Child becomes pale, falls to the floor, brief seizure, rapid recovery. Episodes are due to cardiac asystole from vagal inhibition.

- Syncope
- Migraine
- Benign paroxysmal vertigo: recurrent attacks of vertigo, associated with nystagmus, unsteadiness or even falling. Due to viral labyrinthitis.

**Childhood Epilepsy:**
- Incidence = 0.05%
- Prevalence = 0.5%

**Generalized seizures:**
- Discharge arises from both hemispheres.
- Can be:
  - Absence
  - Myoclonic
  - Tonic
  - Tonic-Clonic
  - Astatic
- Infantile spasms:
  - 4-6 months of age
  - Violent flexor spasms of the head, trunk and limbs – followed by extension of the arms.
  - 2/3rds have an underlying neurological cause.
  - EEG: hypersrhythmia = Chaotic pattern of high-voltage slow waves, and multi-focal sharp wave discharges.
  - Treatment: vigabatrin or corticosteroids.
  - Most infants affected will lose skills and develop learning disability or epilepsy.
- Lennox-Gastaut syndrome:
  - 1-3 years
  - Usually drop attacks, tonic seizures or absence seizures.
  - Neurodevelopmental arrest or regression, behavioural disorder.
  - Usually Hx of infantile spasms.
  - Prognosis is poor.
- Typical (petit mal) absence seizures:
  - 4-12 years
  - Stare momentarily – stop moving – may twitch, FGs or hand minimally.
  - Last: 30-60 seconds
  - No recall, though do realize they have missed something.
  - 2/3rds are female.
  - 95% are in remission by adolescence.
  - 5-10% may go on to develop tonic-clonic seizures in adult life.
- Juvenile myoclonic epilepsy:
  - Adolescence-adulthood
  - Myoclonic
  - Usually shortly after waking – typical Hx is throwing drinks or breakfast around at this time.
  - Response to treatment is usually good, but treatment is required lifelong.

**Focal seizures:**
- Frontal seizures:
- Involve the motor cortex
- Can get clonic movements that travel proximally – Jacksonian march
- Can get asymmetrical tonic seizures – may be bizarre and hyperkinetic
- Temporal lobe seizures:
- Most common of all epilepsies
- Usually last longer than typical absence seizures
- Strange warning feelings e.g. aura with smell or taste
- Automatisms
  - Lip-smacking
  - Plucking at one’s clothing
  - Walking in a non-purposeful manner
- Deja-vu and jamais-vu (feeling of never having been in the same situation before)
- May have impaired consciousness.
- Occipital seizures:
- Distortion of vision.
Altered Consciousness

AVPU
A – Alert
V – Responds to voice
P – Responds to pain only
U – Unresponsive

First line management:
- A, B, C
- Control the C-spine if there is suspected trauma
- Give O2, even if you suspect they are breathing normally
- Look for obvious injuries, rashes etc
- Get an early bedside blood glucose measurement

Glasgow Coma Scale:
- Eye opening: all ages
  o E4 = spontaneous
  o E3 = to voice
  o E2 = to pain
  o E1 = none
  o C = eyes closed e.g. by swelling or a bandage
- Verbal: >5 years
  o V5 = orientated
  o V4 = confused
  o V3 = inappropriate words
  o V2 = incomprehensible sounds
  o V1 = no response to pain
  o T = intubated
- Verbal: <5 years
  o V5 = alert, babbles, coos, words or sentences to usual ability
  o V4 = less than usual ability, irritable cry
  o V3 = cries to pain
  o V2 = moans to pain
  o V1 = no response to pain
  o T = intubated
- Motor: all ages
  o M6 = obeys commands, or normal spontaneous movements
  o M5 = localises to supraorbital pain (>9 months of age) or withdraws to touch
  o M4 = withdraws from nailbed pain
  o M3 = flexion to supraorbital pain (decorticate)
  o M2 = extension to supraorbital pain (decerebrate)
  o M1 = no response to supraorbital pain (flaccid)

Assess and reassess!
A score <8 indicates a deep coma and inability to protect the airway.

Other CNS assessment tools:
- Pupils: size, reactivity, symmetry
- Focal tone and movement
- Signs of raised ICP e.g. HTN, bradycardia, Cheyne-stokes respiration (Cushing’s triad), fixed dilated pupil.
- Fundoscopy
- Hx – development, seizures, medications

Differential Diagnosis:
- Acute global encephalopathy:
  o Toxic-metabolic causes
    - Hypoglycaemia/DKA
    - Inborn errors of metabolism
    - Intoxication
  o Hypoxic-ischaemic
  o Infectious
  o Seizures
- Trauma e.g. diffuse axonal injury
- NAI
- Focal encephalopathy:
  - Trauma
  - Stroke/infarct
  - SOL e.g. haematoma, tumour, abscess

Investigations:
- CT/MRI scan of the brain – early scan is good to rule out surgical pathology, mass effect
- Intoxication screen?
- Consider NAI as a possibility.
- When is it safe to do an LP?
  - Must have stable ABC
  - Avoid if there is risk of herniation:
    - Deep coma
    - Focal signs
    - Signs of increased ICP
  - **A normal CT scan does not imply low ICP

Empirical therapy:
1. IV dextrose if the BM is unavailable
2. Abx – try to culture first though
3. Antiviral if the Hx and signs are suggestive

Common mistakes to avoid:
1. Don’t forget about ABC
2. ABCs and GCS can evolve quickly – assess and reassess!
3. BM must be checked early
4. LP is rarely indicated in the acute stage
5. Always remember to consider NAI and intoxication.
**Childhood screening**
- 6-8 weeks: examination by GP
- 2-2.5 year: developmental assessment by health visitor
- By 5 years:
  - Hearing test: audiometry
  - Vision: screening by orthoptist, Snellen chart and cover test.
  - Measure height, weight. Plot centiles.

**Immunisation programme**

**Immunisation programme**
- **Birth**
  - BCG: if at risk
  - Hep B: if mother positive for HepBsAg
- **2 months**:
  - 5 in 1 (DTaP, IPV, HIB) 1st
  - PCV 1st
  - Rota (oral) 1st
- **3 months**
  - 5 in 1 2nd
  - Rota (oral) 2nd
  - Men C 1st
- **4 months**
  - 5 in 1 3rd
  - PCV 2nd
- **12-13 months**
  - MMR 1st
  - HIB (as 2-in-1) 4th
  - Men C (as 2-in-1) 2nd
  - PCV 3rd
- **3-5 years**
  - 3-in-1 preschool booster (DTaP, IPV) 2nd
  - MMR
- **12-13 years**
  - HPV (3 injections over 6 months), girls only
- **13-18 years**
  - DT/IPV
  - Men C 3rd

**Types of vaccine**
- Diphtheria = inactivated toxin
- Tetanus = inactivated toxin
- Pertussis = acellular pertussis
- Hib = conjugate
- Polio = inactivated
- Rotavirus = live, attenuated
- MMR = live, attenuated
- Pneumococcal = conjugate
- Men C = conjugate
- HPV = subunit

**Immunisation side effects**
- Local reaction: erythema, swelling, pain
1. Left-to-right Shunts

**Atrial Septal Defects (ASD)**
- 2 main types of ASD:
  - Secundum ASD – 80%
    - Defect in the centre of the atrial septum, involving the foramen ovale
  - Partial AVSD/primum ASD – 20%
    - Defect of the atrioventricular septum
    - Characteristics:
      - Inter-atrial communication between the bottom end of the atrial septum and the AV valves (primum ASD)
      - Abnormal AV valves, left AV valve has three leaflets and tends to leak (regurgitant valve)

**Presentation:**
- Often have no symptoms
- May get recurrent chest infections/wheeze
- Heart failure
- Arrhythmias – usually 4th decade onwards
- **Physical Signs**
  - Fixed, widely split 2nd heart sound, this is due to the right ventricular SV being equal in both inspiration and expiration
  - Ejection systolic murmur best heard at the upper left sternal edge – this is due to increased blood flow across the right ventricular outflow tract, due to the left-to-right shunt
  - With a partial AVSD/primum ASD – can get apical pansystolic murmur from the AV valve regurgitation.

**Investigations:**
- CXR: cardiomegaly, enlarged pulmonary arteries, increased pulmonary vascular markings.
- ECG:
  - Secundum ASD: partial RBB is common, right axis deviation due to right ventricular enlargement.
  - Partial AVSD/primum ASD: left axis deviation, due to a defect in the middle of the heart where the AV node is, the displaced node will then conduct to the ventricles superiorly and give an abnormal axis.
- Cross-sectional echocardiography

**Management:**
- If significant, they will require treatment.
- Secundum ASD:
  - Cardiac catheterisation – insert an occlusion device
- Partial AVSD/primum ASD:
  - Surgical correction is required
  - Usually perform catheterisation at 3-5 years old, as this is to prevent the development of right heart failure and arrhythmias in later life.

**Ventricular Septal Defects**
- Account for 30% of cases of congenital heart disease.
- The defects are usually perimembranous (adjacent to the tricuspid valve), or muscular (completely surrounded by muscle).

**Small VSDs:**
- Smaller than the aortic valve in diameter i.e. <3mm
- Clinical features:
  - Usually asymptomatic
  - May have thrill at the lower sternal edge
  - Loud pansystolic murmur at the lower left sternal edge
  - Quiet pulmonary 2nd sound
- **Investigations:**
  - CXR: usually normal
  - ECG: normal
  - Echo: can demonstrate the anatomy of the defect, can also look at the haemodynamic effects using Doppler.
- **Management:**
  - Most lesions close spontaneously.
  - While the VSD is present, need to prevent bacterial endocarditis by maintaining good dental hygiene and abx prophylaxis before dental extraction.

**Large VSDs:**
- Same size or bigger than the aortic valve.
- **Clinical features:**
  - HF with breathlessness, FTT after 1 week old
  - Recurrent chest infections
Cyanotic Congenital Heart Disease

Cyanosis can be due to:
1. Decreased pulmonary blood flow with a right-to-left shunt e.g. tetralogy of Fallot
2. Abnormal mixing of the systemic and pulmonary venous return e.g. transposition of the great arteries and tricuspid atresia.

Tetralogy of Fallot:
- Most common cause of cyanotic heart disease

Clinical Features:
1. Large VSD
2. Overriding of the aorta with respect to the ventricular septum.
3. Subpulmonary stenosis – narrowing of the right ventricular outflow tract
4. Right ventricular hypertrophy

Symptoms:
- Most tetralogy is diagnosed antenatally or after the identification of a murmur in the first month or two of life.
- Rarely can get hypercyanotic spells – SOB, pallor, irritability and inconsolable crying – can lead to MI, cerebrovascular accidents and death if left untreated.

Signs:
- Clubbing
- Loud, harsh ejection systolic murmur at the left sternal edge from day 1 of life
  o May have a single second heart sound – A2
- With increasing obstruction of the right ventricular outflow tract, the murmur will shorten and the cyanosis will increase.

Investigations:
- CXR:
  o Usually normal
  o In an older child – relatively small heart, uptilted apex/boot shape due to the right ventricular hypertrophy.
  o May be a pulmonary artery ‘bay’ = a concavity on the left heart border, where the pulmonary artery and right ventricular outflow tract are normally positioned.
  o May be reduced pulmonary vascular markings.
- ECG:
  o Normal at birth
  o Right ventricular hypertrophy later
- Echo:
  o Can show the cardinal features and anatomy

Management:
- Corrective surgery at 6 months of age:
  o Close the VSD
  o Relieve the outflow obstruction with an artificial patch
- Infants who are very cyanosed in the neonatal period may require a shunt to increase pulmonary blood flow – tube between the subclavian artery and the pulmonary artery = modified Blalock-Taussig shunt. Or they may need balloon dilatation of the right ventricular outflow tract.
- Hypercyanotic spells lasting >15 minutes need prompt treatment:
  o Sedation and analgesia
  o IV propranolol – alpha adrenoceptor agonist – peripheral vasoconstrictor and relieves the subpulmonary muscular obstruction that is causing the reduced pulmonary flow.
  o IV fluids
  o Bicarbonate to correct the respiratory acidosis
  o Intubation and artificial ventilation to reduce metabolic O2 demand.
Infective Endocarditis:

- The highest risk of infective endocarditis is when there is turbulent jet blood flow e.g. with a VSD, coarctation of the aorta and PDA, or if prosthetic material has been inserted.
- Should be suspected in any child or adult with sustained fever, malaise, raised ESR, unexplained anaemia or haematuria.

Clinical Signs:
- Fever
- Anaemia and pallor
- Splinter haemorrhages in the nailbed.
- Clubbing (a late sign)
- Necrotic skin lesions
- Changing cardiac signs
- Splenomegaly
- Neurological signs secondary to cerebral infarction
- Retinal infarcts
- Arthritis/arthralgia
- Haematuria (microscopic)

Diagnosis:
- Blood cultures – most commonly Streptococcus viridans = alpha haemolytic streptococcus
- ECHO:
  - Can confirm the diagnosis by identifying vegetations, but can never exclude it.
    - Vegetations = fibrin and platelets and infective organisms
- Acute phase reactants will be raised.

Management:
- Bacterial endocarditis: high dose penicillin + aminoglycoside: 6 weeks therapy
- If there is an infected prosthetic material, will likely need surgical removal.

Prophylaxis:
- Good dental hygiene
- Abx prophylaxis for:
  - Dental treatment
  - Surgery that is likely to be associated with bacteraemia.