum creatinine (umol/L)} \]
  - 50 x \text{serum creatinine (umol/L)}


**45: Haematuria**

_History_
- Symptom assessment: duration, macroscopic (patient can see it) or microscopic? (NB macroscopic haematuria usually looks tea or coke coloured) Redness is at the start or end of your stream or throughout the stream? Easy bruising or nosebleeds?
- ROS
  - Genito-urinary: dysuria, loin or abdo pain, frequency, problems with flow etc.
  - General: general health/weight loss/appetite/energy
- Risk assessment: Sexually history, overseas travel, past kidney problems, recent strenuous sports such as jogging, family history of PCKD

_Differential_
- Pre-renal: Coagulation defects, sickle-cell disease
- Renal:
  - Infection, tumour, trauma, stones
• Cystic disease
• GN (IgA nephropathy is the only common GN causing macroscopic haematuria)
• Vasculitis or infarction
• Ureters or bladder:
  • Cystitis (infection by far the most common cause of microscopic haematuria) or drug induced eg cyclophosphamide
  • Tumour, trauma, stones
• Prostate: Prostatitis, carcinoma
• Urethra: Urethritis, trauma, foreign body
• Diet: beetroot, berries, red lollies or jelly may colour the urine red

Aids to determining the cause
• Redness at start of stream: Urethral or prostatic lesion
• Painful haematuria: Infection/calculi/renal infarction
• Painless macroscopic haematuria = cancer until proven otherwise
• Loin pain: Pyelonephritis/stones/renal carcinoma/polycystic kidney
• Dipstick positive for blood but no RBCs seen on microscopy suspect myoglobinuria from muscle injury/inflammation.

Examination
• General:
  • Signs of bleeding tendency + anaemia
  • Temp, BP, Pulse
• CVS: Exclude AF, IE with emboli to kidney
• Respiratory: Exclude pleural effusion due to perinephric or renal infection
• Abdomen:
  • Enlarged kidney or spleen
  • Bladder tenderness or enlargement
  • Prostate exam
  • Pelvic exam

Investigations
• Urine: dipstick/microscopy/culture
• Blood: FBC, ESR, U&E, Cr, ASO titres
• Radiology: Ultrasound is "always" the first line renal imaging. Abdo X-ray for stones.
• Biopsy

Ref: OHCM 4th Edition, pg 370; Murtagh, Chapter 68, pp 703 - 711

88: Urinary retention

• Retention is the inability to empty the bladder completely and follows failure of the bladder pressure to overcome urethral resistance. Thus it may be seen in bladder weakness or urethral obstruction.

Differential
• Brain: stroke, Alzheimer's, old age
• Spinal cord: compression (eg cauda equina syndrome), MS
• Nerves: DM, radiotherapy, surgical damage
• Urethral blockage: extrinsic (prostate), intrinsic (eg stones, clots)

The cause in men is nearly always prostatic obstruction.
• History (detecting outflow obstruction): Hesitancy, terminal dribbling, stream, incontinence, strangury, urgency, nocturia, frequency

Acute Retention
• Pain and complete failure of micturition. The bladder usually contains around 600mls.
• May be precipitated by anticholinergics, ‘holding on’, constipation, alcohol, or infection.
• Examination - Abdomen, PR, perineal sensation (cauda equina compression)
• Investigations - MSU, U&E, FBC, PSA, IVU
• Treatment - Catheterisation +/- an elective prostate procedure

**Chronic Retention**
• This is more insidious. Bladder capacity may be > 1.5 litres.
• Presentations - overflow incontinence, acute on chronic retention, lower abdo mass, UTI, renal failure
• Causes - Prostatic enlargement is the commonest cause. Others: pelvic malignancy, CNS disease
• Treatment - Only catheterise the patient if there is pain, UTI, or renal impairment eg urea > 12mmol/L. Institute definitive treatment promptly.

86: Urinary frequency

• See also 71: Polyuria, page 109

Differentiate increased urine production eg:
• Diabetes insipidus/mellitus
• Polydipsia
• Diuretics
• Renal tubular disease
• Adrenal insufficiency
• Alcohol

From frequent passage of small amounts of urine eg
• Cystitis
• Urethritis
• Neurogenic bladder
• Extrinsic bladder compression (eg pregnancy)
• Bladder tumour
• Enlarged prostate

Do full history paying particular attention genito-urinary history:
• Intake of fluids, thirst etc
• Change in appearance of urine
• Change in urine volume or stream - polyuria, nocturia, anuria, stream, hesitancy, dribbling, retention, incontinence, double voiding, renal colic, dysuria, fever, loin pain, urethral discharge, impotence, loss of libido, menses, infertility, pregnancies, vaginal discharge, genital rash.

• Examination - Abdomen, PR, perineal sensation (cauda equina compression)
• Investigations - MSU, U&E, FBC, PSA etc (depends on history)
• Treat cause

87: Urinary hesitancy

• Urinary hesitancy is when on wanting to pass urine there is a delay before starting. It is an obstructive urinary symptom. It is usually associated with poor flow and interruption of stream during voiding.

Causes
• Prostatic hypertrophy - the most common cause
• Strictures
• Tumours
• Urethral valves
• Bladder neck contracture
• Stage fright ie at a crowded urinal when others are waiting to use it

• Full History
• Examination and Investigations - see No 86
• Treat cause
Incontinence of urine and faeces

Incontinence = bladder pressure > urethral pressure

- Stress incontinence - bladder pressure momentarily exceeds urethral pressure (called genuine in the absence of detrusor activity). Small volume. No desire.
- Detrusor instability (urge incontinence) - strong urge to micturate due to detrusor over activity. Complete emptying of bladder follows.
- Overflow incontinence - over distended bladder (without detrusor activity). Small amounts frequently.
- Neurogenic bladder (reflex incontinence) - abnormal spinal reflex activity without desire to micturate. Larger volumes voided.
- True incontinence - defect in anatomical integrity.
  - Congenital eg ectopic ureter
  - Acquired eg pregnancy (long 2nd stage), surgery, radiotherapy.

History

- Symptom assessment: When does it occur. Amount lost
- ROS:
  - GU: Urgency, frequency, nocturia, haematuria, dysuria, flow
  - Neurological
  - Constipation
- Risk assessment: obstetric history, surgery, cancer (bone mets affecting the spinal chord)
- Medications - TCAs, B-blockers, Ca antagonists, diuretics
- Social history - mobility/ADLs, effect on social life.
- NB - the genitals, urinary tract, bowel and spinal chord should rarely be discussed independently.

Examination

- Speculum exam
- Rectal exam
- Neurological exam

Investigations

- MSU, blood glucose, symptom diary may also be very helpful. Refer (eg for urodynamics studies) if empirical therapy fails or the picture is complicated (eg neuro signs).

Management

- General - aid mobility and access to the toilet, treat constipation, reduce tea/coffee, lose weight, reduce smoking, change meds or timing of doses, use of pads.
- Genuine stress incontinence - pelvic floor exercises, oestrogen, surgery (must treat any instability component first)
- Urge/Detrusor instability - bladder retraining, bladder relaxants (oxybutynin)
- Overflow - remove obstruction, intermittent/permanent catheter
- Same principles apply for faecal incontinence. Most common cause is constipation or diarrhoea (usually with reduced mobility.)

Impotence

Differential

- Psychogenic: stress, interpersonal or intrapsychic factors (eg depression, marital problems)
- Neurogenic: Disorders affecting parasympathetic sacral spinal cord (eg MS)
- Diabetes
- Endocrine
  - Androgen deficiency
  - Hypothyroidism
  - Hyperprolactinaemia (secondary to loss of testosterone)
- Vascular
- Drug induced: alcohol, nicotine, pharmaceutical preparations
- Unknown
History
- Nature of onset
- Nature of relationship
- Drug history
  - Alcohol, nicotine, street drugs, anti-hypertensive, anti-depressants, anti-psychotics
- Nocturnal and early morning erections.
- ROS: neuro and psych

Examination
- Rectal examination
- Neurological
  - Lower limbs
  - Genitalia
  - Cremasteric and bulbocavernosus reflexes
- Vascular examination (concentrating on same areas as neurological)
- Mental state

Investigations
- Blood
  - Free testosterone ?androgen deficiency
  - Thyroxine ?hypothyroid
  - Prolactin ?hyperprolactinaemia
  - LH
  - Glucose
  - LFT (especially GGT) ?alcohol

Management
- Psychogenic disorders: psychotherapy and sex behavioural modification
- Hormonal disorders: testosterone; thyroxine; or bromocriptine (depending on hormone deficiency)
- Oral medication: Viagra
- Intrapenile injection: Alprostadil intracavernosal injection (self-administered)
- Transurethral alprostadil: Urethral pellet (start at 250mg)
- Surgery: Malleable prosthesis; inflatable prosthesis; vascular surgery

64: Nocturia

Differential
- Vascular: Recovering phase of acute tubular necrosis
- Neurological: Neurogenic bladder
- Idiopathic: Polydypsia
- Metabolic:
  - Diabetes insipidus (nephrogenic and differential)
  - Any thing that causes increase solute loading to kidney
- Endocrine:
  - Diabetes mellitus
  - Increased glucose loaded to kidney
  - Diabetes mellitus
  - Cushing’s
  - Acromegaly
- Neoplasia: Prostate causing overflow

65: Nocturnal Enuresis

Definition
- Bed-wetting at a time when control of urine could be reasonably expected (usually at age of 5)
What is normal?
- Common up to age of 5:
  - 50% 3 year olds wet beds, 20% of 4 year old, 15% of 5 year olds.
  - Considered a problem if occurs regularly in children > 6 years old
- Causes:
  - Usually no obvious cause
  - Must exclude:
    - UTI
    - Diabetes Mellitus
    - Diabetes insipidous
    - Neurogenic bladder
    - Urinary tract abnormality
- Family history: parents likely to have had delayed continence

Investigations
- After age of 6, investigate cause with
  - Blood glucose
  - Urinalysis
  - Intravenous urogram
  - Ultrasound

Advice for parents
- If no cause found, reassure child that nothing is wrong and that it is a common condition and will eventually go away
- Parents should
  - Not scold or punish child
  - Praise child often, when appropriate
  - Do not stop child drinking after the evening meal
  - Use a night-light to help the child who wakes
  - Bladder retraining
  - Special absorbent pads beneath bottom sheet

Treatment
- Bed wetting alarm system – alarm sounds when urine passed → child wakes → visits toilet
- Vasopressin for temporary relief of symptoms only
- IF PERSISTENT BEYOND EARLY ADOLESCENCE, FORMAL URODYNAMICS

Ref: Murtagh, General Practice 2nd Edition, Chapter 73, pp 749 - 750

71: Polyuria

Definition
- Passing up to 3.5 l/day

Differential Diagnosis
- Diabetes Mellitus
- Cranial Diabetes insipidous (or anything that ↑ solute load to kidney)
- Nephrogenic diabetes insipidous: Inherited or secondary to renal disease
- Hypercalcaemia
- Polydipsia
- Chronic renal failure
- Drugs

Screening questions
- How much fluid do you drink a day?  (Polydipsia)
- Energy levels?  (CRF + Diabetes mellitus)
- Weight loss?  (CRF + Diabetes mellitus)
- Nausea?  (CRF)
- “Bones, groans, abdominal groans and psychic moans”  (Hypercalcaemia)
• Head injury? (Cranial Diabetes insipidous)
• Kidney problems? (CRF, Nephrogenic DI)
• Drugs?

**Investigations**

- Blood: U&E, creatinine, Ca, glucose
- Imaging: AXR, renal imaging if necessary

---

**84: Undescended Testes**

*Problems of non-descent*

- Testicular dysplasia
- Susceptible to direct violence (if in inguinal region)
- Risk of malignant change (seminoma) if 5-10 times greater than normal

*Advantages of early orchidectomy (1 year)*

- Provides optimal chance of fertility
- Corrects indirect inguinal hernias (co-exist in 90%)
- Reduces risk of trauma
- Reduces risk of torsion
- Reduces psychological consequences
- Probably lessens the risk of malignancy

*Undescended testis*

- Probably mechanical cause
- Cannot reach bottom of scrotum despite manual manipulation
- Usually normal, but may become dysplastic if left outside the scrotum
- Can occupy the following positions
  - Intra-abdominal
  - Inguinal canal
  - Emergent (just outside external ring)
  - High scrotal
  - Mid-scrotal

*Retractile testis*

- Can be manipulated into scrotum irrespective of position in which it is first located.
- Testes can be present in scrotum in warm bath, and retracted out when cold
- Cremasteric reflex absent in 1st few months after birth and maximal between 2 and 8 years.

*Ectopic testis*

- Has left the normal path of descent and cannot be manipulated into the scrotum
- Can be found in:
  - Perineum
  - Upper thigh (femoral)
  - Base of penis
  - Anterior abdominal wall
  - Superficial inguinal pouch

*Ascending testis*

- Was in scrotum in infancy but subsequently moved back into groin because spermatic cord failed to elongate at same rate of body growth

*Examination*

- Place on finger on each side of the neck of the scrotum to prevent the testes being retracted out of the scrotum with the other hand.
- Palpate gently for a testis
- If impalpable the fingertips of one hand are placed just medial to anterior superior iliac spine → move firmly towards the pubic tubercle where the other hand wait to entrap the testis
- The diagnosis depends on determining the rang of movement
Optimal time for surgery

- Optimal age = 12-18 months
- Production of spermatozoa is adversely affected following testes still undescended from 2 years old onwards

- Ref: Murtagh, General Practice 2nd Edition, Chapter 93, pp 909 - 911

IVP (Intravenous pyelography) = IVU (Intravenous urography)

- Provides excellent anatomical and functional information
- Useful for demonstrating the renal contours and the presence of scarring
- Very good at outlining the anatomy of the collecting system for the diagnosis of reflux nephropathy
- Excellent for demonstrating renal obstruction and the site of obstruction
- Dependent on the renal uptake of contrast. There is little point performing an IVU in the presence of significant renal impairment i.e. creatinine > 200umol/L

- **Preparation:** Laxatives, nil by mouth (unless uraemic or myeloma) for 12 hours
- **Contrast reactions** to IV reagents occur in 1/1000 patients and death by anaphylaxis in 1/40000. Reactions include hives, bronchospasm, and/or pulmonary oedema. Ask about a history of atopy or allergy to iodine or seafood. Premedication with corticosteroids is effective in reducing the incidence of reactions. The newer non-ionic contrast agents available should be used in this situation. When renal function is impaired ensure good hydration (IV fluids) pre- and post-study.
- Always look at the plain film for renal size and calcification anywhere in the urinary tract

**Stages of the IVP**

- **Control**
- **Nephrogram phase** – Contrast reaches renal tubules i.e. taken up by kidney tissue. Usually immediate, within the first minute, and brief. It is said to be:
  - Intense in obstruction and GN
  - Prolonged in obstruction, ATN, chronic GN, and renal vein thrombosis
  - Absent in a non-functioning kidney (infarction, severe GN)
  - Delayed in renal artery stenosis (spec 80%, sens 85% if unilateral)
- **Pyelogram phase** – Contrast appears in renal pelvis and calyces
- **Ureters, and bladder phase** – Can see these structures outlined
- Empty bladder and check emptying

- Look for these abnormalities:
  - Absent kidney: obscured by bowel gas or perinephric abscess, renal agenesis, nephrectomy (look for amputated 12th rib)
  - Small kidney: (<10cm) chronic pyelonephritis, renal artery stenosis. Bilateral in chronic renal failure (chronic GN or pyelonephritis)
  - Scarred outline: pyelonephrosis, ischaemia, TB.
  - Low kidney: (normal T12-L2) hepatomegaly, transplants, congenital.
  - Pelvicalyceal filling defects: one calyx not seen in tumour, TB, partial nephrectomy. Irregular filling defect – clot or tumour. Smooth defect – papilloma, renal artery aneurysm, pressure from vessel.

- Some patterns:
  - Obstruction: Prolonged and dense nephrogram, clubbed calyces, mega ureter.
  - Chronic pyelonephritis: Small scarred kidneys, thin cortex, clubbed calyces.
  - Papillary necrosis: Linear breaks at papillary bases.
MCU (Micturating Cystourethrogram)

- Involves filling the bladder with contrast via a urethral catheter, removing the catheter, and imaging the urinary tract during and following micturition.
- Used to demonstrate or exclude VUR and to study bladder emptying.
- Remains important for the assessment of vesico-ureteric reflux in children. (All children less than 2 years of age with a proven UTI should have a MCU).
- Is not an appropriate part of the investigation of the adult female with recurrent bacterial cystitis if the IVU is normal.

- Ref: Kumar and Clark pg 529
Blood, Endocrine and Metabolic

129: Abnormal Biochemistry in Blood or Urine

- Do not interpret biochemical results except in the light of clinical assessment. If there is a disparity trust clinical judgement and repeat biochemistry.

Biochemistry results: Major disease patterns

- Artefacts: Correct calcium for albumin
- Dehydration: Urea ↑, Albumin ↑ (useful to plot change in patient’s condition), Haematocrit ↑, Creatinine ↑
- Renal failure: Creatinine ↑, Urea ↑, Anion gap ↑, K ↑, HCO ↓.
- Thiazide and Loop diuretics: Na ↓, HCO ↑, K ↓, Urea ↑

<table>
<thead>
<tr>
<th>Bone disease</th>
<th>Calcium</th>
<th>Phosphate</th>
<th>Alk Phos</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteoporosis</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Osteomalacia</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Paget’s</td>
<td>N</td>
<td>N</td>
<td>↑↑</td>
</tr>
<tr>
<td>Myeloma</td>
<td>↑</td>
<td>↑,N</td>
<td>N</td>
</tr>
<tr>
<td>Bone Metastases</td>
<td>↑</td>
<td>↑,N</td>
<td>↑</td>
</tr>
<tr>
<td>Primary Hyperparathyroidism</td>
<td>↑</td>
<td>↓,N</td>
<td>N,↑</td>
</tr>
<tr>
<td>Hypoparathyroidism</td>
<td>↓</td>
<td>↑</td>
<td>N</td>
</tr>
<tr>
<td>Renal Failure (low GFR)</td>
<td>↓</td>
<td>↑</td>
<td>N,↑</td>
</tr>
</tbody>
</table>

- Hepatocellular disease: Bilirubin ↑, AST ↑, ALT ↑,(ALT is usually higher than AST in viral hepatitis), (ALP mildly ↑, Albumin↓)
- Cholestasis: Bilirubin ↑, GGT ↑↑, ALP ↑↑, usually extrahepatic cholestasis if > 350iu/L (AST ↑)
- Myocardial infarct: AST ↑, Troponin T&I ↑, LDH ↑, CK ↑ (CK-MB)
- Diabetes Mellitus: Glucose ↑, Bicarb↓
- Addison’s disease: K ↑, Na↓
- Cushing’s Syndrome: May show K ↓, HCO ↑, Na↑
- Conn’s Syndrome: May present with K ↓, HCO ↑ (and high BP). Na ↑ or normal
- Diabetes insipidus: Na ↑, (both hypercalcaemia and hypokalaemia may cause nephrogenic diabetes insipidus)
- Inappropriate ADH secretion: Na ↓ with normal or low urea and creatinine
- Excess alcohol intake: Evidence of hepatocellular disease. AST 2x > ALT is specific (but not sens) for alcoholic liver damage. Early evidence in GGT ↑, MCV ↑, ethanol in blood before lunch
- Some immunodeficiency states: Normal serum albumin but low total protein (low as immunoglobulins are missing)

Lab results: When to take action NOW

- On receiving a dangerous result first check it’s name and date
- Go to the bedside. If the patient is conscious, turn off any IV fluids (until fluid is checked: a mistake may have been made) and ask the patient how he/she is. Any fits, faints, collapses or unexpected symptoms?
- Be sceptical of an unexpectedly wildly abnormal result with a well patient.
- When in doubt repeat the test.

Potentially dangerous results

- Calcium (uncuffed and corrected) <2mmol/L or >3.5mmol/L
- Glucose <2mmol/L or >20mmol/L
- Potassium <2.5mmol/L or >6.5mmol/L
- Sodium <120mmol/L or >155mmol/L

Ref: OHCM 4th Edition, pg 625
130: Abnormal Blood Count

**Anaemia**

<table>
<thead>
<tr>
<th>Low MCV</th>
<th>Normal MCV</th>
<th>High MCV</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Iron deficiency</td>
<td>• Chronic disease</td>
<td>• B12 or folate deficiency</td>
</tr>
<tr>
<td>• Thalassaemia</td>
<td>• Bone marrow failure</td>
<td>• Alcohol</td>
</tr>
<tr>
<td>• Chronic Disease</td>
<td>• Renal failure</td>
<td>• Liver disease</td>
</tr>
<tr>
<td>• Sideroblastic</td>
<td>• Hypothyroid</td>
<td>• Hypothyroidism</td>
</tr>
<tr>
<td></td>
<td>• Haemolysis</td>
<td>• Marrow infiltration</td>
</tr>
<tr>
<td></td>
<td>• Pregnancy</td>
<td>• Myelodysplastic syndromes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Reticulocytosis (eg haemolysis)</td>
</tr>
</tbody>
</table>

**Neutrophils**

<table>
<thead>
<tr>
<th>Increased in</th>
<th>Decreased in</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Bacterial infections</td>
<td>• Viral infections</td>
</tr>
<tr>
<td>• Myeloproliferative disorders</td>
<td>• Tb</td>
</tr>
<tr>
<td>• Leukaemia</td>
<td>• B12 or folate deficiency</td>
</tr>
<tr>
<td>• Disseminated malignancy</td>
<td>• Marrow failure</td>
</tr>
<tr>
<td>• Trauma, burns, and surgery</td>
<td>• Chemotherapy</td>
</tr>
<tr>
<td>• Haemorrhage</td>
<td>• Sepsis</td>
</tr>
<tr>
<td>• Infarction and inflammation</td>
<td></td>
</tr>
<tr>
<td>• Polymyalgia and PAN</td>
<td></td>
</tr>
</tbody>
</table>

**Lymphocytes**

<table>
<thead>
<tr>
<th>Increased in</th>
<th>Decreased in</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Viral infections</td>
<td>• Steroid therapy</td>
</tr>
<tr>
<td>• CLL</td>
<td>• SLE</td>
</tr>
<tr>
<td>• Toxoplasmosis; whooping cough</td>
<td>• Uraemia</td>
</tr>
<tr>
<td>• EBV</td>
<td>• Post chemotherapy or radiotherapy</td>
</tr>
<tr>
<td></td>
<td>• Marrow infiltration</td>
</tr>
</tbody>
</table>

**Eosinophils**

- Increased in:
  - Asthma and allergic bronchopulmonary aspergillosis
  - Allergy
  - Autoimmune: PAN, urticaria, skin disease, eczema, pemphigus
  - Parasite infection
  - Malignant disease

**Monocytes**

- Increased in
  - Acute and chronic infections (eg Tb, myxoedema, protozoa)
  - Malignant disease (AML, Hodgkin’s disease)
  - Myelodysplasia

**Basophils**

- Increased in
  - Viral infections
  - Urticaria
  - Myxoedema
  - Post splenectomy
  - CML
  - UC
  - Haemolysis
Polycythaemia rubra vera

Ref: OHCM 4th Edition, pp 572 and 578

131: Fluid/Electrolyte Abnormalities

**Hyponatraemia (= hypo-osmolar state)**

Is the patient dehydrated?

- Yes
  - Is urinary Na > 20mmol/l
    - Yes
      - Na and H2O lost through kidneys
        - Diuretic excess
        - Osmolar diuresis (raised glucose, raised urea)
        - Addison’s disease
        - Renal failure (diuretic stage)
        - Nephrocalcinosis
    - No
      - Na and H2O lost elsewhere
        - Diarrhoea
        - Vomiting
        - Fistula
        - Burns

- No
  - Is the patient oedematous
    - Yes
      - Nephrotic syndrome, Cardiac failure, Cirrhosis of liver, Renal failure
    - No
      - Is urine osmolality > 500mmol/kg
        - Yes
          - SIADH
        - No
          - Water overload, Severe hypothryoidism, Glucocorticoid insuffic.

Symptoms and signs: Signs of water excess (eg confusion, fits, hypertension, cardiac failure, oedema, muscle weakness

Management:
- If not dehydrated & renal function good
  - Na >125mmol/l → Treatment rarely needed
  - Na <125mmol/L → restrict water to 0.5 – 1l/day (or frusemide slowly if necessary and replace Na and K loses)
- If dehydrated renal function good → give solution ISOTONIC TO PATIENT
- Aim to raise Na by 0.5-1mmol/L/hr if mildly symptomatic and no more than 12mmol/l in first 24 hours.
- Raise NA by 3-6mmol/L acutely if patient convulsing
- WATCH OUT FOR CENTRAL PONTINE MYELINOSIS

**Hypernatraemia (= hyperosmolar state)**

- Causes: Usually due to water loss in excess of sodium loss. Also consider replacement of losses with fluid of incorrect osmolality, or fluid loss without water replacement (eg diarrhoea, vomit, burns
IMPARIED FLUID INTAKE OR POLYURIA

- Hypernatraemia with hyperosmolality
- Hyperosmolality without hypernatraemia

**Check Fluid balance**

- ↓ fluid intake and urine output
- ↓ LOC
- Lesion of thirst centre

**Polyuria**

- Osmotic diuresis (urea, glucose, mannitol)
- Check urine glucose, serum urea, creatinine

**Diabetes mellitus**
**Normal**
**Renal impairment**

- Check urine & serum osmolality and renal response to ADH

**Central diabetes insipidous**
**Nephrogenic DI**

- Symptoms and signs: Thirst, confusion, coma, fits *with signs of dehydration*
- Management
  - Stop water loss
  - Water orally if possible
  - Otherwise dextrose 5% IV SLOWLY (4L/24hr)

**SIADH**
- Diagnosis: *concentrated urine (Na >20mmol/L) with hyponatraemia or low plasma osmolarity* (and absence of hypovolaemia, oedema or diuretics)
- Causes
  - Malignancy
  - CNS disorders (stroke, SAH….)
  - Chest disease (Tb, pneumonia)
  - Metabolic disease (porphyria)
  - Drugs (opiates)

**Hyperkalemia**
- **Check acid-base status (must correct abnormal pH before analysis)**
- Causes:
  - Oligouric renal failure
  - K sparing diuretics
  - Metabolic acidosis (DM)
  - Addison’s
  - Rhabdomyolysis
  - Artefact: Haemolysis
  - Drugs: eg ACE inhibitors
- Signs and symptoms: Cardiac arrhythmias and sudden death (ECG: tall tented T waves, small P wave, wide QRS)
- Treatment:
  - Treat underlying cause
  - Stabilise cardiac tissue: IV calcium gluconate
  - Increase ICF K shift: insulin + glucose
  - Reduce further K absorption: K binding resins
- Promote renal losses: diuretics, mineralocorticoids, dialysis.

**Hypokalaemia:**
- **Check acid-base status (must correct abnormal pH before analysis)**
- **Causes:**
  - Diuretics
  - Vomiting, and diarrhoea
  - Cushing’s syndrome/steroids/ACTH
  - Conn’s syndrome
  - Alkalosis
  - Renal tubular failure
- **Signs and symptoms:**
  - Muscle weakness, hypotonia, cardiac arrhythmias, muscle cramps and tetany
  - ECG: Small or inverted T wave, prominent U wave, prolonged PR interval, depressed ST segment
- **Treatment**
  - If mild (>2.5, no symptoms): Oral potassium supplement
  - If severe (<2.5, dangerous symptoms): IV K cautiously, not more than 20mmol/hr, not more than 40mmol/L

**Hypercalcaemia**

```
Hypercalcaemia

<table>
<thead>
<tr>
<th>Albumin raised</th>
<th>Albumin normal or low</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea raised</td>
<td>Urea normal</td>
</tr>
<tr>
<td>Dehydration</td>
<td>Cuffed specimen</td>
</tr>
<tr>
<td>Urea normal</td>
<td>Phosphate ↓ or norm.</td>
</tr>
<tr>
<td>Phosphate ↑ or norm.</td>
<td>Phosphate ↑ or norm</td>
</tr>
<tr>
<td>1° or 3° hyperparathyroid</td>
<td>Alk phos ↑</td>
</tr>
<tr>
<td>Alk phos norm</td>
<td>Bone mets</td>
</tr>
<tr>
<td></td>
<td>Myeloma</td>
</tr>
<tr>
<td></td>
<td>Sarcoioidosis (↑ plasma protein)</td>
</tr>
<tr>
<td></td>
<td>Thyrotoxicosis</td>
</tr>
<tr>
<td></td>
<td>Vit D excess</td>
</tr>
</tbody>
</table>

**Symptoms:** Bones, stones, abdominal groans and psychic moans (refer to OHCM pg 644)

**Treatment**
- Bloods: U&E, Mg, Cr, Ca, PO4, ALP
- Fluids:
  - Rehydrate with IV 0.9% saline (eg 4-6 L/24hours as needed)
  - Correct hypokalemia and hypomagnesia (monitor U&E)
  - Diuretics: Frusemide 40mg/12hr IV, once rehydrated
  - Bisphosphonates:
  - Steroids: Occasionally used (eg in sarcoidosis)

**Hypocalcaemia**
- **Causes**
  - Thyroid or parathyroid surgery
  - Chronic renal failure
• Hypoparathyroidism or pseudohyperPTH  
• Osteomalacia  
• Over hydration  
• Pancreatitis  
• Symptoms: Tetany, depression, parathesiae, carpo-pedal spasm, neuromuscular excitability.

Management:
• Symptoms mild:  
  • Calcium 5mmol/6hour; daily plasma levels  
  • If necessary alfalcaldoid 0.5 – 1ug/24hr PO  
  • Severe: 10ml calcium gluconate 10% IV over 30min. Repeat as necessary

Other important things  
• Dehydration: ↑Urea, ↑albumin ▲haematocrit, ▲PCV  
• Renal failure: ↑creatinine, ↑urea, ▲anion gap, ↑K, ▲HCO3  
• Thiazide and loop diuretics: ↓Na, ▲HCO3, ▲K, ▲urea  
• MI: ↑AST ▲CK ↑Trop I  
• Addison’s: ↑K, ↓Na  
• Cushing’s: May show ↑K, ↓Na, ↓HCO3  
• Conn’s: ↑Na, ↓K, ↑HCO3  

• Refer to OHCM pg 625 for others.


### 132 Arterial Blood Gas

**Metabolic acidosis**

- ↓pH, ↓CO2, ↓HCO3  
- Anion gap = (Na + K) – (HCO3 – Cl)  
- High anion gap:  
  - Ketoacidosis: diabetics, alcoholics, starvation)  
  - Lactic acidosis: hypoxia  
  - Ingestion: methanol, salicylates  
  - Renal failure  
- Normal anion gap (loss of alkali):  
  - Diarrhoea  
  - Ileal conduit  
  - Renal tubular acidosis (kidneys either leak HCO3 or cannot excrete H)  
  - Hypoaldosteronism

**Metabolic alkalosis**

- ↑pH, ↑HCO3, ↑CO2  
- Causes:  
  - Loss of acid: vomiting, NG suction  
  - Chloride diarrhoea  
  - Diuretics and volume depletion – lead to enhanced HCO3 reabsorption  
  - ↑aldosterone or mineralocorticoid  
  - Cushing’s syndrome  
  - Hypokalaemia – enhances HCO3 reabsorption  
  - Ingestion of alkali (anatacids)

**Respiratory alkalosis**

- ↑pH, ↓CO2, ↓HCO3 (hyperventilation)  
- Causes:  
  - Hypoxia  
  - Lung disease: PE, asthma  
  - Anxiety  
  - Fever, sepsis
- Salicylate overdose: stimulates respiration + will subsequently develop a metabolic acidosis

**Respiratory acidosis**
- $\downarrow \text{pH}, \uparrow \text{HCO}_3, \uparrow \text{CO}_2$ (hypoventilation)
- Causes:
  - Pulmonary disease
  - CNS depression
  - Muscular disease
  - Asphyxia, smoke inhalation

Ref: Dr G. Pidgeon’s 5th Year Renal Handout.

---

28: Diabetes Mellitus

**Presentation**
- Asymptomatic
- **Polyuria, Thirst**
- Weight loss, Lack of energy
- Superficial infections
- Complications: Ketoacidotic, Retinopathy, Polyneuropathy, Impotence, Arterial disease.

**Diagnosis**
- Fasting glucose $> 7.0$ mmol/L
- Random blood glucose $> 11.1$ mmol/L on two occasions
- 2 hour Glucose tolerance test $> 11.1$ mmol/L

**Treatment**
- The care of diabetes is based on self management by the patient, who is helped and advised by those with specialised knowledge.
- A good relationship with the patient is essential as the understanding and cooperation of the patient is the key to good glycaemic control.

**Complications**
- IHD
- Renal failure
- Stroke
- Neuropathy
- Vascular disease (eg claudication)
- Retinopathy
- Infections

**Risk Factors**
- High cholestrol
- Hypertension
- Smoking
- Alcohol
- Obesity

**Education/negotiation**
- Explanation of Diabetes (for type 1):
  - Due to lack of insulin from damage to pancreas
  - What insulin does: shifts sugar from blood to cells – without it there is too much sugar in the blood
  - Leads to symptoms: weight loss, polyuria, etc
  - Also leads to nasty complications over time
  - Requires lifelong replacement with insulin
  - Monitoring blood glucose (with finger prick) and adapting treatment accordingly (IDDM)
  - Lifestyle measures:
- **Exercise**: “Give sensible suggestions eg walking the dog, taking the stairs instead of lift etc instead of advising to join Gym”.
- **Diet** (low fat diet, low glycaemic index foods eg bread, pasta etc)
- **Weight loss** - If patient is over weight
- **Reduce risks**: Stop smoking (call the Quitline: 0800 778 778)

**Medication:**
- Explain that when ill more, not less, insulin is required
- Consider exposing to a ‘hypo’ to show how to abort it with sweets etc

**Medical management:**
- Introduce to specialist nurse, dietician, chiropodist, and diabetic association
- Regular follow up
- Referral is necessary to: ophthalmology, renal, cardiology, etc
- Must inform LTSA (if patient has a driving licence)?

**Drugs**
- Sulfonyleureas (Thin NIDDM’s)
- Biguanides (Fat NIDDM’s)
- Glucosidase inhibitors
- Insulin (IDDM’s and NIDDM’s not controlled by above measures)
- Treat hypertension vigorously. ACE inhibitors reduce progression of kidney disease. Treat hyperlipidaemia.

**Complications**
- Vascular disease
- Kidneys - Check urine regularly for protein ie dipstick
- Retinopathy - Arrange regular fundoscopy for all patients
- Cataracts
- Diabetic feet - Educate on foot care eg daily foot inspection, comfortable shoes etc
- Neuropathy
- Autonomic neuropathy - postural hypotension, impotence, diarrhoea at night
- Hypoglycaemia (if on insulin or sulphonylureas)

### 134: Test and Interpret Capillary Glucose

**Advantage Meter**
- Calibration – Before using a strip from a new pack, insert the code key found in the pack into the meter. It ensures the meter is using the latest data.
- Insert test strip. – Meter turns on automatically.
- Obtain a small drop of blood.
  - Washing the patients hand/finger is important
    - Reduces the risk of infection in puncture site
    - Makes sure that no food residue is on the hands that will give a falsely high result
    - Washing in warm water improves the circulation
  - Obtaining blood from the side of the finger is recommended as it is easier and less painful. If blood is not coming out get them to wiggle the finger (squeezing causes bruising)
- Touch and hold the drop of blood to the right-hand edge of the yellow window. – The blood will automatically be drawn into the yellow window of the test strip, and the test will begin.
- Make sure the yellow window is completely filled with blood. – Additional blood can be applied to the edge of the strip within 15 seconds of the first drop.

**Miscellaneous**
- Only use test strips that are designed specifically for advantage meter. Strips aren’t interchangeable between different brands of machine.
- Store strips between 4 and 30 degrees, out of direct sunlight
- Check expiration date on test strips before use
Interpretation

<table>
<thead>
<tr>
<th>Condition</th>
<th>Fasting Blood Glucose</th>
<th>2 hours post glucose tolerance test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes</td>
<td>&gt; 7.0 (was 7.8) mmol/l</td>
<td>&gt; 11.1 mmol/l</td>
</tr>
<tr>
<td>Impaired Glucose Tolerance</td>
<td></td>
<td>&gt; 7.8</td>
</tr>
<tr>
<td>Impaired Fasting Glycaemia (new</td>
<td></td>
<td>&gt; 6.0</td>
</tr>
<tr>
<td>category)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Tests for thyroid disorders

- Thyroid function tests

<table>
<thead>
<tr>
<th>Condition</th>
<th>TSH</th>
<th>T4</th>
<th>T3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypothyroidism</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Primary</td>
<td>↑*</td>
<td>↓*</td>
<td>N or ↓</td>
</tr>
<tr>
<td>• Secondary</td>
<td>N or ↓</td>
<td>↓</td>
<td>N or ↓</td>
</tr>
<tr>
<td>Hyperthyroidism</td>
<td>↓*</td>
<td>↑</td>
<td>↑*</td>
</tr>
<tr>
<td>Sick euthyroid</td>
<td>N or ↓</td>
<td>N or ↓</td>
<td>N or ↓</td>
</tr>
</tbody>
</table>

*main test

- False values
  - T3 and T4 ↑ in pregnancy, thyroglobulin excess, and oestrogens
  - T3 and T4 ↓ with salicylates, NSAIDs, phenytoin, corticosteroids, carbamazepine, etc.

- Thyroid isotone scanning: is indicated if
  - Area of thyroid enlargement
  - Hyperthyroid without thyroid enlargement
  - Hyperthyroid with one nodule
  - If subacute thyroiditis is a possibility
  - Ultrasound: distinguishes cystic from solid (cancer) nodules
  - Thyroid autoantibodies
  - Fine needle aspirate
  - CT: determine extent of neck compression from thyroid enlargement

Hyperthyroidism

- Symptoms
  - Weight loss despite increase appetite
  - Frequent stools
  - Tremor
  - Irritability; frenetic activity; emotional lability
  - Dislike hot weather; sweating
  - Oligomenorrhea

- Signs
  - Tachycardia (even when sleeping); AF
  - Warm peripheries
  - Fine tremor
  - Thyroid: enlargement, nodule, bruit
  - Myopathy
  - Graves: bulging eyes, lid lag, virtilgo, pretibial myxoedema

- Causes
  - Graves: women 30-50yrs; autoantibodies against TSH receptors; associated with IDDM and pernicious anaemia
  - Toxic adenoma
  - Subacute thyroiditis: goitre (often painful); ↑ESR; probable viral cause
  - Self-medication: raised T4 low T3
  - Toxic multinodular goitre

- Treatment
  - Carbimazole (SE agranulocytosis)
Partial thyroidectomy
Radioactive I131

**Hypothyroidism**
- Symptoms
  - Increase weight, decrease appetite
  - Constipation
  - Dry skin, hair falling out, swollen ankles
  - Dislike cold
  - Lethargy, depression and mental slowing
  - Hoarseness
  - Menorrhagia
- Signs
  - Goitre
  - Bradycardia
  - Dry skin and hair
  - Slowly relaxing reflexes
  - CCF
  - Non-pitting oedema
- Causes
  - Autoimmune (Hashimoto’s)
  - After thyroidectomy or radioactive treatment
  - Drug induced: anti-thyroid drugs, amiodarone
  - Iodine deficiency
- Treatment: Thyroxine

**Thyroid examination**
- Observe while swallowing water
- Palpate from behind. Find isthmus then move laterally
- Palpate lymph nodes
- Listen for bruits
- Test vocal cords (recurrent laryngeal nerve)


<table>
<thead>
<tr>
<th>43: Galactorrhoea</th>
</tr>
</thead>
</table>

- Usually due to hyperprolactinaemia (but normoprolactineamic galactorrhoea does occur, the causes of which are poorly understood)

**Hyperprolactinaemia**
- Symptoms
  - Women: oligo- or amenorrhoea, infertility, vaginal dryness, weight gain, apathy
  - Men: impotence, ↓ facial hair
  - Both: ↓ libido, delayed or arrested puberty, symptoms relating to the primary cause
- Causes
  - Physiological - pregnancy, breastfeeding, stress, REM sleep, nipple stimulation, coitus
  - Drug induced - DA antagonists (Maxolon, phenothiazines), oestrogen, opiates, cimetidine
  - Pathological - prolactinoma, acromegaly, stalk compression, idiopathic, PCO, primary hypothyroidism, chest wall injury, renal failure, liver failure
- Investigations
  - At least three prolactin levels should be checked. Non-stressful between 10am-12.
  - Visual fields
  - Anterior pituitary function - IGF-1, T4 and TSH, LH and FSH
  - MRI/CT of pituitary fossa
- Management
• Micro-prolactinoma (<10mm) - bromocriptine (SE =N+V, anorexia, hypotension. Increase dose slowly). Stop on becoming pregnant (pregnancy does not increase tumour size)
• Macro-prolactinoma (most common cause in men) - try bromocriptine unless there are visual symptoms, pressure effects or pregnancy is contemplated then surgery is indicated

76: Short Stature

*Short stature = height below the 5th centile*

• If both parents are short, their child will likely be short too (constitutional short stature accounts for 80% of short children). Likely height of a boy = (mother's height + 13cm + father's height)/2. For a girl do the same but subtract 13cm from father's height.

*Variations from normal:*
• Familial (genetic) short stature. Below 3rd centile but growing parallel ie normal growth rate. Parents are usually short and pubertal development usually occurs at the appropriate time
• Constitutional delay in growth and pubertal development Common, short stature during childhood, delayed puberty and eventually catch up to peers. Often a family history

*Pathological causes of short stature*
• Intrauterine growth retardation
• Chronic disease - any system, growth failure usually with ass fall in weight velocity. A hormone or endocrine problem is unlikely to be the cause of poor growth if both weight and height are affected
• Skeletal disorders - Usually familial eg achondroplasia. Major features, \( \uparrow \) upper body to lower body segment ratio. Limbs usually short and weight gain is usually normal
• Iatrogenic - Corticosteroid excess in children. Growth failure ass with weight gain. Irradiation to head/neck may result in pituitary hypofunction may lead to poor growth of spine and trunk
• Chromosomal abnormalities and syndromes - eg Turners syndrome
• Psychosocial - Poor/ineffective diet, physical or psych abuse, fall off of weight gain and linear growth
• Endocrine - least common pathological cause. Hypothyroidism, GH def, Cushing’s syndrome, adrenal insufficiency. Fall off of linear growth exceeds fall in weight gain

*History*
• Height/weight compared to peers, duration of short stature
• Birth details
• Family history - mid parental height

*Examination*
• Height/weight
• Assess pubertal status
• General physical exam including BP - chronic disease, nutritional state, dysmorphic features
• Goitre or other signs of hypothyroidism - dry hair/skin, bradycardia, \( \downarrow \) reflexes
• Evidence of midline brain development syndromes which may result in hypopituitism eg cleft palate
• Visual fields to exclude possibility of a pituitary lesion

*Management*
• Plot height/weight, compare with parents

*Investigations*
• Bone age xray
• FBC and ESR
• Urea, creatinine, and electrolytes
• Calcium and phosphate
• Thyroid function tests
• Chromosomes (girls only)
Treatment
- Girls < 152cm
- Boys < 162cm
- Can use biosynthetic GH in GH def, Turner syndrome, growth retardation secondary to renal insufficiency, intrauterine growth retardation, and severe idiopathic short stature.
Skin

22: Common skin lesions

History
- What is the lesion?
  - Colour. Raised or flat
  - Is it itchy or painful
  - Scaly, dry or oozing
- Where is the lesion?
  - Localised, central or acral (=worse at peripheries)
  - Is the head involved
  - Are mucus membranes affected
- Is this related to a systemic illness (eg hypothyroidism, measles) or is it purely a "skin disease" (eg eczema)
- Do contacts have a similar rash?
- Past hx, meds (could this be a drug rash?), possible allergens, family history

Examination
- Observation - size, colour, shape
- Palpation - raised or flat, solid or fluid, texture (rough, scaly or smooth)
- Determine which part of skin is affected. The epidermis is never involved alone. Epidermis and dermis = crusting, weeping and/or vesicles. Dermis alone = papule or nodule.
- Ask yourself, could the appearance have been modified by treatment?
- What is the distribution of the rash? Widespread, central, peripheral or face
- Always exam mouth, scalp, nails, hands and feet

Diagnosis
- Psoriasis
- Acneiform eruptions: acne, rosacea, perioral dermatitis
- Dermatitis: atopic (eczema), contact, irritant, seborrhoeic
- Autoimmune: pemphigus, pemphigoid
- Drugs
- Infections - virus, bacteria, fungus
- Urticaria (itchy maculopapular rash) - includes scabies, insect bites, pediuclosis (lice)
- Sun damage (including cancers)

Refs:
- Murtagh 2nd Ed, p957
- For an algorithm of diagnosis by lesion characteristics see Talley and O'Connor p444.

72. Pruritus

Skin disease (usually localised)
- Very itchy
  - Infections - chicken pox (adolescents and adults)
  - Dermatitis - atopic or contact
  - Urticaria (itchy maculopapular rash) - includes scabies, insect bites, pediuclosis
  - Grover's disease (transient acantholytic dermatosis)
  - Dermatitis herpetiformis - most (?)all have celiac disease
- Mild to moderately itchy
  - Infections - tinea, candida
  - Psoriasis
  - Pityriasis rosea
  - Stress itching/lichen simplex
  - Lichen planus - flat tipped papules on wrists and legs. Cause unknown.
  - Asteatosis (dry skin)
- Often not itchy
• Infections - warts, viral exanthemata, impetigo, tinea
• Psoriasis
• Cancers
• Seborrhoeic dermatitis

Systemic disease (usually generalised)
• Liver disorders
• Malignancy - Hodgkin's (30%), leukaemia, disseminated cancer - ?due to uraemia?
• Haematological disorders - Fe deficiency, PRV
• Endocrine disorders - ↑↓ thyroid, DM, hyperparathyroidism
• Drugs
• Senile pruritus (old dry skin)
• Other: Tropical infection/intestinal worms, irritants (eg fibreglass), PAN, coeliac disease (rare).

Psychological and emotional disorders
• Depression/anxiety
• Psychosis
• Parasitophobia

Pruritus ani
• Haemorrhoids
• Infection - candidiasis, herpes, trichomonas, UTI, threadworms, scabies, pediculosis
• Hygiene - too little or too much
• Irritants - soaps, tight clothing
• Menopause (oestrogen deficiency)
• Other - e.g. local cancer, psychological

Ref: Murtagh 2nd ed; page 965

39: Eczema

Differential
• Atopic eczema:
  • Cause: Multifactorial, but genetic component present
  • Distribution: Infants: Cheeks of face, folds of neck and scalp, groin
  Adult: Flexure surfaces
  • Diagnosis: Itch; typical morphology and distribution, dry skin, history of atopy, chronic relapsing dermatitis
  • Plan: Prevent scratching, Emollients, moisturisers, steroid, etc…

• Irritant dermatitis:
  • Causes: Detergents, soaps, oils, solvents, alkalis, etc…
  • Distribution: Most frequently hands involved
  • Diagnosis: Itchy, inflamed skin; red and swollen; papulovesicular; may be later dry and fissured
  • Plan: Avoid all irritants if possible; good hand care; emollients; moisturisers, steroids, gloves, etc…

• Allergic dermatitis:
  • Causes: Type IV reaction to allergen (e.g. nickel, latex…)
  • Distribution: Depends on cause (e.g. hands if latex glove allergy)
  • Diagnosis: Site and shape of lesion suggests contact; linear lesions; allergic causes may be found by patch testing; improvement when off work or on holiday.
  • Plan: Determine cause and remove it; wash with water and avoid soap; steroids etc..

• Adult seborrhoeic dermatitis:
• **Cause:** Due to yeast overgrowth in skin
  • **Distribution:** Scalp (dandruff), eyebrows; nasolabial folds; cheeks; flexures
  • **Diagnosis:** Red, scaly eruptions
  • **Plan:** Mild topical steroid/antifungal.

• **Discoid eczema:**
  • **Cause:** Stress
  • **Distribution:** Often symmetrical; mainly on legs, also buttocks and trunk
  • **Diagnosis:** Chronic, red, coin shaped plaque; crusted, scaling and Itchy
  • **Plan:** Same as atopic dermatitis

• **Pityriasis alba:**
  • **Cause:** Subacute form of atopic eczema
  • **Distribution:** More common around mouth and on cheeks, but can occur neck and upper limbs.
  • **Diagnosis:** White patches (on the faces) of children and adolescents
  • **Plan:** Reassurance, emollients, restrict use of soap and washing.

• **Dyshidrotic eczema:**
  • **Cause:** Stress
  • **Distribution:** Sides of digits or palms, larger vesicles on palms and soles of feet.
  • **Diagnosis:** Itchy vesicles on finger, or palms and soles
  • **Plan:** As for atopic eczema; potent fluorinated corticosteroids topically; oral steroid may be necessary.

• **Asteatotic eczema:**
  • **Cause:** Related to dry skin; elderly – diuretics, hypothyroid, excessive washing
  • **Distribution:** Usually on legs
  • **Diagnosis:** Superficial fissures creating a crazy paving pattern
  • **Plan:** Mild soap or soap substitute; moisturiser; +/- topical steroid

• **Ref:** Murtagh, General Practice 2nd Edition, Chapter 101, pp 972 - 988

### 89: Urticaria

#### Features
- Onset usually over minutes
- Transient erythema
- Transient oedema
- Transient itch

#### Classification according to site
- **Superficial:**
  - Affecting superficial dermis
  - Occurs anywhere on body, especially limbs and trunk
- **Deep:**
  - Affecting subcutaneous tissues
  - Angioedema
  - Occurs anywhere but especially periorbital region, lips and neck

#### Causes
- **Allergies** (dramatic and potentially very serious):
  - Azo dyes
  - Drugs: penicillin and other antibiotics
  - Food: eggs, fish, cheese, tomato, others
  - Infection: bacteria, parasites, protozoa, yeast
- **Pharmacological:**
  - Drugs: penicillin, aspirin, codeine
- Foods: eggs, shellfish, nuts, strawberries, chocolate, artificial food colourings, wheat, soybeans
- Plants: nettles, other
- SLE
- Physical:
  - Cholinergic: response to sweating induced by exercise and head
  - Heat, cold, sunlight
- Pregnancy (last trimester)
- Unknown (idiopathic) – 80%; possible psychological factors

**Investigations**
- FBC & film – look for eosinophilia or parasites
- ANF and DNA binding – consider SLE
- Challenge tests (patch testing)

**Management**
- Avoid any identifiable cause
- Avoid salicylates and related food preparations
- Antihistamines (eg cetrizine 10-20mg daily)
- Luke warm bath with Pinetarsol or similar soothing bath oil
- Topical 0.5% hydrocortisone – apply every 4 hours for itching
- If severe: prednisolone 50mg once daily for 10-14 days.

Ref: Murtagh, General Practice 2nd Edition, Chapter 100, pg 971
Obstetrics and Gynaecology

159: Contraception

- Ideal contraceptive is 100% effective, only desirable side-effects, readily reversible, and able to be used un-supervised
- Reference: OHCS + numerous pamphlets

**Education: Starting on the OCP**

- **Starting:**
  - Start on the first day of your period (so you know she is not pregnant)
  - Begin pill in the red section on the appropriate day of the week
- **Taking the pill:** every day, at about the same time
- Safe to have sex from seven days after starting the pill or straight away if starting on the first day of period.
- If you miss a pill, then take it when you remember
- The seven day rule:
  - If you have:
    - Missed a pill by more than 12 hours
    - Diarrhoea
    - Vomiting
    - On antibiotics
  - Continue to take the pill
  - You are not protected for 7 days after the end of the diarrhoea/antibiotics etc. Use barrier methods
  - If you are on the last week of a packet, go straight onto the next 7 hormone pills

**Education: OCP and DVTs**

- What is a clot:
  - Describe DVT and PE
  - Most DVTs are not fatal
- Assess risk factors for DVT:
  - Past history of a clot
  - Obesity
  - Hypertension
  - Hyperlipidaemia
  - Smoking
  - Family History
- Explanation of risk:
  - 1/100,000 per year are fatal
  - Much lower rate than in pregnancy
  - Approx 1/10,000 risk of being killed in a car accident
- Discuss options:
  - Pros and cons of 2nd generation pill
  - Other contraceptive options
- Any other concerns?

**Natural Family Planning**

- No intercourse from 6 days before to 2 days after ovulation – free and no drugs
- Monitor fertility by:
  - Checking cervical mucus – clear and stretchy when fertile
  - Temperature ↑ 0.3 C after ovulation (affected by fevers, drugs, drink)
- Success if regular cycles, dedication and self-control
- Peak effectiveness is 2% - usually 10 - 20 % (pregnancies per woman years)

**Barrier Methods**

- Low health risk, need high motivation, some STD protection
- Condoms, Caps +/- spermicide, female condom (Femidom)
- Don’t use oil-based lubricant or anti-thrush cream with condom
Spermicide gives extra protection

**IUCD**
- Very effective (failure rate 1-2 per 1000 woman years)
- Inhibit implantation and may impair sperm migration
- Need replacing every 3 – 5 years
- Best in older, parous women in stable relationships
- Contraindications: Pregnancy, high risk for STD, undiagnosed vaginal bleeding, very heavy periods, previous ectopic pregnancy
- Complications:
  - Can be expelled from a nulliparous or distorted (eg fibroids) uterus
  - Ectopic pregnancy more likely (1 in 2000)
  - Associated with PID following insertion or STD
- Mirena – carries levonorgestrel (a progestrone) → ↓risk of implantation and lighter periods. Lasts 3 years. 20% experience reversible amenorrhea. Expensive

**Hormonal Contraception**
- **Combined Oral Contraceptive (CoC)** - 99% effective
  - = oestrogen (usually ethinylestradiol) + progestogen
  - Action: G type mucus + ↓GnRH (→ no ↑FSH or LG surge). ‘Puts the ovary to sleep’
  - Low dose is ≤ 30 μg oestrogen. Adverse effects are dose related ⇒ give lowest dose that gives good cycle control.
  - Take for 3 weeks, then pill free for a week → withdrawal bleed
- Contraindications:
  - Cancer (oestrogen dependent eg endometrial, breast) or undiagnosed PV bleeding
  - Liver disease
  - CVS reasons
    - Raised DVT risk: past DVT/PE, family history of DVT/PE
    - Raised arteriosclerosis risk: > 35 and smoker, raised lipids, DM, HT, BMI >30, family history, previous MI, stroke
  - Pregnancy or breastfeeding
  - Migraine with aura or for > 72 hours (status migrainosus) or requiring ergotamine. Pill → 4 times risk of ischaemic stroke. Contra-indicated in any woman with migraine if >1 other risk factor for stroke (lipids, BP, diabetes, etc)
- Side-effects: (usually worse when starting the pill for first 3/12)
  - Head: headache, N+V, change in mood
  - Weight: ↑ or ↓
  - Libido: ↑ or ↓
  - Skin: chloasma, acne, photosensitivity, hirsutism
  - Menstrual: inter-menstrual bleeding, breast tenderness
- Risks:
  - Pregnancy
  - DVTs. Risk ↑ sharply over 40 – 1:2500 for non-smokers, 1:500 for smokers.
    - 35/100,000 on the pill per year develop a clot, one dies ⇒ two deaths per year in NZ
    - Risk increases 3-4 times over population risk on 2nd generation, 6 – 8 times on 3rd generation.
    - No ↑risk with PoP
  - Increased risk of cervical and breast cancer
  - Does not protect against STDs
  - Special precautions: Family history of DVT, ↑BP or breast cancer; epilepsy, diabetes, illnesses causing diarrhoea (eg Crohn’s)
  - Drugs interfering with the pill: liver enzyme inducers (eg anticonvulsants, rifampicin). Consider higher dose pill
  - Benefits of CoC: 99% effective, reversible, lighter periods, ↓PMS, ↓ovarian and endometrial carcinoma (but slightly ↑risk of breast cancer), ↓endometriosis
  - Benefits
- Decreased risk of ovarian and endometrial cancer
- Decreased benign breast and ovarian disease
- Decreased menstrual disorder, ectopics, fibroids, endometriosis
- Monitoring: 6 monthly-BP check. Check weight and breasts etc if > 35. Up to date with smears?
- Starting the pill: On day 1 of cycle, or day of TOP, 3 weeks post-partum or 2 weeks after mobilisation after major surgery. Contraceptive cover immediate
- Missed pill: 12 hours late OK, after that the seven day rule (also if diarrhoea) – take 7 active pills before unprotected sex (eg if pill free days coming then skip them and go straight onto the next pack)
- Stopping:
  - 66% menstruate within 6 weeks, 98% by 6 months
  - At menopause: Stop at 50 with > 1 years amenorrhoea. CoC masks menopause, so stop at 50 and use non-hormonal method.
- **Progesterone Only Pill (PoP) (=mini-pill) - 2 – 4% failure**
  - → cervical mucus hostile to sperm (G Type mucus) + prevent ovulation in some + ↓ tubal motility. Effectively a barrier method. Woman may still ovulate. Small risk of follicular cyst (one that doesn’t pop) → pain with full bladder or rectum
  - Worst side effect: erratic bleeding. Some women have amenorrhoea. Less risk of weight gain, acne, depression, breast tenderness
  - Benefits: OK if breast-feeding, smokers .35, history of DVT, heart conditions, IDDM. No increased risk of cancer
  - Contraindications: History of ectopic pregnancy, breast cancer, liver disease or enzyme inducing drugs
  - Must be taken same time each day (+/- 3 hours).
  - Starting on the PoP: Alternative precautions for 7 days
  - If pill missed then at risk for 2 days. Safe again after a further 2 days.
- **Depot progestogen**
  - Safe, simple and effective (failure rate 0.4 – 1.2 %). Suppress ovulation, G type mucus, ↓ motility and implantation
  - Eg Depot-provera – deep IM 12 weekly, given during first 5 days of cycle, 5 days post partum if bottle feeding, 6 weeks if breast feeding.
  - Contraindications: pregnancy, abnormal undiagnosed vaginal bleeding, acute liver/cardiac disease
  - Advantages: no oestrogen, ↓PMS, secret, no compliance problems, good with GI disease, ok with breast feeding, etc… Particularly good around major surgery, epileptics, after vasectomy and bowel disease
  - Problems: irregular bleeding – usually become amenorrhoea, also weight gain and acne. May also ↑depression and ↓libedo. Median delays of 10 months return to ovulation on stopping.

**Sterilisation**

- Reversal is only 50% successful ⇒ see it as irreversible
- Tubal ligation has 1% failure (1:200) – 10 times worse than vasectomy and same as IUCDs
- Vasectomy – easier than tubal ligation, but takes up to 3 months before stored sperm used up. Need to be tested and have 2 sperm-free ejaculates. Has been discussion of ↑risk of prostate cancer – best evidence says no association.

**Emergency Contraception**

- Ask why: unprotected intercourse, condom broke, etc. If no condom, then check why. If indicated: ‘Are you worried about infection?’ and ‘Was it OK with you that it all happened the way it did’ [checking for non-consensual intercourse]
- Ask:
  - How long ago was sex, LMP, Regular partner (→ ↓risk of STD), Medications
  - Other unprotected sex this cycle
  - Previously had an ECP – any side effects. Sometimes nausea +/- vomiting with old Progesterone only ECP

2003 OSCE Handbook
• Other conditions. Old Oestrogen + Progesterone ECPs required history of DVT and focal migraine
• Prescribe: Nordiol 2+2/Antinaus 5mg or 2/Microval 25+25
• Discuss:
  • How to take it - 12hrs apart
  • Pregnancy test in three weeks
  • Ongoing contraception, other advice
• Emergency IUCD: inserted within 120 hours of unprotected intercourse. Screen for STDs. Prophylactic cover if suspected.

173: Abnormal Cervical Smear

• The most important information is that an abnormal smear does NOT mean you have cancer.
• An abnormal smear does mean that your smear picked up some 'at risk' cells from your cervix.
• Those at risk cells may, given enough time (usually years) eventually develop into cancer or they may return back to normal.
• It is fairly common to have an abnormal smear result. 1 in 5 women have some changes and 1 in 20 need further investigation.
• We can't tell which at risk cells will turn into cancer and which ones will return to normal so we follow up all abnormal smears.
• Cells are extremely unlikely to progress into cancer in the time you wait for your next smear or for your colposcopy appointment.
• Colposcopy is an examination similar to having your smear taken but the doctor looks through a colposcope which magnifies the cervix. The cervix will be painted with a weak vinegar-like solution to show up any abnormal areas. A tiny sample may be taken from the cervix. This is sometimes uncomfortable but is usually not painful.

Bethesda coding system (show results to the patient and go through them with her)
• A-code - adequacy. If the smear is unsatisfactory a repeat smear will need to be taken immediately.
• B-code - category of smear result. Includes a recommendation for the timing of the next smear or referral to a specialist.
  • Within normal limits
  • Outside normal limits
    • Inflammation - sometimes due to infection but is often a normal finding in sexually active women. If there is an infection the report may give the cause eg thrush. Sometimes other tests are indicated eg high vaginal swab.
    • Low-grade Squamous Intraepithelial Lesion (LSIL) = HPV changes and/or CIN 1 (mild) 50-60% may return to normal in a few months. 10% of CIN 1 progress to CIN3). Repeat smear in 6/12. If still abnormal then refer for colposcopy.
    • High-grade Intraepithelial Lesion (HSIL) = CIN 2 (moderate), CIN 3 (severe), carcinoma in situ - referral for colposcopy. 20-70% CIN 3 become cancers.
• C-code - diagnosis codes for additional diagnostic information about the smear

163 & 156: Abnormal Menstruation

Menstrual History
• Date of last menstrual period (LMP)
• Normal cycle: number of days bleeding/length of cycle
• Regularity
• Current cycle
• How heavy is the bleeding (number of pads, flooding, clots)
• Associated pain
• Bleeding in between periods
• Bleeding post-coitally:
  • Deep ⇒ pathology
• Superficial \(\Rightarrow\) usually psychological
• Bleeding since menopause
• Age at menarche and menopause
• Previous pelvic surgery

**Amenorrhoea**

- Primary amenorrhoea: failure to start menstruating. Investigate in a 16 year old or a 14 year old with no breast development. Is there other pubertal delay? When did her mum start menstruating? Usually normal.
  - Hypothalamic/pituitary failure (LH/FSH low) eg Turner’s syndrome
  - Gonadal failure (LH/FSH high) eg testicular feminisation
- Secondary amenorrhoea: when periods stop for > 6 months, except for pregnancy:
  - Hypothalamic/pituitary causes common. Eg stress, anorexia, breast-feeding, ↑PRL, change in weight, severe disease. Test with a 7-day progesterone challenge. If withdrawal bleed following, then there is enough oestrogen to produce an endometrium
  - Ovarian causes are uncommon: Polycystic ovarian syndrome, tumours, premature menopause - ask about flushes etc
- Oligomenorrhoea: infrequent periods: common in the young and the nearly menopausal.
  Consider polycystic ovary syndrome, rapid weight change, ↑PRL, hypothyroidism or primary oligomenorrhoea

**Menorrhagia**

- = Excessive blood loss (technically > 80ml lost/cycle – but hard to measure)
- Causes:
  - ?Hypothyroidism: cold intolerance, weight gain, constipation, goitre, etc
  - Younger: PCOD, pregnancy, dysfunctional uterine bleeding (diagnosis of exclusion, no pelvic pathology, associated with anovulatory cycles. If young, may settle)
  - Older (>35): IUCD, fibroids, endometriosis, adenomyosis, polyps, pelvic infection
  - Perimenopausal: ?endometrial carcinoma (especially if > 90Kg)
  - Haematological: low or dysfunctional platelets (not coagulopathy)
- Treatment if pathology known: Progesterone, CoC, Mirena, anti-PGs (eg NSAIDs), Danazol, etc, endometrial resection, ablation or hysterectomy

**Inter-menstrual bleeding**

- May follow mid-cycle \(\downarrow\) in oestrogen.
- Also cervical polyps, ectropion, carcinoma, cervicitis and vaginitis, IUCD, hormonal contraception (spotting), endometriosis

### 175: Speculum Examination

- Explanation while dressed. Check experiences with past exams
- Ensure chaperon if male
- Have available: light, additional light source and mirror for the patient. Name smear slides.
- Check bladder is empty
- Clear instructions to patient on what clothes to remove and position. Cover with sheet
- Position: flat on back on firm surface, unless prolapsed, obese or soft bed, in which case left lateral position (like recovery position)
- Pulse: indicator of anxiety
- BP
- General physical exam as indicated
- Vaginal Exam:
  - Bivalve Speculum: warm and check temperature. Introduce at 45 degrees then rotate. Use narrow speculum for nulliparous, wider speculum for multiparous, and paediatric for child or sometimes post menopausal. Use Sim’s Speculum for prolapse (left lateral position). Warm blade and check on patient's thigh, little (preferably no) lubricant if doing a smear.
  - Check size, shape, position and appearance of cervix, view transformation zone and os. Nulliparous or multiparous cervix
  - Bimanual:
• Check uterus for size, shape, consistency, tenderness and mobility.
• Check adnexa for abnormal swelling or tenderness
• Normal tube and ovaries are not palpable.
• Explain results when fully dressed.

161: Dysmenorrhoea

= painful periods, may be associated with sweating, tachycardia, headache:

<table>
<thead>
<tr>
<th>Primary</th>
<th>Secondary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>&gt; 20</td>
</tr>
<tr>
<td>Onset of pain</td>
<td>Prior to bleeding</td>
</tr>
<tr>
<td>Duration</td>
<td>Through out menses</td>
</tr>
<tr>
<td>Intensity</td>
<td>Begins at any time, progressively worsens</td>
</tr>
<tr>
<td>Aetiology</td>
<td>Outside uterus: Endometriosis</td>
</tr>
<tr>
<td></td>
<td>In uterine wall: Adenomyosis</td>
</tr>
<tr>
<td></td>
<td>Fibroid/polyps</td>
</tr>
<tr>
<td></td>
<td>Cervical Stenosis</td>
</tr>
<tr>
<td></td>
<td>Inside uterus: Pelvic Infection</td>
</tr>
<tr>
<td></td>
<td>Chronic sepsis (eg Chlamydia)</td>
</tr>
<tr>
<td></td>
<td>Conditioned behaviour</td>
</tr>
</tbody>
</table>

• Treatment:
  • Reassurance
  • CoC: at least 3 month trial, combine with NSAIDs if necessary
  • Progestogens: day 5 – 25
  • PG inhibitors
  • Exercise
  • De-conditioning, eliminate secondary gains

157: Abnormal Vaginal Discharge or Bleeding

• See also 163 & 156: Abnormal Menstruation, page 132

Premenopausal bleeding

• Intermenstrual bleeding
  • Follows a mid-cycle fall in oestrogen - idiopathic, hormonal contraception.
  • Other - pregnancy-related, IUCD, ectropion, causes of post-coital bleeding.

Postcoital bleeding

• Vagina - vaginitis (any cause)
• Cervix
  • Cervicitis (any cause)
  • Trauma
  • Polyps - endocervical
  • Cancer

• Investigations: speculum and pelvic exam, smear, swabs, pregnancy test

Postmenopausal bleeding (=endometrial cancer until proven otherwise)

• = Bleeding >1yr after menopause. If on HRT then called irregular peri-menopausal bleeding on HRT. If >60 on HRT called postmenopausal bleeding on HRT
• Check it is vaginal bleeding, not urethra or rectal
• Vagina - atrophic vaginitis: fragile → trauma, and ↓secretions → ↑infection
• Cervix - erosions, cervicitis, cancer
• Endometrium - polyps (fibroid or endometrial), hyperplasia, cancer
• Foreign bodies (eg pessaries)
• Oestrogen withdrawal (HRT or ovarian tumour)
• Investigations
  • Distinguish from peri- or post-menopausal on HRT
  • Cervical smear
  • Transcervical US (for endometrial thickness) and transabdominal US (for masses)
  • <5mm → return in 4-6 months (5-15mm in non-pregnant menstruating women)
- 5-9mm → endometrial sampling (90% normal proliferation, 5% atypia, 5% Ca)
- >9mm → Dilatation and curettage
- Same procedure for HRT but slightly ↑ risk of endometrial Ca so manage more aggressively
- Management - for endometrial Ca need TAH/BSO

Vaginal discharge
- Normal physiological discharge - normally no odour or itch. May be aggravated by the Pill. Reassure, advise to wear cotton underwear, avoid douching, bath instead of showering.
- Vaginitis
  - Candida - thick white. No odour, normal pH. Itch, soreness, redness
  - Bacterial - grey, watery, profuse. Fishy smell with KOH. pH 5-6. No itch or soreness.
  - Trichomonas - yellow/green, frothy, profuse. Fishy smell. pH 5-6. Soreness
  - Chemical, atrophic (postmenopausal), 'sandbox' vaginitis in children due to dirt.
- STDs/PID
  - Other - polyps, cancer, fistula, foreign body eg tampon, endometriosis (brown discharge).
  - Do speculum and pelvic examination, pH test, amine whiff test with KOH, wet film microscopy and take swabs and culture if the diagnosis remains uncertain or there is concern about STDs.

169: Premenstrual Syndrome (PMS)
- = Recurrence of symptoms, whether emotional or physical, occurring the pre-menstruum but with complete absence of symptoms in the post-menstruum. Severe symptoms in 5% of women
- DSM 4 has ‘Premenstrual Dysphoric Disorder’ as a research criteria
- Main symptoms:
  - Depression, irritability, tiredness, headache, bloating, breast tenderness.
  - Plus 150 others!
  - Classify as mild, moderate or severe on the basis of interference with daily function
  - Use of a symptom diary over 2 months is very valuable
- Diagnosis:
  - History
  - Exam to exclude gynaecological and endocrine disorders
  - Tests: rule out thyroid, PRL, secondary dysmenorrhoea (eg endometriosis)
- Differential:
  - Psychiatric: depression or anxiety with premenstrual exacerbation
  - Medical: anaemia, hypothyroidism, cancer, SLE, menopause if > 45, renal causes, polycystic ovary
- Management:
  - Education
  - Life-style changes: diet, exercise, ↓ smoking
  - Psycho-therapy if psych history, for coping skills, or to manage secondary gains or conditioning
  - Drugs:
    - Suppression of ovulation. Eg with CoC – although this can give symptoms (eg depression, ache, etc)
    - Fluoxetine 20 mg only when symptoms occurring (30% remission, minimal side effects)
    - Debated remedies include evening primrose oil, Vitamin B6 (pyridoxine) in low dose (neuropathy in high dose)
    - Very high placebo rates
- Aetiology:
  - Multifactorial – includes biological, psychological and societal factors
  - Biological hypotheses include abnormal response to ovarian hormones, Mineralocorticoid effects, prostaglandins, etc.
Menopausal Symptoms

- Menstrual disturbance - periods become less frequent and irregular over 3-5 yrs
- Vasomotor symptoms - associated with LH surge
  - Hot flushes (don't necessarily blush)
  - Palpitations
  - Night sweats.
- Genitourinary symptoms
  - Atrophic vaginitis - bleeding, dyspareunia, post-coital bleeding
  - Atrophic urethritis - frequency, incontinence, urgency, dysuria
- Mood swings, depression

- Occur in >85% in first 6 months. Most symptoms settle 2-3 yrs after last period.
- Later sequelae of oestrogen deficiency → CVS disease, osteoporosis.
- Need to continue contraception for 1yr after last period.
- Diff diagnosis - depression, anaemia, thyroid disease, hyperPTH, gynaecological disorders.
  (Test for high FSH)

HRT (replacing normal physiological dose of oestrogen (cf COC))

- Indications - not a cure for all problems especially mood symptoms. Try listening, lifestyle changes, vaginal lubricants or topical oestrogens.
  - Distressing symptoms
  - Significant CVS risk factors
  - Significant osteoporosis
- Contraindications:
  - Pregnancy or breast feeding
  - Hx breast or endometrial Ca
  - Undiagnosed vaginal bleeding
  - Liver disease, ↑LFTs
  - Past PE
- False contraindications:
  - High cholesterol - it is protective (cf COC doses of progesterone which is bad)
  - Smoking
  - ??Past DVT - probably not a contraindication
  - ??Hypertension - only if currently uncontrolled
- Side effects and risks:
  - PV bleeding/periods (depends on type of HRT), breast tenderness, N+V, ↑weight, headache, skin changes, PMS (ie symptoms caused by the COC may return)
  - Rare: migraine, DVT asthma, raised BP, cholestasis, alopecia
  - ↑Risk of breast cancer if > 5 yrs. ↑1%/yr if taking oestrogen and ↑8%/yr if taking oestrogen and progesterone (JAMA 283:485-89, 2000). Mammograms less sensitive. One study showed that the risk returns to normal 2 yrs after stopping.
  - If has severe heart disease then risk ↑in first yr but ↓after that.
- Types:
  - Cyclical - continuous oestrogen, progesterone for 10-12 days/cycle (with 3-4 days of bleeding 2-3 days after it is stopped). Good if immediately postmenopausal. Cycle them for a while then change to continuous after a year.
  - Continuous - continuous oestrogen and progesterone. No periods as there is stable endothelium. Don't start until >1yr after last period. Ovary may occasionally surge causing break through bleeding which then needs investigation.
  - If no uterus then just give continuous oestrogen.

Patient Assessment for HRT

- LMP - regular before that? PV bleeding since then? Vaginal dryness?
- Sexually active?
  - Contraception - present and previous. Side effects from COC?
  - Dyspareunia, post-coital bleeding
- Vasomotor symptoms: hot flushes, night sweats, palpitations
• Urogenital symptoms: Dysuria, frequency, incontinence, vaginal dryness
• Mood: anxiety, irritability, etc
• Past medical history:
  • Breast/endometrial cancer or undiagnosed vaginal bleeding
  • Liver disease
  • CVS history:
    • CVS problems
    • Hypertension, hyperlipidaemia, DM
    • DVT/PE
• Medication and allergies
• Family history: Osteoporosis, endometrial or ovarian cancer, cardiovascular disease, DVT/PE

171: Routine Antenatal Care

• Key issue for many indicators is serial measurement

General exam
• Weight and height → BMI
• Pulse, blood pressure (sitting)
• Teeth, signs of thyroid disease, signs of anaemia
• Heart and lungs (eg wheeze, mid systolic murmur common, pan systolic and diastolic abnormal)
• Breast exam, including nipples.
• Oedema
• Varicose veins
• Abdominal exam (see 174: Obstetric Palpation, page 139)

Tests
• Urine dipstick from 20 weeks for albumin (ie proteinuria) and glucose.
• Check Hb and Rh antibodies (eg at 28 weeks) and do glucose challenge test.

Pelvic exam (booking exam)
• Speculum exam
  • HVS where indicated for chlamydia, gonorrhoea, BV, candida, trichomoniasis
  • Cervical smear only if not up-to-date
• Bi-manual – uterus size consistent with dates and not adnexal masses. Uterus becomes an abdominal organ (rather than pelvic) at 12 weeks

Booking tests:
• Blood:
  • FBC: check for anaemia
  • Blood group: check if Rh –ive. If so, mark clearly in notes. Give Anti-D following birth or invasive procedure
  • RBC Antibodies (eg Anti-D, Anti-ABO, etc)
• Serology:
  • Syphilis (VDRL): treat with course of penicillin IM
  • Hepatitis B: if +ive, test and immunise partner and close contacts. At birth give Hep B IgG and Hep B vaccine to baby (repeat at 1 and 6 months)
  • Rubella: If negative for Rubella and pregnant then NO vaccine (it’s a live vaccine). Stay away from kids. If she gets sick, repeat serology 2 – 3 weeks later to see if it was Rubella. 70% fetuses affected in 1st trimester, drops to < 5% by 16 weeks.
  • Discuss/offer HIV testing
• MSU for protein, bacteria and glucose
• Ultrasound if dates unsure (otherwise offer morphology at 18 weeks)
• Tb if high risk (immigrant, family contact, etc)
• Sickle cell anaemia if black
• α-feta protein/triple test if at risk of Down
• If > 35 then offer amniocentesis
158: Assessment of Fetal Welfare

- The main aim of these tests is to detect fetal hypoxia. Most have high false positive rates so need to be interpreted in light of the clinical picture and other test results.

**Cardiotocography (fetal heart rate monitoring)**

- Records heart rate and response to uterine contractions. Check baseline, variability, accelerations, decelerations.
  - Normal base rate = 110-150bpm. Abnormal = <100bpm, >170bpm or sinusoidal pattern.
  - Normal (reactive) trace = variability > 5 bpm and at least 4 accelerations of at least 15bpm in 20 minutes. False negative rate is 6.8 / 1000.
  - Intermediate trace - widely spaced accelerations and reduced variability - further evaluation is needed in these cases.
  - Abnormal (non-reactive) trace - the presence of decelerations, absence of accelerations and reduced variability is a very serious sign and immediate delivery should be considered.

**Doppler Ultrasound and Blood flow velocity Waveforms**

- Estimates blood flow in umbilical artery, placenta and uterus
  - Not proven as a routine screening tool but does benefit high-risk pregnancies.
  - Low blood velocity implies high resistance to flow. Placental resistance changes with gestation so accurate age is needed. As placental resistance (due mostly to tertiary stem villi) increases end-diastolic frequencies cannot be seen. Further increases in resistance leads to flow reversal through the umbilical artery, which is preterminal.
  - In high risk pregnancies with abnormal Doppler waveforms, low dose aspirin improves outcomes but does not benefit those with abnormal waveforms (and increases abruption rates)

**Ultrasound scan**

- (sens=75-94%, spec=88-100 for size +amniotic fluid vol)
  - Fetal size (biparietal diameter, abdominal circumference, femur length). Serial measurement of fetal growth is of most use.
  - Amniotic fluid estimation
  - Assess fetal breathing - decreases in response to hypoxia, hypoglycaemia and alcohol/sedatives

172: Shock in Pregnancy

**Hypovolaemic**

- Bleeding in early pregnancy (= bleeding before 20/40)
  - Miscarriage - incomplete or septic.
  - Ectopic pregnancy - USS to exclude uterine pregnancy + quantitative βHCG. Do Rhesus screen and give anti-D if positive
  - Trophoblastic disease - complete or partial hydatiform mole, choriocarcinoma
  - Antepartum haemorrhage (= bleeding after 20/40) - NO vaginal exam until USS excludes praevia:
    - Placental abruption - shock may be out of proportion to visible blood loss. Constant pain with a tense and tender uterus.
    - Placenta praevia - shock in proportion to visible losses. No pain. No uterine tenderness.
  - Ruptured uterus - rare in the West. 70% due to rupture of previous CS scar. Rupture usually in third trimester or during labour

**Distributive**

- Inverted uterus - Occurs during third stage labour. Shock may occur without haemorrhage.
  - Amniotic fluid embolus - Anaphylactic type response to amniotic fluid. Dyspnoea, hypotension +/- seizures +/- DIC. Often at end of 1st stage or after delivery.
  - Adrenal haemorrhage
  - Septicaemia - may lack classical signs such as pyrexia. Rx = co-amoxyclov + metronidazole

**Cardiogenic**

- Pulmonary embolus, decompensated heart disease
Management

- ABC
- Insert large bore IVs. Blood for tests and cross match and then give colloids to maintain BP>100mmhg. Get expert help urgently.
- Delivery is usually necessary. (Laparoscopy or D+C if early in pregnancy)
- Shock in pregnancy may lead to renal failure and Sheehan's syndrome (pituitary necrosis).
- CPR in pregnancy. If >20/40 need to roll/push uterus to the left to prevent compression of the IVC. Breath and push faster and start defibrillating at full. Success very unlikely unless immediate caesarean performed.

- Ref: OHCS 5th Ed. p106

166: Pain in Abdomen in Pregnancy

Usually early in pregnancy (<20/40)

- Ligament stretching
- Miscarriage
- Ectopic pregnancy

Usually later in pregnancy (>20/40)

- Braxton-Hicks contractions
- Labour
- Abruption - triad of abdominal pain, uterine bleeding and vaginal bleeding.
- Uterine rupture - usually in third trimester or during labour
- Uterine fibroids - increase in size esp in 2nd trimester. May tort if pedunculated or undergo red degeneration (inflammation). Rx is analgesia with resolution in 4-7 days.
- Uterine torsion - rotates > 90 degrees causing pain, tense uterus, shock, urinary retention.
- Pre-eclampsia - due to liver congestion.

At any time during pregnancy

- Ovarian tumours - torsion or rupture of a cyst. Confirm with USS. 2-5% of tumours are malignant. Removal at 16/40 or if discovered late leave until postpartum.
- Pyelonephritis - common at around 20/40. UTI should always be carefully excluded.
- Appendicitis - not more common in pregnancy but ↑mortality esp after 20/40
- Cholecystitis - subcostal pain, nausea, vomiting. Jaundice is uncommon (5%). USS to confirm stones.
- Rectus sheath haematoma - suspect violence but may rarely occur due to straining. USS is useful.

Management

- Always consider that it may be the beginning of labour
- USS may be very useful.
- When there is any uncertainty about the diagnosis laparotomy should be performed by an obstetrician

174: Obstetric Palpation

- See also 171: Routine Antenatal Care, page 137
- Observation - size, shape, symmetry scars, stria, hernias, lie
- Fundal height - 1cm/week. Drops a bit after 36/40. Can just palpate uterus in the abdomen at 12 weeks. At 20 weeks up to umbilicus.
- Palpation - lie and presentation from 32 weeks.
  - Find pole (palpate form fundus to symphysis) = lie (long axis of baby cf. long axis of mother)
  - Find front and back = position (relation of occiput to maternal pelvis)
  - Find head = presentation (part foremost in birth canal) and descent (amount of presenting portion in the birth canal)
  - Fetal Heart rate by monitor over fetal shoulder (usually from 10-12 weeks) Normal is 110/120 to 160 bpm.
164: Normal Labour

- **Definition:**
  - Regular contractions (usually 3 in 10 minutes, lasting 40 – 50 seconds)
  - **Cervical change:**
    - More anterior
    - Effaced: depth of ‘rim’ normally 2 cm, 50% effaced = 1 cm
    - Dilated
    - Soft (hard = like forehead, normal = like nose, soft = like chin)
  - +/- Show (mucus plug) or ROM (rupture of membranes)

- **Examination:**
  - Mother: monitor BP (hypo → ?blood loss, hyper → ?pre-eclampsia), pulse, temperature (eg infection if prolonged period post-rupture)
  - Foetal position: by abdominal inspection and palpation. 2/3rds of babies are head first with back on the left. Descent – what portion of the head is below the pelvis (eg 3/5ths)
  - Fundal height:
  - Foetal welfare: Foetal heart, fetal movement, CTG for 20 minutes.

- **Fetal position – Definitions:**
  - Lightening: = baby dropping. → ↓SFH. 1st pregnancy: 2 – 3 weeks before. 2nd pregnancy: may not be till 2nd stage of labour
  - Fetal lie: relation of fetal spine to mother’s spine. Longitudinal (cephalic or breech), transverse, oblique (unstable lie)
  - Fetal presentation: portion of the fetus in the birth canal:
    - Cephalic (96%): vertex, sinceput, brow, face
    - Breech (3%): Frank (extended – ‘foot in mouth’), Complete (knees and hips flexed), Incomplete (footling)
  - Transverse or oblique (1%)
  - Fetal Attitude: “posture” of the fetus, eg extended neck
  - Fetal Position: Relation of occiput (vertex) to the maternal pelvis. Left or Right, Anterior, Posterior, or Transverse, eg

**Stages of labour**

- **Stage 1:** Cervical effacement and dilation - Friedman phase – plot on a partogram:
  - Latent phase (cervical softening). 20 hours in nullip, 14 hours in multip
  - Active Phase: Acceleration phase and deceleration phase (= transition). In primips the cervix dilates 1.0 – 1.2cm/hr and head descends 1cm/hr. In multips cervix dilates 1.5cm/hour head descends at 2cm/hr
  - **Stage 2:** begins at 10 cm dilated and ends with delivery of the baby. 2 hours in primip, 45 minutes – 1 hour in multip.
  - **Stage 3:** separation and expulsion of the placenta
    - Active management of 3rd stage
      - Especially if risk of PPH (big baby/twins/previous PPH/anything that makes the uterus big eg polyhydramnios).
      - Give 5 – 10 units Syntocinon (IV if risk of PPH, IM otherwise) when shoulder delivers
      - Can use syntometrin (oxytocin + a little ergometrine – contraindicated if hypotension)
      - If PPH then IV infusion following bolus (T½ of Syntocinon is 3 – 5 minutes)
      - Complications of Syntocinon: hyperstimulation (→ ↑fetal hypoxia), uterine rupture, water intoxication (Syntocinon is like ADH), uterine muscle fatigue (→ post-delivery uterine atony → ↑risk of PPH)
      - Signs of placental separation: sudden rush of blood, uterus rises, cord lengthens
      - OK to wait if no heavy bleeding. Gentle traction on chord with supra-pubic pressure (stops uterus coming down) or fundal massage and maternal bearing down without traction
      - Can manually deliver (place hand into uterus and separate) – if no haemorrhage then wait for anaesthesia
      - Then inspection, repair, rectal exam
Pain Relief

- Inhalation agent (eg nitrous oxide)
- Epidural: complications – hypotension, urinary retention, total spinal block, prolonged expulsive effort
- TENS
- Narcotics eg pethidine: action lasts 3 hours and can cause fetal respiratory distress – don’t give if delivery expected within 3 hours

168: Premature labour

- = Labour < 37 weeks
- 8% of babies, 85% of neonatal deaths
- Over-diagnosed:
  - Over 80% diagnosed will deliver at term without treatment. Hard to diagnose – regular uterine contractions are normal, cervical changes in labour can be subtle
  - Braxton-Hicks contractions are usual from 30 weeks but are not painful
  - History: Is it true labour: check nature of contractions, urinary frequency (?UTI), backache, spotting or a change in vaginal discharge (normal in 3rd trimester – lots, white, non-smelling).
- Risks:
  - Strongest association is previous preterm birth (↑4 times risk)
  - Previous mid-trimester abortions (2 or more) – not 1st trimester spontaneous abortions
- Investigations: temperature, BP, pulse, SFH, view cervix for clots etc (do NOT view cervix if risk of praevia – do US first)
- Aetiology:
  - Spontaneous: 40%
  - Multiple pregnancy: 10%, ↑10 times risk
  - Maternal or fetal conditions (25%)
  - Premature, preterm rupture of membranes (PPROM)
  - APH
  - > 28 weeks, 80 – 90% survival
  - > 32 weeks, similar survival as term babies but complications
- Management:
  - Consider tocolysis (inhibiting labour):
    - Inhibit uterine contractions – allows time for steroids to work and for transfer to neonatal unit
    - β agonists – Ritodrine and Salbutamol (risk of pulmonary oedema) prolong labour for ~ 24 hours. Adverse effects: maternal and fetal tachycardia, vasodilation → ↓BP. Also a range of other drugs…
    - Contraindications: fetal distress, severe pre-eclampsia, APH, hypotension, tachycardia
    - If cervix is > 4cm then it should be allowed to progress – shouldn’t use tocolytics
    - ↑Not if PROM (premature rupture of membranes). Can → ↑risk of infection
    - Steroids: dexamethasone and betamethasone (crosses placenta, prednisone doesn’t) - 2 shots 12 hours apart. Always give first even if close to delivery. → maturation of lungs if between 24 and 34 weeks (↑surfactant production → ↓fetal distress syndrome) and neonatal better BP control post delivery
    - Delivery. If < 26 weeks then vaginal delivery. C-section more likely if multiple pregnancy or breech. Epidural analgesia preferable to narcotics (→ respiratory depression)

160: Delay in Labour

- See 164: Normal labour for normal length of stages

Abnormal Labour

- = Labour does not progress normally. Due to problems with:
  - Power – eg hypoactive uterine contractions, or hyperactive (eg spasm)
• Passage – disproportion between the size of the pelvis and the fetus (eg scarred cervix)
• Passenger – abnormal lie, presentation, position or structure of the fetus
• Psyche – excessively anxious or sedated mother, conduction (ie epidural) anaesthesia may weaken lower uterine contractions and therefore not assist head rotation and flexion

• Types:
  • Protracted labour – takes longer than normal
  • Arrested labour – progresses normally then stops = no change in cervix for 2hrs or no descent for 1hr. During active stage, progress = either further dilation or further descent
  • Can happen at any stage

• Evaluation:
  • Pulpate or monitor uterine contractions
  • Perform cervical exam
  • Determine lie/position of fetus
  • Review medication and check history

• Treatment:
  • Hypertonic contractions – pain medication, Syntocinon
  • Hypotonic contractions – Syntocinon, AROM (artificial rupture of membranes)

• Risks to foetus in distress:
  • Hypoxia +/- ischaemia
  • Trauma
  • Meconium aspiration (meconium = first stool. Abnormal to find it in amniotic fluid).
  • Infection

Causes of Abnormal labour

• Abnormal Presentations:
  • Breech
  • Face (rather than occiput first). Occurs with complete extension. Mentum (chin) anterior can be delivered vaginally. Don’t use forceps and Syntocinon
  • Brow. Incomplete flexion (midway between face and vertex). Converts to either face or occiput – can’t deliver as brow
  • Occiput transverse: Head can’t flex and rotate from transverse to occiput anterior. Gets stuck at iliac spines.
  • Occiput posterior (ie face up): 5 – 10 %, prolonged second stage, painful labour (lots of back pain), bigger tears and episiotomies
  • Abnormal fetal structure: macrosomia, hydrocephalus, hydrops, meningocoel
  • Pelvic abnormalities: Inlet, mid, or outlet

167: Postpartum Haemorrhage

• Primary PPH:
  • = loss of > 500 ml < 24 hours after delivery
  • Limitations: estimating loss is difficult and loss may be concealed
  • Causes:
    • Uterine atony (90%). Eg in anything that causes large uterus – twins, polyhydramnios, etc
    • Genital tract trauma during delivery (7%)
    • Coagulation defect
  • Management:
    • Resuscitate mother. Test bloods for coagulopathy
    • Rub up a contraction + IV oxytocics
    • Deliver placenta and inspect for completeness
    • Inspect genital tract for trauma. Eg vaginal lacerations, ruptured uterus
    • If bleeding continues ⇒ uterine atony. IM prostaglandins + other procedures

• Secondary PPH:
  • = Loss of any volume of blood > 24 hours and < 6 weeks post delivery. Usually 1 – 2 weeks after
  • Cause: retained placenta/clot, often infected
  • Risk factors: abnormal placentaion or accessory lobes on placenta
• Diagnosis: Ultrasound +/- signs of infection: fever, tender uterus, offensive lochia (discharge after delivery)
• Management: curettage with US guidance + antibiotics (Broad spectrum + anaerobic cover)

• Pharmacology:
  • Syntocinon: action lasts 20 – 30 minutes, causes hypotension, H2O retention, contraindicated in CV disease (eg pre-eclampsia). Used for labour induction or augmentation
  • Ergot alkaloids (eg Ergometrine). For PPH. Causes hypertension and vomiting. Contraindicated in hypertension.
  • Prostaglandin F2α. IM for PPH. Contraindicated in Asthma, CV disease

• Sequalae:
  • Massive bleed → shock and death
  • Puerperal anaemia and morbidity
  • Sheehan’s syndrome: ischaemia of anterior lobe of pituitary → pan-pituitary insufficiency
  • Fear of further pregnancies

• Prophylaxis:
  • Active management of 3rd stage
  • Elective C-section if placenta praevia
  • 50% risk next time, reduces to 20% if active management
  • If at risk, then have active management of 3rd stage labour, have wide bore cannula in place and specialist backup available

165: Normal Puerperium

• = The time in which the reproductive organs return to their pre-pregnant state - usually 6 weeks after delivery.
• Uterus- involutes from 1kg to 100gm. Pelvic organ by about 10 days. Afterpains are felt (especially while suckling) as it contracts.
• Cervix - becomes firm and internal os closes by 3 days. External os closes by 3 weeks.
• Lochia -red for days 1-3 (lochia rubra), yellow until two weeks (lochia serosa), white until six weeks (lochia alba).
• Breasts - milk replaces colostrum by 3 days. Breasts are swollen, red and tender with physiological engorgement at 3-4 days.
• Weight - by 6 weeks usually lose about 60% of weight gained during pregnancy
• Postnatal blues occur in up to 80% before the first two weeks. Weepiness and anxiety but function not usually affected.
• Pregnancy related hypertension should have resolved if mild by 2 weeks, if severe by 6-8 weeks.

170: Puerperal Pyrexia

• = Temp > 38 C on any 2 days in the first 14 days after delivery or miscarriage, exclusive of the first 24hrs. Assumed to be due to infection until proven otherwise. 90% infections will be genitourinary. Caesarean section has 10X risk (10-30%).

Causes

• Endometritis - most common cause. Uterine tenderness on palpation, foul smelling lochia, malaise. May progress to peritonitis with guarding and rebound and then abdo distension and absent bowel sounds. Suspect abscess if spiking temperatures despite ABs.
• Onset in first 24 - 48hrs suspect Strep agalactiae
• Later onset suggests mixed infection
• Fever onset > day 7 suggest Chlamydia.
• UTI - often asymptomatic
• Thrombophlebitis - leg pain, tenderness, swelling. Obese, high parity, age>30, immobile.
• Wound infections - consider the possibility of necrotizing fasciitis - rare but serious.
• Respiratory tract infection - first ask are the symptoms really due to PE?
• Mastitis - usually 2-3 weeks post partum due to S. aureus.
Examination
- Respiratory, breasts, abdominal, legs, pelvic.

Investigations
- Bloods - FBC, culture, creatinine and electrolytes
- MSU - urinalysis and culture
- Pelvis US or CT - locate pelvic abscess or retained placenta
- High vaginal and/or uterine swabs
- CXR

Management (a medical emergency so don't delay)
- General therapy - food and fluids, correct anaemia, analgesia, relief or urinary retention.
- Local therapy - treatment of infected wounds, drainage
- Empirical antibiotic therapy eg co-amoxiclav + metronidazole (+/gentamicin) IV until fever resolves then change to oral therapy.
- If thrombophlebitis suspected add anticoagulant therapy (usually IV heparin)
- Surgery - for drainage of abscess or removal of retained placenta.

146: Postpartum Depression

<table>
<thead>
<tr>
<th>Postpartum Blues</th>
<th>Frequency</th>
<th>Symptoms</th>
<th>Course</th>
<th>Risks &amp; Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>50 – 80%</td>
<td>Weepiness, anxiety, irritability, poor concentration, euphoria</td>
<td>3 – 10 days post partum</td>
<td>Treatment: support, reassurance, monitor. Function not usually affected</td>
</tr>
<tr>
<td>Postpartum Depression</td>
<td>10 – 15 %</td>
<td>Common symptoms: anxiety, spontaneous crying, lack of interest in baby, guilt, hard making decisions, panic attacks, especially insomnia &amp; fatigue. Mood may fluctuate. Obsessional thoughts. Suicidal ideation.</td>
<td>Not before 3rd day. 80% onset within first 6 weeks. Gradual onset – may not be apparent until 4th or 5th month. Duration 6 – 9 months.</td>
<td>30 – 50% chance of recurring in next postpartum period. May approach 100% if history of mood disorder and previous postpartum depression. Treat as for MDE.</td>
</tr>
<tr>
<td>Puerperal or Postpartum Psychosis (No criteria in DSM4)</td>
<td>0.1 – 0.2 %</td>
<td>Severe, liable (unstable) mood. Obsessional thoughts (eg about baby’s health). Suicidal ideation. Delusions (eg baby defective), suspicious, persecuted, confused</td>
<td>Acute onset &lt; 14 days post partum. Good prognosis. Duration 2 – 3 months</td>
<td>30 – 50% chance of recurring in next postpartum period. Treatment: Ensure safety. Antidepressant, Lithium, antipsychotic. Consider ECT early – safe and effective.</td>
</tr>
</tbody>
</table>

- Screen at post-natal check up (6 weeks) using Edinburgh Postnatal Depression Scale (EPDS), but still needs careful clinical assessment
- Differential: hypothyroidism (more common post-partum), recurrence of previous psychiatric illness, initial presentation of a psychiatric illness (eg schizophrenia of anxiety disorders – postpartum period increases risk), adjustment disorder with depressed mood
- Considerable overlap in the risk factors for major depression, post-partum depression, inadequate parenting and child abuse
- Treatment:
  - Check whether drugs enter breast milk.
  - Indications for antidepressants similar for those for other mood disorders
  - If agitated or anxious, more sedating antidepressants are appropriate (eg imipramine) or even small doses of antipsychotics
Paediatrics

121: Neonatal Examination Normal Variants

Initial Examination
- Immediately after birth: APGAR, major congenital anomalies

More complete assessment (in first 24hrs of life)
- Growth – weight, length, head circumference
- General inspection
  - Dysmorphisms- eyes, ears, mouth (cry)
  - Colour- central, peripheral (hands and feet may normally be blue)
  - Respiratory effort- grunting, indrawing, flaring alar nasi
- Posture and movements
  - Normal: hips abducted, partially flexed, knees flexed. Arms adducted, maybe flexed at elbow. Hands closed, not tightly, fingers over thumb, mouth closed unless crying
  - Abnormal: hypotonia/irritability
- Skin- colour, rashes (Mongolian spots, birth marks, naevus flammeus)
- Examination of systems
  - Head: effects of birth (scalp oedema, cephalhaematoma, subconjunctival haemorrhages)
  - Neck- upper airway
  - Chest, cardiorespiratory
    - Breasts often prominent, 10-15mm breast tissue palpable, ‘witches milk’
    - Heart rate reduces from 150-180/min in 1st 15-20 min of life → 90-120 beats/min at rest, and up to 180 /min when crying.
    - Resp rate changes from 60-80 breaths/min in 1st 15-20 min of life → 40-60 /min after 30 min. Breathing can often be irregular (changing rate rapidly), it is difficult/laboured breathing that suggests respiratory problems
  - Abdomen, GI, GU – 3-4cm of palpable liver acceptable in 1st yr of life. Occasionally tip of spleen may be felt. Lower poles of kidneys palpable. Bladder can be briefly palpable when baby about to pass urine. Wide variation size/shape/pigmentation of male genitalia, in girls labia minora and clitoris are often partly exposed.
- Limbs and bones: Hips
- Neurological status
- Neonatal reflexes: Stepping, Walking, Moro, Grasp, Rooting (touch cheek and mouth moves)
- For more detail see Practical Paediatrics, 4th Ed. Chapter 29.

117: Infant Low Birth Weight

Definitions:
- Prematurity: < 37 = weeks Preterm, < 33 = weeks Very preterm
- Birth weight (relevance to Pacific Island Babies – usually heavier):
  - < 2.5 Kg: LBW
  - < 1.5 Kg: VLBW
  - < 1.0 Kg: Extremely low birth weight

Differentials
- Also measure head circumference and length
- Differentiate:
  - Asymmetrical growth retardation (relative preservation of head size)
  - Symmetrical growth retardation
- Consider:
  - Incorrect dates
  - Idiopathic/genetic/ethnic differences
  - Prematurity
  - IUGR
    - Maternal illness:
      - Poor nutrition
- Smoking
- Other maternal illness: PET, gestational DM, antiphospholipid
- Placental insufficiency/twin-twin syndrome, etc
- Fetal illness:
  - Metabolic
  - Congenital heart disease
- Watch for complications of prematurity/low birth weight: eg hypoglycaemia, respiratory distress, impaired sucking/swallowing and gastric emptying (neurologically immature), hyperbilirubinaemia, hypothermia

### 116 and 122: Infant and Neonatal Jaundice

- See section 54: Jaundice, Page 15

### 123: Neonatal Respiratory Distress

#### Differential of Neonatal Respiratory Distress

- Differential of dyspnoea
  - Heart failure
  - Cerebral hypoxia
  - Metabolic acidosis
  - Respiratory Causes
- Upper airway obstruction:
  - Choanal Atresia: failure of formation of nasal passages. Baby goes blue until someone opens the mouth. Can’t pass NG tube. Can be unilateral
  - Congenital masses: nasal encephalocele and nasal dermoid. Care with nasal intubation. Beware the midline lesion
  - Pierre Robin Sequence: short jaw, cleft palate and tongue falls back and obstructs. Nurse prone
  - Subglottic Stenosis: due to intubation trauma in a preterm baby
- Lower airway causes:
  - Lung hypoplasia
  - Diaphragmatic hernia
  - Meconium aspiration
  - Neonatal respiratory distress syndrome (ie prem and ↓surfactant)
  - ARDS
  - Transient tachypnoea of the newborn (TTN) - resolves in 24hrs

### 126: Projectile Vomiting

#### Pyloric Stenosis

- History:
  - 4M:1F
  - From ages 1-3 wks normal GI function, then progressive GI dysfunction aged 3-6 wks.
  - FHx in 15%
  - Vomiting (how much, contents/colour): Occasional spilling initially, followed by several days of non-bilious vomiting. Vomiting is post-prandial, forceful, never bile stained, can be blood stained.
  - Constipation + dryish nappies from dehydration (dehydration depends on amount vomiting/duration of symptoms)
  - Weight loss (Growth Charts)
- Examination:
  - Peristaltic waves may be visible (travel L→R in LUQ)
  - Pyloric tumour palpable in RUQ behind the abdominis rectus muscle (test feed to make easier to palpate, stand on baby’s left and palp with L hand)
  - Weight, nutritional status [see 113. Failure to Thrive]
- Investigation:
  - Electrolytes (may be normal is short Hx), capillary blood gases (for alkalosis)
  - Ultrasound for pyloric tumour
Management:
- Fluid resuscitation and electrolyte correction (MUST do before surgery):
- Maintenance fluid: 1/2 normal saline + 5% dextrose + 10 mmol KCL (in 500mL).
- Replacement fluid: from estimate of degree of dehydration (mucous membranes, fontanelle, tissue turgor) + weight loss. Same kind of fluid as maintenance fluid, infuse the replacement either over 24hrs, or double the maintenance rate until the vol calc for replacement is completed.
- Surgery - pyloromyotomy: post op normal feeds after 24hrs, minor vomiting commonly continues for 1-2 days, Pyloric tumour does not recur.

Differentials
- Gastro-oesophageal reflux: may have similar Hx but usually the babies are well and continue to gain weight and look well fed when present.
- Other causes of vomiting: sepsis, UTIs, meningitis, gastroenteritis, chest infection should be excluded.

Bile stained vomit differential
- Sepsis
- Atresia (below sphincter of Oddi)
- Malrotation with volvulus
- Necrotising enterocolitis
- Obstruction - incarcerated hernia, imperforate anus, Hirschsprung’s

118: Infant Respiratory Distress

Differential of dyspnoea
- Heart failure
- Cerebral hypoxia
- Metabolic acidosis
- Respiratory causes: asthma, bronchiolitis, pneumonia

Differential of Stridor
- Retropharyngeal abscess
- Croup
- Epiglottitis
- Foreign Body
- Angio-oedema
- Peritonsillar abscess
- Laryngomalacia
- Tracheomalacia
- Adenoid and tonsillar hypertrophy
- Tracheitis

Differential of Wheezing
- Common:
  - Asthma
  - ‘Happy wheezer’
  - Bronchiolitis
- Uncommon:
  - Inhalation/foreign body
  - Cystic Fibrosis
  - Heart Failure (+ Sweat when feeding + poor feeding/cyanosis)
  - Aspiration
  - Immune deficiency
  - Can confuse wheezing with soft stridor: eg laryngomalacia. Inspiratory sound
  - Rare congenital causes: cysts, tumours, lobar emphysema, tracheomalacia/bronchomalacia (not properly formed → floppy)
  - Pneumonia
  - Passive smoking contributes to all the above
120: Neonatal Central Cyanosis

**Differentiate if heart or lung disease**
- History and examination
  - Relationship of cyanosis to birth
  - Presence of apnoea
  - Level of respiratory distress
- Investigations:
  - CXR
  - ABG/oxygen saturation monitoring
  - Hyperoxia test
  - Echocardiography

<table>
<thead>
<tr>
<th>Cyanotic CHD</th>
<th>Acyanotic CHD</th>
<th>Respiratory Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe cyanosis</td>
<td>Variable cyanosis</td>
<td>Variable cyanosis</td>
</tr>
<tr>
<td>Mild tachypnoea</td>
<td>Variable tachypnoea</td>
<td>Variable tachypnoea</td>
</tr>
<tr>
<td>No major respiratory effort</td>
<td>Variable respiratory effort</td>
<td>Variable respiratory effort</td>
</tr>
<tr>
<td>+/- heart murmur</td>
<td>+ heart murmur, + CHF</td>
<td>No cardiac signs</td>
</tr>
<tr>
<td>First 1-7 days</td>
<td>First 1-4 wks</td>
<td>Often at birth</td>
</tr>
</tbody>
</table>

**Differentials**
- Hypoventilation: narcotics, apnoea of prematurity, sepsis, metabolic (hypoglycaemia), seizures
- Mechanical interference with lung inflation: upper airway obstruct, abdo distension, pneumothorax/chylothorax, thoracic and sternal deformities, secondary lung hypoplasia
- V-P mismatch with lung disease: transient tachypnoea of newborn, infection, aspiration, hyaline membrane disease, pulmonary oedema, lung haemorrhage, primary congenital abnormalities of lung
- R to L shunt: cyanotic congenital heart disease, persistent pulmonary hypertension of the newborn
- Methaemoglobinemia

**Management**
- Treat the cause

115: Febrile Unwell Child

**History**
- Hx presenting complaint, past medical Hx (multiple congenital abnormalities, neonate, immunocompromised or from a high risk population), immunisation Hx, infectious contacts, overseas travel

**Examination**
- General appearance: level of activity/social interaction, peripheral perfusion and colour. Do they look toxic?
- Vital signs: pulse, respiration, BP
- Exclude: full fontanelle, neck stiffness, respiratory distress, abnormal percussion and auscult findings in chest, otitis, mastoiditis, pharyngitis, rhinitis, lymphadenopathy and hepatosplenomegaly, abdo distension, abdo tenderness/masses, bone and joint tenderness/swelling, skin rashes/petechiae/purpura, skin infection.

**Clues to serious illness in a young infant**
- Responsiveness and activity
- Feeding
- Urine output
- Breathing
- Pallor when febrile
**Differentials**
- Common infections: URTI, otitis media, tonsillopharyngitis, croup, bronchiolitis, gastroenteritis, vaccine preventable diseases.
- Serious infections: meningitis, meningococcaemia, pneumonia, TB, UTIs, osteomyelitis, septic arthritis, cellulitis.

**Investigations**
- FBC, ESR, CRP
- Blood culture
- Culture from any foci of infection
- Urine culture, urinalysis
- CXR
- CSF (lumbar puncture)

**Management**
- As appropriate to cause
- Fluids if dehydrated
- Fever management: dress in light clothing, freq small drinks of water/dilute juice, paracetamol for discomfort of fever >39°C (15mg/kg, 6hrly over next 48hrs, max 90mg/kg/day).

### 114: Febrile Seizure

- See also 74: Seizures, page 54

**Benign febrile convolution**
- 2-5% all children, occur age 6 months-5yrs
- Temp usually >38.5°C
- FHx 1st degree relative 26-31%
- Simple (75%) – generalized, brief (<15min), doesn’t recur within 24hrs.
- Complex (25%) – focal, prolonged, recurrence within 24hrs (16%). Less likely to be benign febrile syndrome.

**Epilepsy (first epileptic fit unmasked by fever)**
- Can’t really diagnose epilepsy on a single seizure

**Acute symptomatic seizures – meningitis, encephalitis, poisoning etc**
- Especially consider in children who seem more unwell than expected, vomiting etc.

**Management**
- Stop seizure (after 5-10min): home – rectal 0.5mg/kg diazepam, hospital- iv diazepam.
- Find cause for the fever and treat
- Lower the temperature
- Then additional to benign febrile convolution:
  - Long-term anticonvulsants – risk usually outweighs benefit, perhaps use intermittently with fever in children who have freq seizures.
  - Parental counselling
    - 30% overall have recurrence, 9% >3, 75% recurrences are within yr after 1st fit
    - Risk factors for recurrences, epilepsy, other outcomes

### 113: Failure to Thrive

**Approach**
- History of presenting complaint
  - Diet (WHAT GOES IN)- what, how much, if complicated involve dietician
    - Take through a day of what the child eats incl. snacks
    - Who feeds them?
  - Assess parent’s knowledge base: Budget limitations and food prep skills
  - Poos (WHAT COMES OUT)- steatorrhoea
• Chronic illness - cardiac, renal, neurological
• Vomiting
• Past medical History – ABFWIMPS (Anti-natal, birth, feeding, weight, immunisations, milestones, past medical history, social):
  • Guthrie card done?
  • Growth charts: help pinpoint start of problem, in FTT due to poor nutritional intake they lose wt before linear growth declines and head circumference growth is last to decline. Severe reduction in head circumference suggests that there has been intrauterine growth retardation.
• Development
• Social Hx- postnatal depression
• Examination
  • Nutritional assessment
  • End of bed- Fat/thin, energy, pallor
  • Muscle and fat stores: Muscle wasting- quadriceps, gluteal muscles
  • Integument, teeth, bones and eyes (for signs of vitamin/trace element deficiencies)
  • Signs of abuse or injury: Neglect- child appropriately dressed? Dirty body? Widespread/ulcerating nappy rash?
  • Signs of chronic disease
  • Assess suck, chew, swallow (are they a drooler?)
  • Observe interaction of parents with child
• Differentials
  • Non-organic FTT: feed well and gain wt when admitted to hospital, Ix don’t reveal organic cause
  • Organic causes:
    • Failure of intake:
      • Cannot eat: short bowel syndrome, sucking/swallowing/chewing/weakness problems, other GI anomalies (may need NG tube or gastrostomy), dyspnoea, neuro, anorexia due to organic disease
      • Will not eat: behavioural, psychological, family issues
    • Abnormal losses: vomiting (pyloric stenosis), stools, urine
    • Abnormal utilisation: chronic infection, metabolic disorders, endocrine disorders, constitutional/genetic/intrauterine lesions.
• Management
  • Note: possible that parents concerned because, especially in the 2nd year of life, appetite reduces and activity increases and weight gain rate slows. If growth charts normal (i.e. no growth problem) reassure parents and discuss this normal change.
  • If uncertainty about intake/growth or more complex problem admit to hospital for observation assess management (monitor intake and weight gain while in hosp)
  • Investigations:
    • FBC (including MCV)
    • Urine culture and microscopy
    • CXR
    • Anti-endomysial and antigliadin antibodies (depending on history - only see coeliac disease after exposure to gluten)
  • Management of non-organic FTT
    • Knowledge, attitudes, practices, barriers.
    • Validate, educate, encourage, reinforce.
    • GET HELP: usually need intensive support and monitoring needs to be guaranteed.
  • For more detail see Chapter 19- Failure to Thrive, in ‘Practical Paediatrics 4th Ed’

119: Napkin Dermatitis

Main differentials
  • Note: these may occur in combination
  • Contact irritant dermatitis:
Clinical features- erythema on convexities (folds spared). If skin allowed to dry becomes glazed, then cracking and scaling appears. Localised, thick walled vesicles appear on buttocks and pubis ⇒ punched out ulcers (heal without scarring). In circumcised males meatal ulceration can occur.

Management- thick ointment/paste (zinc cream/paste) to make inert barrier to protect skin. Frequent changes to contact time with soiled nappies. If severe inflammatory add mild steroid cream (hydrocortisone 0.5-1%). Anticandidal agents if candida also present. Time out of nappies.

Candida:
Clinical features- likes damp skin, is common in infants (nappy area, in mouth esp. inside cheeks, hands if sucked). Skin lesions whitish, erythema, moist, satellite lesions. Affects perianal fold and central flexures, symmetrical distribution. Increased risk where broad spectrum antibiotic use common. Oral steroids may contribute. Can lead to systemic spread in immuno comp.

Management- oral nystatin or ketoconazole and nystatin cream to affect ed skin for 7-10 days. If oral candida + breast-feeding will need to treat mother’s nipples

Seborrhoeic dermatitis:
Clinical features- can involve scalp, eyebrows, forehead, retro-auricular fold, folds of neck, axillae, groin (bipolar distribution – top and tail). In the scalp there is little erythema. In the intertriginous areas that erythema is more marked and there may be maceration. Scaling (greasy) may occur in the scalp and on the face, but is absent in the intertriginous areas. Is not the same as adult seborrhic dermatitis, most are atopic dermatitis in infancy and the rest are infantile psoriasis.

Management- same as atopic dermatitis in infancy. Avoid scratching, reduce irritants (e.g. soaps and perfumes), moisturise skin (emollients e.g. aqueous cream, white soft paraffin), mild hydrocortisone 1% ointment not cream (reduces inflammation, doesn’t address cause).

If severe consider Leiner’s disease (complement deficiency), plus other immune def assoc.

Psoriasis:
Clinical features- occurs as nappy rash in the infant. May occur after a napkin irritant dermatitis, also triggered by systemic and local infections. FHx freq in early onset cases. Appears as sharply marginated erythematous plaques with scale that persists. Psoriasis may be found elsewhere including in the flexures.

Management- 0.5% crude coal tar useful for napkin psoriasis. For mild cases mild steroid creams for face and flexures, stronger steroid creams for trunk/limbs, coal tar creams, dithranol, daivonex ointment. In severe psoriasis UVB, PUVA, methotrexate, neotigason, azathioprine.

Other differentials to consider for napkin eruptions
Atopic eczema, miliaria (heat rash fades rapidly on cooling), infection (herpes, Staph, molluscum), acrodermatitis (Zn def), congenital syphilis, scabies, insect bites, granuloma gluteum infantum (following strong steroid cream use).

Other differentials to consider for napkin eruptions
Atopic eczema, miliaria (heat rash fades rapidly on cooling), infection (herpes, Staph, molluscum), acrodermatitis (Zn def), congenital syphilis, scabies, insect bites, granuloma gluteum infantum (following strong steroid cream use).

Shake hands with Parent - put them at ease, complement their baby/child

General question - Any worries about your child’s development?
Ask about child’s vision/hearing
Tell me about your child’s vision/hearing?
How do you know they can see/hear?
What small or distant things/quiet sounds can your child see/hear?
Run through developmental categories: Gross motor, Fine motor-adaptive, Expressive language, Receptive language, Personal-Social skills and play, Self care skills
Use the format:
I notice your child is.... (item from Gross motor skills). What other clever things like that is he/she doing?
• Run through Gross motor developmental milestone items in order, getting the child to demonstrate if possible, until you get to two items in a row that the child can’t yet do. This is the ceiling for Gross motor development.

• Summarise Gross motor development by saying “so your child can do... (items that child is able to do from Gross motor category), but hasn’t quite yet mastered...., so his/her Gross motor developmental age is.... .

• Note down child’s Gross motor developmental age.

• Repeat above for Fine motor-adaptive, Expressive language, Receptive language, Personal-Social skills and play, and Self care skills.

• Estimate child’s age - average of ages determined for each category.

• Emphasise strengths (categories where developmental age is above average) and summarise weaknesses.

• Ask the parent what they think they can do to help improve child’s weaknesses. Help them plan strategies.

• Promote general safety/anticipatory guidance

112: Faecal Soiling

• Interview child and parents separately

History

• Of the soiling

• Assoc. constipation/withholding/absence of warning

• Toileting behaviour

• Associated behaviours e.g. hiding soiled underwear

• Parent’s management style

• Medications, general illnesses

Examination

• General growth and development

• Inspection of perineum

• Inspection of lumbosacral area, neuro exam of lower limbs

• Abdo exam (PR usually NOT necessary)

Differentials

• Exclude medical causes of constipation e.g. Hirschsprung’s, spina bifida/sacral agenesis/hypothyroidism/hypercalcaemia.

Management

• Explain normal anatomy/function of rectum and anus

• Explain pathology: UNEXPELLED FAECES → rectal distension → depressed sensory reception → no urge → training ceases → hard stools (water reabsorption) → pain/fissure → fear + withholding → further retention of stool → UNEXPELLED FAECES

• Explain management plan (reasons behind it)
  • Structured toileting programme: toilet for 10 min after each meal, rewards for sitting, diary.
  • Simple laxatives: before give oral laxatives need enema to clear out hard stools
  • High fibre diet and adequate fluids
  • Frequent visits (for support of both parents and child)
  • Refer if major psychopathology

111: Childhood Immunisation

• Questions could take the form of:
  • a parent with questions about immunisation
  • anticipatory guidance (e.g. at 6 week check)
  • if wanting child immunised may need to ask questions around contraindications.
**Approach**

- Help parent clarify exactly what they want to know.
- Knowledge, attitudes/fears, practices, barriers, motivation (then summarise this back to them)
- Validate, reinforce, education, reinforce again.
- Where parent is on “readiness to change” model influences the information you will give them.
- Expected to know:
  - The schedule
  - Genuine and non-genuine contraindications to immunisation
  - Some common myths and be able to discuss the risks vs the benefits of immunisation

**The schedule**

<table>
<thead>
<tr>
<th>Route</th>
<th>DTaP-IVP</th>
<th>HepB-Hib</th>
<th>HepB</th>
<th>MMR</th>
<th>DTaP/Hib</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IM (vastus lateralis)</td>
<td>IM (vastus lateralis)</td>
<td>Oral</td>
<td>SC (upper arm)</td>
<td>IM</td>
</tr>
<tr>
<td>6 weeks</td>
<td>*</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 months</td>
<td>*</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 months</td>
<td>*</td>
<td></td>
<td>*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15 months</td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td></td>
</tr>
<tr>
<td>4 years</td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td></td>
</tr>
</tbody>
</table>

**Contraindications**

- Genuine contraindications:
  - Systemically unwell or temp > 38.5
  - Live vaccines contraindicated in immunosuppressed (e.g. malignancy, those on chemotherapy), pregnancy. Note immunosuppressed may still receive inactivated vaccines
  - Sever reactions that contraindicate further DTP doses: encephalopathy within 7 days. Immediate severe allergic/anaphylactic reaction (applicable to any vaccine).
  - If recently had blood transfusion or Ig dosing may have to postpone giving live virus vaccines
  - In children where there are contraindications or history of worrying reactions discussion with or referral to a paediatrician is important before further immunisation.

- Non-genuine contraindications:
  - **Family History**: Adverse event following vaccination, epilepsy.
  - **Age**: Prematurity. Over the age recommended in the schedule.
  - **Medical conditions**: Stable neuro condition e.g. cerebral palsy, Down’s. Asthma, eczema, hay-fever or allergy. Contact with infectious disease. Jaundiced at birth.
  - **Treatments**: Antibiotic treatment, inhaled or low-dose topical steroids, replacement steroids. Recent or imminent surgery.
  - **Other**: Child’s mother is pregnant, child is breast fed. Belief in homeopathy.
  - **Certain common Side Effects of previous vacs**: local swelling/redness, fever and irritability with pertussis vacs. Fever, malaise and rash with MMR.

- Common myths:
  - Vaccination does not cause asthma, SIDS, IBD or autism.
  - also see “Answers to Common Questions” in MOH Immunisation Handbook

- Vaccine reactions
  - Common (10-15%): local swelling, redness, pain, fever - reduce with paracetamol
  - Uncommon: persistent crying (>3hrs). 1:100fever > 40.5 1:300
  - Rare: <1:1000 high pitch cry, seizure, hypotonic hypo-responsive episode, anaphylaxis <1/million encephalitis or poliomyelitis
## Benefits and risk

<table>
<thead>
<tr>
<th>Vaccine preventable disease</th>
<th>Risk of infection</th>
<th>Risks of disease</th>
<th>Risk of vaccine</th>
<th>Vaccine efficacy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hep B</strong> Subunit vaccine</td>
<td>Contagious, via blood/sexual transmission. Vertical transmission.</td>
<td>Chronic carrier state in 90% newborns, 25% young children. 25% carriers → chronic active hepatitis and cirrhosis (25% of these → hepatocellular carcinoma in 50s).</td>
<td>5% local redness and tenderness</td>
<td>85-90%</td>
</tr>
<tr>
<td><strong>Diphtheria</strong> Toxoid vaccine</td>
<td>Contagious, spread by nasal droplets</td>
<td>Severe sore throat, fever, cervical lymphadenopathy. Life threatening comps: Up to 25% upper airway obstruction, toxin induced myocarditis Approx 75% neuro problems (vocal cord paralysis, ascending paralysis)</td>
<td>Local reactions and mild fever may occur.</td>
<td>&gt;80%</td>
</tr>
<tr>
<td><strong>Tetanus</strong> Toxoid vaccine</td>
<td>Organism present in soil and animal faeces, can be inoculated into trivial wounds.</td>
<td>Muscle rigidity, painful spasms. NZ case fatality 10%. Mortality in advanced cases 60%.</td>
<td>Local reactions may occur.</td>
<td>Approx. 100%</td>
</tr>
<tr>
<td><strong>Pertussis</strong> Acellular subunit vaccine</td>
<td>Highly contagious (nasal droplets, coughing) Regular epidemics every 3-5 yrs. Infection risk universal without vac</td>
<td>Nasal discharge, cough, may ⇒ whooping cough. Cough persisting 3-4 months complications: 1-3% anoxic seizures 0.1-0.3% encephalopathy. Children &lt;1 yr: 80% hospitalised, 0.2% die</td>
<td>50% local reaction/fever. Irritability common. Adverse reactions less common in DTaP (10-15%). 1/ million assoc with encephalopathy (no causal link estab)</td>
<td>Whole cell (DTPw) 60-90%. DTaP may be higher.</td>
</tr>
<tr>
<td><strong>Hib</strong> Subunit vaccine</td>
<td>Contagious, nasal droplet (before vac was common cause life threatening bacterial infection in under 5s)</td>
<td>Acute febrile illness, may ⇒ Meningitis (5% die, 10% severe sequelae, 20-30% functional disabilities). Epiglottitis (1% die). Pneumonia, empyema, sep arthritis, peri orbital/facial cellulitis</td>
<td>Few SEs, &lt;5% mild local reactions</td>
<td>&gt;95%</td>
</tr>
<tr>
<td><strong>Polio</strong> IPV: injectable subunit vaccine</td>
<td>Spread by faeces, saliva</td>
<td>Fever, headache, nausea, vomiting. 1% severe muscle pain, neck + back stiffness ⇒ paralysis. 5-10% case fatality, 50% who survive poliomyelitis have permanent paralysis</td>
<td>&lt;1% diarrhoea, headache, muscle pains. 1/ 2.5 mill recipients/close contacts develop paralysis (immuno sup more common), does not occur with IPV</td>
<td>Live attenuated &gt;90% after 3 doses</td>
</tr>
<tr>
<td><strong>Measles</strong> Live attenuated vaccine</td>
<td>Contagious, nasal droplets, cough. 100% risk infection without vac.</td>
<td>Fever, nasal discharge, cough, conjunctivitis, rash. Complications: 0.5% mortality 10% otitis media 1-5% pneumonia</td>
<td>10% local discomfort 1% malaise, mild fever, rash 1/1 mill encephalitis</td>
<td>65% at 9 mo 95% 15 mo (seroconver)</td>
</tr>
</tbody>
</table>
Epidemics every 7 yrs.
0.1% encephalitis (15% die, 25% severely disabled)
1/100 000 subacute sclerosing panencephalitis
(1000x less likely than with wild virus)

**Mumps**
Live attenuated vaccine
Contagious, saliva
Epidemic cycles every 3-4 yrs
70% fever, swollen salivary glands. 15% aseptic meningitis,
0.2% encephalitis
20% post-pub males painful orchitis
0.02% case fatality
Rare. 1% swelling of salivary glands
1/3 mill mild encephalitis
Mumps
Live attenuated vaccine
Contagious, nasal droplets
Fever, headache, conjunctivitis, 50% rash, generalised lymphadenopathy.
50% adolescent/adults frank arthritis, arthralgia
1/5000 encephalitis
90% 1st trimester embryos abortion/major congenital abnormalities
10% local discomfort.
5% adolescents/adults arthralgia, 1% non-infectious rash.

| 124: Non-Accidental Injury |

**History**
- Disclosure - child, sibling, abuser or other parent
- Physical signs
- Behavioural signs - fear, aggression, attachment disorder
- Developmental signs
- Parent/family signs:
  - No history given for injury, history changes, partial history
  - Unbelievable explanation (account not compatible with developmental age or injury)
  - Unreasonable delay in seeking medical help
  - Previous similar episodes
- Risk factors: e.g. child handicapped/behaviourally difficult, poor parental mental health, substance abuse, domestic violence.

**Examination**
- General assessment:
  - Growth, conscious state, child’s behaviour and play behaviour, language.
  - Appearance and interaction with parents. Parent’s affect/behaviour abnormal or concerning.
- Head
  - Bruising
  - Ears: bruising, ruptured ear drum
  - Eyes: retinal haemorrhages, periorbital bruising
  - Mouth: torn frenulum, teeth, dental hygiene
- Chest: Bruising/other skin lesions, tenderness over ribs, pneumothorax
- Abdomen: Bruising/other skin lesions, acute abdo: ruptured liver/spleen/gut
- Limbs: Skin lesions (bruises/burns/cuts/abrasions), #s
- Genital area and buttocks: bruising, bleeding, expert should do exam in suspected sex abuse
- Developmental: delay, consistence with history
- Review of systems: any evidence for other causes e.g. coagulopathy

**Differentials**
- Bruising: Mongolian spots, coagulopathies and coin rubbing
- Cigarette burns: bites or vesicles
- Hot fluid burns: may be non-intentional (accidental)
- Fractures: osteogenesis imperfecta, pulled elbow, spiral #s of tibia in the toddler
Investigations
- FBC and coagulation studies- for all significant bruising
- Consider specific examinations by ophthalmologist, ENT surgeon
- CXR, limb x-rays as appropriate +/- skeletal survey and/or bone scan
- CT if head injury suspected
- Consider urine toxicology

Management
- Documentation important: body chart, measure lesions, explanations for lesions, photos
- Team approach: doctor/paediatrician, CYPFS, Police, social workers, Court
- Role of doctor:
  - Responsibility to report (not mandatory), no authority themselves to investigate
  - Hx, thorough exam, clear/accurate records, provide a report if required
  - Evidence of findings in court of law
  - Ensure appropriate follow up for child and family
  - Can arrange admission to hospital to aid safety of children, no legal authority to protect children
Consent - Being adequately informed about a health procedure, given a choice, no coercion.

Surgical Consent
- **Why** is the operation needed: eg to reduce pain and to reduce complications
- **Alternatives**: what will happen if you don’t
- **How**: Explain the procedure:
  - General anaesthetic
  - Detail of surgery, including where the incisions will be
  - For example, why they can get by without their gallbladder
- **What to expect**: time in hospital, time off work, future function
- **Risks**
  - General risks: bleeding, infection, damage to adjacent structures, DVTs, anaesthetic risk
  - Risk specific to procedure eg bile leak

See Right 7, the ‘Code of Health and Disability Services Consumers’ Rights’
- Consent must be informed ie effective communication is the key. Communication should be:
  - Clear - in the patient’s native tongue if possible (interpreter).
  - Verbal and written - depending on seriousness of procedure.
  - Open ie why certain tests are being done should be explained to the patient without alarming them unnecessarily.
  - Age-appropriate
- Information must be given by a person competent to give the information and be given at the right time and in the right place
- Regardless of age, race, etc, an individual must be able to understand:
  - That they have a choice
  - Why they are being offered a treatment/test/intervention
  - What is involved
  - Probable benefits, risks, side effects, failure rates
  - Alternatives

Specific to HIV testing
- Specific consent should always be obtained before testing for evidence of HIV infection. General consent to admission to hospital or to a treatment procedure is not enough.
- Pre-test counselling
  - Purpose of testing for anti-HIV (often called an HIV test)
  - How long the results will take to return
  - Meaning of a negative ‘HIV test’. Inc explanation of ‘window period’(approx 1 mth)
  - Meaning of a reactive (positive) screening anti-HIV test. 1-2/1000 false positive
  - Meaning of a positive (confirmatory) anti-HIV test. (Western blot)
- A patient giving consent for anti-HIV testing should be made aware of:
  - Medical implications of a positive anti-HIV test result
  - Psychological issues - counselling support needs to be offered
  - Notification of AIDS. HIV is not a notifiable disease, AIDS is.
  - Social implications
  - Implications for insurance
  - Indeterminate anti-HIV Western blot tests - repeat testing in 4-8 weeks
  - Preserving confidentiality - coded blood specimen
  - Future preventive aspects
  - How the results are to be obtained
  - Any financial costs involved
- Post-test counselling
  - Giving the results of the tests **must** be done in person
  - Explanation of test results - the result and its medical significance
  - Negative test result
• Absence of detectable antibody
• Possible significance of a window period
• Changes in lifestyle behaviours
• Positive test result
• Repeat specimen to exclude identification error
• Arrangements for counselling and support

31: Discuss Screening Test

• See also 173: Abnormal Cervical Smear, page 132

**History**

• Smear - LMP, abnormal bleeding, post-coital bleeding, abnormal discharge, smoking.
• Mammography - age, past history of breast disease, family history.

**Discuss benefits of screening**

• Cervical smears 98-100% success rate for treatment of pre-cancers. Women most likely to get cervical Ca are those not regularly screened. All women between 20-69 who have been sexually active should be screened.
• Mammography reduces chances of dying from Ca by about 33%. Less accurate if <50 or on HRT. All women aged 50-64 should be screened (its free) and at younger age for strong family history of breast Ca.

**Discuss disadvantages of screening**

• Uncomfortable or embarrassing.
• Anxiety created by false positives.
• May give false negatives. This means that actual abnormalities are missed.
• About 10% of breast cancers are not detected on mammography, therefore if you find a breast lump you should still go and see your doctor.
• For mammography may also get scarring as a result of diagnostic open biopsies.

**Explain what the screening test involves.**

• Smear - discuss feelings and views about smear test. Explain where cervix is. Explain the procedure and show equipment used. Might be uncomfortable but rarely is it ever painful. Give option for a female smear taker (where possible). Smear does not equal STI check although this can be done at the same time if you discuss it with your smear taker first.
• Mammography - X ray of breasts. The breast is flattened between two plates to improve the accuracy and reduce amount of radiation needed. Most women have some discomfort and some find it painful (<10%). Tell the radiographer if this is the case.

**Explain what a positive result means (ie a screening test does not give a diagnosis)**

• Smear - trying to find precancerous lesions. Abnormal smear result is rarely cancer, but may indicate cell changes that may (but not always) become cancer in the future. 80% of smears are normal. 13% show inflammation or infection. 7% show abnormal cells.
• Abnormal mammography does not necessarily mean Ca, but indicates that further investigation is needed. Less than 1 in 10 women with an abnormal mammogram will have cancer.

**Discuss what happens if result is positive**

• Abnormal smear - If low grade repeat smear in 6/12. If high grade refer for colposcopy. (See 173: Abnormal cervical smear)
• Abnormal mammogram - referral for further investigation. This may be USS, FNA, core biopsy, open biopsy or wire localisation. If cancer is found then Rx involves removal of lump or breast, lymph nodes and possibly radiotherapy.

**Discuss how they will be notified about result**

• Smear - a letter from the national register after your first smear and then only if you have an abnormal smear. Can also find out result from your smear taker.
• Mammography - notified by letter from mammography service.
Benefits of being on the cervical smear national register

- Reminder if you or your GP forget that you are due
- Enables tracking when moving from part of the country to another part.
- Information monitoring and evaluation
- Stand-alone system so not linked to any other health records.

Consider giving written info and time to think about it and decide.

78: Starting Medication

- Understanding the purpose of the medication is vital to informed consent and to adherence to treatment

Drug History

- Drug history: For each drug they’re on or have recently taken:
  - Name of drug and dose
  - Why did you start
  - Why did you stop
  - Did it help
  - Any side effects
  - What drugs have you had previously for the same condition
- Using any Oral Contraceptives, OTCs, herbal remedies, supplements
- Medical history
  - Are you pregnant or breast-feeding
  - Do you have any medical illness, especially:
    - Liver disease
    - Kidney disease
    - Bleeding disease
    - GI disease (e.g., malabsorption, chronic diarrhea)
  - Do they have any drug allergies
  - Any family history of problems with medications
  - Social history: Do you smoke or drink

Starting a new drug

- Used the drug before or know anyone else who has: any misapprehensions?
- On any other medications: check interactions
- Check contraindications: pregnant or breastfeeding?
- Allergies?
- Smoke or drink?
- For the drug to be prescribed:
  - Why the medication is needed
  - Name of drug and how it works
  - Dose and how to take (when and with/without food)
  - What it is expected to achieve
  - How long until an effect should be noticed
  - When to stop/likely duration and any requirements when stopping (e.g., anti-depressants – taper off)
  - Restrictions: eg no alcohol with metronidazole, no grapefruit juice with OCs
  - Possible side effects
  - Whether the medication is addictive
  - Importance of continuing with other non-drug treatment
  - Questions?
  - Arrange follow up
- What are the alternatives (see Management Options page 7)

Compliance

- Few people take their medication as prescribed. Non-compliance is multi-factorial:
  - Rapport with doctor
  - Not knowing how to take medication
• Not understanding the importance of the drugs
• Taking many drugs
• Anticipation or experience of side-effects
• Forgetfulness
• Impaired physical function or other disability
• Community and family support

• Strategies for improving compliance:
  • Education about disease and treatments (spoken instructions are quickly forgotten)
  • Simplifying drug regimes (fewer drugs and fewer doses)
  • Involving carers
  • Education about common side effect which may subside
  • Use of drug diaries, calendars, medication charts
  • Use of ordinary bottle tops (not childproof) for elderly people
  • Large print labels
  • Dosage forms (eg small pill size, pleasant taste)
  • Compliance aids such as pill trays and blister packs
  • Return or destruction of old drugs

**99: Perform a Medline Search**

**The question**
• Written as: The patient and the problem, the intervention compared with the control and the clinical interest. Eg "In patients over the age of 70 with AF does aspirin compared with no treatment reduce the incidence of stroke."

**The search terms**
• Enter the term and select the most appropriate MeSH term(s) that it is mapped to.
• Click on the term to show the tree. Choosing "explode" (exp) will include the selected MeSH term as well as all terms further down the tree in your search.
• Choosing "focus" (*) will only select those articles in which the search term has been judged to be the main topic of the article. (NB - "explode" and "focus" are completely independent operations and can be used simultaneously.)
• Subheadings - specific headings that are useful include drug therapy (dt) and diagnosis (di).
• Useful search terms are (exp prognosis) and (exp "sensitivity and specificity").

**The combinations**
• Search terms may be combined by "and" or "or" operations.

**The Limits**
• Left until the end. Normally you will always have to limit to "human", "English", years and possibly also sex.
• Limiting to a specific journal, author or title is done by clicking on the separate icons at the top of the main search page

**94: Make a Referral to an Allied Health Professional**

When referring a patient to an allied health professional it is advisable (but usually not essential) to be polite at all times (eg saying please and thank you) and to share the full details of the case with the professional concerned.

It is also advisable to NOT make demeaning remarks about the inadequacies/lack of competence of the allied health profession or its training programme even if you believe this to be true. Likewise, passing comment on the allied health professional’s relative poor pay compared with that of a doctor is best avoided. Discussion about BMWs, Jaguars and expensive international holidays should be kept to the minimum.

Please note that these guidelines do not apply when dealing with dentists.
Sample Questions

Station 17

Instructions to the Student

You are a trainee intern at the antenatal clinic and your next patient is Mrs DM, who is 25 years old and in her second pregnancy. She is presently at 28 weeks gestation with a blood pressure of 150/95 and 2 plus of protein in the urine.

Take a relevant history from Mrs DM pertaining especially to the high blood pressure.

At 4 minutes you will be stoped and asked to outline suggestions about management.

Instructions to the Simulated Patient

The student will be given marks for the way that s/he establishes rapport with you as well as for the “medical content” of the answers.

If the student asks you a closed-ended question, then you should reply to that question only. If the student asks you an open-ended question, you should feel free to expand on the answer you give.

Do your best to put yourself in the actual role of the patients and respond as you might in that situation. If you feel uncomfortable, for example, you would offer less information.

You are Mrs DM, in your second pregnancy with high blood pressure. The student will take a history from you pertaining especially to the high blood pressure. You have had high blood pressure in your previous pregnancy and this was first noted at around 28 weeks. You have not had high blood pressure in between your pregnancies or in the first 20 weeks of this pregnancy. You have attended antenatal care regularly since 6 weeks gestation.

In the first pregnancy you needed induction of labour at 34 weeks because you had excessive pain and vaginal bleeding. Your blood pressure was also too high and could not be controlled by medication. You had a normal vaginal delivery of a healthy boy weighing 1.5 kg.

Presently you feel well and do not have a headache, feel nauseated or dizzy, do not have visual disturbances or epigastric pain.

Apart from anxiety about the pregnancy you feel well.

Questions which the examiner will ask the patient when the student has left

1. How well did you understand the student?
   Very: 1  Moderately: 0.5  Not at all: Nil

2. How likely would you be to come back and discuss your concerns with this student again?
   Very: 1  Moderately: 0.5  Not at all: Nil