FLUIDS & ELECTROLYTES

Total Body Water

a. TBW is higher

			ECF	ICF
i.	prem	~ 85%	**45%**	40%
ii.	term	~ 75%	40%	35%
iii.	12/12	~ 60%	27%	33%
iv.	adult	~ 60%	**20%**	40%

b. both fat and muscle content increase with age

c. decreases in TBW are predominantly due to ***ECF decreases***

d. as ECF decreases, ICF increases ~ 35% → ~ 43% @ 3 mth

e. predicted ***body weight*** < 9 yrs ~ (2 x age) + 9
> 9 yrs ~ 3 x age

• higher proportion of TBW in younger children cf. adults is due to their relatively larger ***ECF***
• organs with more ECF (skin and brain) are a higher proportion of body weight, and those with more ICF (muscle and viscera) are a lower proportion
• ***obligatory water loss*** in urine depends on,

1. endogenous renal ***solute load***
   - proportional to caloric expenditure and VO_2, which are higher in infants
2. renal ***concentrating ability***
   - limited ability to dilute / concentrate urine cf. adult,
   i. infant ~ 200-800 mosm/l
   ii. adult ~ 80-1200 mosm/l

• this, combined with a higher solute load (VO_2) and higher insensible losses, makes infants more prone to develop water deficits

Daily Water Requirement	
Day 1	~ 2 ml/kg/hr[1]
Day 2	~ 3 ml/kg/hr
Day 3 - 12 Months	~ 4 ml/kg/hr[2]
10 - 20 kg	~ 40 + 2 ml/(kg>10)/hr
> 20 kg	~ 60 + 1 ml/(kg>20)/hr

[1] kcal/kg/hr can be substituted for ml/kg/hr

[2] some say 120 ml/kg/day for day 4 and over

- ***Sodium Requirement***

a. days 1 & 2 - low urinary Na^+ loss & high insensible water losses
→ risk of ***hypernatraemia***
→ use 5-10% dextrose

b. ≥ 3 days → 2-4 mmol Na^+/kg/day

- ***Potassium Requirement***

NB: similar to Na^+ → ~ 2-4 mmol K^+/kg/day
≤ 0.5 mmol/kg/hr
* absence of anuria / severe oliguria

• therefore, example of maintenance fluids might be,

a. day 1 → 5% or 10% dextrose at 2 ml/kg/hr

b. ≥ day 2 → 5% dextrose + Na^+ 40 mmol/l
K^+ 20 mmol/l

@ 4 ml/kg/hr

Elemental Requirements

sodium	2-6	mmol/kg/d
potassium	2-4	mmol/kg/d
calcium	0.5-1	mmol/kg/d
magnesium	0.5-1	mmol/kg/d
phosphate	0.4-1	mmol/kg/d
glucose neonates[1]	10-15 ~ 20	g/kg/d g/kg/d

[1] glucose 20 g/kg/d ~ 80 kcal/kg/d
~ 80% of energy requirement

NB: ***glucose*** requirement → ~ 4-6 mg/kg/min S.K.
~ 6-8 g/kg/day

~ 6-8 mg/kg/min N.M. for neonates

Replacement Solutions and Composition			
Solution	%	mmol/ml	Infusion rate
NaCl	20 %	3.4	~ 0.6 x TBW x (125-[Na^+])
KCl	7.5 %	1	0.5 mmol/kg/hr
$NaHCO_3$	8.4 %	1	~ 0.5-2.0 ml/kg ∝ BE & pH
$CaCl_2$	10 %	0.68	0.1-0.2 mmol/kg/hr
Ca-gluconate	10 %	0.22	as above
$MgSO_4$	49.3%	2	0.4 mmol/kg/hr

- ***Clinical Assessment***

a. ***mild dehydration*** **~ 5%** loss of body water
- thirsty, irritable
- poor tissue turgor
- dry mucous membranes

b. ***moderate dehydration*** **~ 10%** loss of body fluid
as 5% plus,
- tachycardia, oliguria
- sunken fontanelles
- poor capillary refill

c. ***severe dehydration*** ³ **15%** loss of body water
as 10% plus,
- hypotension, anuria
- tachypnoea
- sunken eyeballs
- skin mottled, cold peripheries
- diminished / absent peripheral pulses

NB: ≥ 20% may result in ***coma***

- ***Investigation***

a. body weight = ***best guide***

b. serum [Na^+] ~ water balance

i. urine Na^+ < 20 mmol/l = hypovolaemia

ii. urine Na^+ > 40 mmol/l
+ oliguria = ATN, renal failure, etc.

Adjustment Factors for Fluid Requirements	
Decreased	
• hypothermia • high ambient humidity	- 12% / °C
• head injury • IPPV (ADH) • high ADH • paralysis (decreased BMR)	x 0.7
• inactivity	
• IPPV with humidified gases	x 0.75
• renal failure	x 0.3 + urine output
• SIADH	
Increased	
• hyperthermia	+ 12% / °C
• ambient temperature > 31°C	+ 30% / °C
• radiant heater, phototherapy	x 1.3
• motor activity • air currents • low ambient humidity	
• age (preterm infant ~ 1.0-1.5kg)	x 1.2
• hyperventilation	x 1.2
• dry or cool inspired gases	
• burns - day 1 - day 2 & after	+ (4x $\%SA_{burn}$)% + (2x $\%SA_{burn}$)%

Management - Hypovolaemia/Dehydration

a. give adequate volume of colloid/crystalloid to restore circulatory status
   - NSA-5% ~ 10-20 ml/kg
   - SPPS used to cause vasodilatation (diluted HSA-conc to 5% OK)
   - if no response to 20 ml/kg then presume other cause for hypotension & consider insertion of a CVC line

b. IVT = deficit + maintenance + ongoing losses

c. replace ***deficit*** over next 24 hrs
   - ~ ½ deficit over 8 hrs, remainder over 16 hrs
   - if hypernatraemic, then replace over 48 hrs

- ***Hyponatraemia***

a.	renal loss	- poor renal conservation
b.	breast milk	- low Na^+ content
c.	inappropriate ADH	- IPPV /CPAP - head injury or CNS disease - respiratory disease
d.	excess water intake	
e.	hypotonic IV fluids	

NB: → ileus
hypotension
listlessness
± convulsions

R_X	H_2O excess	- water restriction ± Na^+/frusemide
	Na^+ deficit	- hypertonic saline (20% = 3.4 mmol/ml)
		- correct to [Na^+] ~ 125 mmol/l at ≤ 2 mmol/l/hr
	mmol Na^+	~ **0.6** x (**125** - [Na^+]) x weight

- ***Hypernatraemia***

  a. dehydration
  b. inadequate fluid intake
  c. diarrhoea
  d. radiant heaters
  e. osmotic diuresis
  f. $NaHCO_3$

  NB: ± can be associated with ***hyperglycaemia*** & ***hypocalcaemia***

  R_X normal saline ~ 10-20 ml/kg to correct ***volume deficit***,
  then correct water deficit over 48^+ hrs (rapid → ***cerebral oedema***)
  → fall in $[Na^+] \leq 2$ mmol/l/hr

- ***Oedema***

  a. premature
  b. excess fluid intake
  c. inappropriate ADH
     - CNS or lung disease
     - IPPV
     - serum osmolality ≤ 270 mosm/l
     - urine osmolality > 270 mosm/l
  d. capillary leak
     - hypoxia, acidosis
     - ischaemia, sepsis
  e. heart failure
  f. renal failure
  g. hypoalbuminaemia
  h. multiple of above

  NB: R_X fluid restriction ± diuretics
  albumin / blood volume replacement
  dialysis

- ***Hypocalcaemia ± Hypomagnesaemia***

  a. "sick neonates" within first few days of life

  b. neonate of diabetic mother

  c. large volume IV fluids

  d. exchange transfusion with citrated blood (transient)

  e. diarrhoea

  f. cows milk feeding * high phosphate content

     → jitters, tetany, cardiac arrhythmias & convulsions

  g. normal daily requirement,

     i. Ca^{++} ~ 1.0 mmol/kg/day

     ii. Mg^{++} ~ 0.3 mmol/kg/day

     iii. R_X - maximum rate of 0.1 mmol/kg
     - $CaCl_2$ = 0.68 mmol/ml
     - Ca gluconate = 0.22 mmol/ml

  NB: * rickets is not uncommon in small preterm neonates

- ***Rx Hyperkalaemia***

  a. calcium ~ 0.1 mmol/kg

  b. HCO_3^- ~ 1.0-2.0 mmol/kg

  c. glucose ~ 0.5-1.0 g/kg
     insulin ~ 0.1 U/kg

  d. cation exchange resins - Resonium 1 g/kg
     ± sorbitol 1.5 mg/kg

  e. dialysis

- ***Pyloric Stenosis***

  a. gastric fluid composition

Na^+	~	80	mmol/l
K^+	~	20	mmol/l
H^+	~	30-120	mmol/l
Cl^-	~	150	mmol/l

  b. methods of assessment

     i. body weight change

     ii. clinical assessment of % dehydration

     iii. Cl^- deficit = 0.5 x body weight x (110-[Cl^-])/110

     iv. urinary Cl^- excretion

Nutrition

- a survey of hospitalised paediatric patients demonstrated evidence of acute malnutrition in 30%
- the critically ill child has problems of decreased intake and increased metabolic demands →

    a. poor wound healing

    b. reduced immune response

    c. lack of growth

    d. reduced energy and protein stores

- the metabolic requirements of children are higher and the stress response results in a drain of energy and protein stores

    → increased utilization of glucose, glycogen and fat
    except in sepsis where this utilization is impaired

- the aim of nutritional support is to provide ordinary caloric requirements, as well as those needed for growth and development, without fluid retention
- assessment of appropriate caloric assimilation is difficult,

    a. body size - weight, height, & head circumference

    b. tissue composition - skinfold thickness

    c. biochemical & immunological parameters
        - creatinine/height index
        - albumin, transferrin
        - CMI by skin testing and lymphocyte count

- however, a simple nutritional assessment system is required because those suggested for adults ***have not*** proved useful in paediatrics
- fat administration prevents essential fatty acid deficiency and when metabolised produces less CO_2, which may be important in patients with respiratory distress
- there are recommended daily allowances for vitamins and minerals in children
- daily monitoring of ***caloric intake*** is important
- the choice of caloric administration (enteral or parenteral) depends on disease processes and adequacy of gut function

Daily Nutritional Requirements			
Carbohydrate	10-15 ≤ 20	g/kg/d neonates	4.1 kcal/g
Protein	2-3	g/kg/d	5.3 kcal/g
Fat	1-3	g/kg/d	9.3 kcal/g
Newborn 1 year 7 years 12 years 18 years	120 90 75 60 30	kcal/kg kcal/kg kcal/kg kcal/kg kcal/kg	CHO ~ 65% Protein ~ 10% Fat ~ 25%
Synthamin 17 (g-N) is 10%		= 100g protein/1000ml ~ 0.5 kcal/ml	
→ 2.0 g/kg/day		= 20 ml/kg/day	

• disease processes requiring ***increased*** caloric expenditure are,

a. fever
b. surgery
c. sepsis
d. cardiac failure
e. respiratory failure
f. burns
g. malnutrition

■ ***Enteral Nutrition***

• enteral feeding maintains better gut function and has less complications
• diets include,

a. homogenised food - causes less diarrhoea and abdominal distension
b. formula - with added calories (as CHO) if volume is limited
c. elemental diets - simple sugars, AA's, elements
- where digestive ability is limited
± abdominal distension / diarrhoea

• ***nasal tubes*** are difficult to maintain long term, and obstruct the nares resulting in an increase in work of breathing which is important in the presence of respiratory failure

Parenteral Nutrition

• parenteral nutrition is required where enteral feeds are precluded because of disease or surgery
• most common indications are for,

a. primary gastrointestinal diseases
   - short bowel syndrome
   - inflammatory bowel disease

b. supportive therapy for prematurity

c. necrotizing enterocolitis

d. neoplasia

e. burns

f. pre- / postoperatively
   - small bowel atresia
   - TOF
   - gastroschisis ± omphalocole
   - diaphragmatic hernia

• long term central venous administration is via percutaneous or surgically inserted small bore silicone catheters
• peripheral administration has fewer complications and is technically easier, but has limitations in the amount of calories that can be delivered
• also, problems with long-term IV maintenance in children
• when given intravenously, glucose, protein and fat should be introduced slowly over 3-4 days
• monitoring is aimed at assessing the effects of therapy and avoiding complications,

a. daily
   - weight, temperature, ? fluid overload
   - catheter related problems
   - ***glycosuria***

b. 3x / week
   - electrolytes and glucose

c. 2x / week
   - urea, creatinine, Ca^{++}, Mg^{++}, phosphate

d. 1x / week
   - LFT's, Hb, triglyceride levels (when fat emulsion is used)
   - head circumference and length

• technical, infectious, metabolic and psychiatric complications are similar to those in adult patients
• decreased fat clearance reduces capillary blood flow and affects white cell and platelet function
• thus, lipid is relatively contraindicated in,

a. liver disease

b. bleeding disorders

c. pulmonary hypertension

d. premature neonates

e. sepsis

• serum lipaemia and triglyceride levels should be frequently monitored when fat is commenced or clinical conditions change

- ***Caloric Requirement TPN***

a. 5% dextrose (100 ml/kg/day) ~ 20 kcal/kg/day
~ 1/5th of the basal requirement

b. many ill neonates/small children are unable to absorb adequate nutrients from the GIT
→ institute TPN early

c. nutrient solutions for paediatric use have high concentrations of Ca^{++}, Mg^{++} & $PO_4^{=}$
→ incompatible with fat emulsion

d. dislodged canulae / unavailable solution should be supplemented immediately to prevent ***rebound hypoglycaemia***

e. complications include
   i. line related problems
   ii. hyperglycaemia / glycosuria
   iii. rebound hypoglycaemia
   iv. extravasation, tissue necrosis
   v. hypoproteinaemia, hyperlipidaemia
   vi. electrolyte imbalance, acidaemia
   vii. uraemia, cholestatic jaundice
   viii. sepsis

CONGENITAL HEART DISEASE

1.	***incidence***	~ 6-8:1000	live births	
2.	***acyanotic***	~ 25%	VSD	(30)
		~ 17%	PDA	(10)
		~ 7%	ASD	(7)
3.	***cyanotic***	~ 11%	Fallot's tetralogy	(5)
		~ 8%	transposition	(5)
		~ 3%	tricuspid atresia	
4.	***obstructive***	~ 7%	PS	(7)
		~ 6%	coarctation	(6)
		~ 4%	AS	(5)

- ***Classification***

1.	***obstructive***	- aortic stenosis - pulmonary stenosis - coarctation of the aorta - interrupted aortic arch - aortic atresia - mitral atresia & stenosis - cor triatriatum (accessory LA)
2.	***increased*** pulmonary blood flow	- ventricular septal defect - patent ductus arteriosus
	acyanotic	- ASD, ostium secundum / primum type - total anomalous pulmonary venous connection - complete atrioventricular canal - truncus arteriosus - aortic pulmonary window - ruptured sinus of valsalva - LV to RA shunt - coronary arterial fistula
3.	***decreased*** pulmonary blood flow	- tetralogy of Fallot - pulmonary atresia with intact ventricular septum
	cyanotic	- tricuspid atresia - Ebstein's anomaly - hypoplastic right ventricle - transposition of the great arteries - "corrected" transposition of the great arteries - double outlet right/left ventricle - single ventricle - cardiac malposition

4. ***miscellaneous*** cardiac lesions
   - congenital heart block
   - congenital mitral insufficiency
   - anomalous left coronary artery
   - pulmonary arteriovenous fistula
   - endocardial fibroelastosis
   - cardiac tumours

Initial Management

NB: treatment is aimed at improving ***oxygenation*** and ***cardiac output*** to enable stabilisation and transfer to a tertiary unit

- marked ***cyanosis*** presenting in a newborn is usually caused by CHD
  a. P_{aO2} ~ 40-60 mmHg is well tolerated
  b. commonly P_{aO2} ~ 30 mmHg

- ***acidosis*** reflects failure of oxygen transport
- however, ***oxygen*** is only helpful where there is,
  1. an element of ventilation/perfusion mismatch, or
  2. pulmonary hypertension

- positive pressure ventilation, muscle relaxation and sedation reduce work of breathing and help left ventricular performance, provided venous return is not reduced or the lungs overdistended

- where a ***patent ductus arteriosus*** is required for maintenance of,
  a. pulmonary blood flow - right to left shunts, or
  b. systemic blood flow - coarctation
     - hypoplastic left heart syndrome

  intravenous PGE$_1$ (~ 0.01 µg/kg/min) can be life-saving

OBSTRUCTIVE CONGENITAL HEART DISEASE

Pulmonary Stenosis

a.	incidence	~ 7% of CHD - males ~ females
b.	pathology	~ 95% = valvular stenosis - most have a ***patent foramen ovale*** - few have a true ASD - some have a hypoplastic RV
c.	clinical symptoms	- usually none and normal growth
	severe lesions	- dizziness, hypoxic spells - cyanosis and right sided failure - anterior chest pain ± angina - sudden death
d.	signs	- high pitched SEM ± click - RV heave - delayed and soft S_2
e.	ECG	~ 50% RVH ± strain, RAD
f.	CXR	- RVH - oligaemic lung fields
g.	operative indications	- gradient ≥ 50 mmHg → open pulmonary valvotomy + closure of foramen ovale - if hypoplastic RV leave FO open
h.	complications	- RVF - cyanosis, respiratory failure ~ 50% of deaths occur in the 1st year

Aortic Stenosis

- four types of aortic stenosis are recognised,

a. valvular aortic stenosis * most common

b. subvalvular aortic stenosis

c. supravalvular aortic stenosis

d. asymmetrical septal hypertrophy

- ***Valvular Aortic Stenosis***

a. incidence — ~ 7% of CHD
- predominantly in ***males***

b. pathology
- the valve is frequently ***bicuspid***
- aorta and aortic annulus are small
~ **20%** → ***associated CHD***

c. clinical symptoms — - usually none, with normal growth

severe lesions
- LVF or syncope
- anterior chest pain ± angina
- sudden death

infants
- cyanosis with severe LVF
- respiratory distress
- poor ventricular function 2° to,

i. subendocardial ischaemia

ii. endocardial fibroelastosis

d. signs
- SEM at LSE ± click
- may be absent in severe LVF
- LV heave

e. ECG — - LVH ± LV strain, ischaemic changes

f. CXR
- usually normal or show only LVH
- the ascending aorta may be dilated

infant
- the cardiac outline is large
- pulmonary venous congestion present

g. operative indications → ***commissurotomy***
≥ 50 mmHg gradient
- symptoms of syncope, LVF
- ECG changes of ischaemia

- unless associated AI it is rarely necessary to insert a prosthetic valve in a child
- thus, they suffer from progressive thickening and calcification of the valves, requiring continued follow-up ± repeat operations

h. complications
- LVF, pulmonary oedema
- angina, IHD ± MI
- respiratory failure
- sudden death
- re-stenosis postoperatively

- ***Subvalvular Aortic Stenosis***

- is caused by a discrete fibromuscular segment of the LV outflow tract
- this is seldom seen in infants
- it has a good prognosis as operative resection of the band is possible and recurrence is uncommon

Supravalvular Aortic Stenosis

- this is usually an ***isolated lesion***, not associated with mental retardation or genetic defect
- however, **~ 20%** of patients known to have supravalvular stenosis, also show,
    a. mental retardation
    b. "elfin facies"
    c. strabismus
    d. dental anomalies
    e. narrowing of the pulmonary & peripheral systemic arteries
    f. many with hypercalcaemia

- the aorta has an "hour-glass" deformity just above the valve, which may be improved with a prosthetic patch

Asymmetrical Septal Hypertrophy

- disease of cardiac muscle and results in disproportionate thickening of the ventricular septum
- ***autosomal dominant*** inheritance ~ 50% familial
- the muscle mass may, or may not result in ***outflow obstruction***
- the severity of any obstruction increases during systole and is proportionate to,
    a. the inverse of the LVES volume
    b. the force of contraction
    c. the cross sectional area of the LV outflow tract

- physiological events associated with increased catecholamines or SNS activity worsen obstruction, as do pharmacological agents with sympathomimetic action
- the common symptoms are,
    a. chronic fatigue
    b. episodes of syncope and angina
    c. dyspnoea on exertion

- ***operative resection*** is frequently difficult due to the diffuse nature of the muscle disease
- LBBB frequently follows operative resection

Coarctation of The Aorta

a.	incidence	**~ 10%** of CHD * ***males*** ~ 2x females
b.	associated with	- Marfan's syndrome - Turner's syndrome - ***berry aneurysms*** ~ 25-50% have ***bicuspid valve*** (ie. develop AS later)
c.	site	~ 98% distal to the left subclavian artery ~ 2% proximal (ie. to isthmus)
d.	clinical symptoms	- headaches, epistaxis - lower limb weakness, cramps, claudication - congestive failure
e.	signs	- upper limb hypertension, LV thrust - weak femoral pulses - radio-femoral delay - collateral circulation - scapulae, post. intercostals - axillae, epigastrium - hypertensive retinopathy
f.	murmurs	- collateral bruits - crescendo/decrescendo ESM - AS/ESM $\propto$ bicuspid valve - S_3, S_4 with loud S_2 & LVH
g.	ECG	~ 50% LVH ± strain
h.	CXR	- LAH, LVH - prominent left subclavian - "3 sign" $\equiv^{t}$ pre/post-dilatation - ***notching*** of ribs 3-7
i.	complications	- malignant hypertension - CVA / SAH - LVF - endocarditis

• in most patients, blood flow to the lower extremities is not reduced at rest
• however, pulse pressure and exercise tolerance are significantly reduced
• in ***infants***, coarctation may produce severe LVF and there is a high incidence of associated anomalies, particularly ***PDA*** and ***VSD***
• untreated, the first year ***mortality*** ~ **75%**
• many children are asymptomatic and undergo normal development
• operative repair is indicated as soon as practicable, before hypertension & secondary vessel changes occur
• ***residual hypertension*** after operative frequently remains a problem
• re-stenosis & re-operation is less common after patch repair than end-to-end anastomosis

CHD WITH INCREASED PULMONARY BLOOD FLOW

~ 50% of all CHD shunt blood from the systemic to the pulmonary circulation
- the most common in this group include VSD, PDA, atrial defects and atrioventricular canal
- factors which contribute to this include,

    a. thicker walled, less compliant LV

    b. SVR ~ 10 x PVR

    c. mean LV & systemic pressures are ~ 8x RV & pulmonary

- the increased pulmonary blood flow results in,

    a. vascular congestion

    b. ↑ RV work load ± ***RV failure***

    c. ↑ frequency of respiratory ***infections*** & ***growth retardation***

    d. ↑ pulmonary vascular pressures & PVR

        → ↑ mean PAP ~ 2x with a 3x increase in flow

    e. ↑ LAP & LVEDP *ventricular interdependence

    f. ↑ lung water

- the rise in PVR is at first passive, ***hyperkinetic pulmonary hypertension***
- later this progresses to pulmonary vascular disease & progressive hypertension,

    1. stage 1 - muscular hypertrophy of the media of arterioles
    2. stage 2 - proliferation of the intima
    3. stage 3 - hyalinization & fibrosis of the media and adventitia

- these changes are more likely with lesions associated with large increases in flow and ***pressure***,

    a. VSD

    b. complete AV canal

    c. truncus arteriosus

- residence at high altitude and chronic hypoxaemia also favour its development
- patients with advanced pulmonary disease and reversal of shunt flow, ***Eisenmenger's syndrome***, cannot be helped by operation
- ***pulmonary banding*** is a palliative technique to reduce pulmonary flow
- however, the addition of a ***fixed resistance***,

    1. is detrimental under any physiological condition which would increase flow
    2. becomes inadequate with growth

ASD - Ostium Secundum

a.	incidence	- secundum defects are the ***commonest*** ASD ~ 2% of CHD (~ 95% of total ASD's)
b.	pathology	- defects in the region of the ***fossa ovalis*** - may be single or multiple - usually largest of the atrial defects
c.	signs/symptoms	- usually asymptomatic and acyanotic - normal growth & development - RV lift - S_2 widely split and fixed - grade 1-3/6 pulmonary ESM (murmur ***is not*** from ASD flow) - diastolic flow murmur at lower LSE - CCF rare in children but occurs in adults
d.	complications	- infective endocarditis - paradoxical embolism - arrhythmias, increasing with age - progressive PVD and RV failure are relatively ***rare***

ASD - Ostium Primum

a.	incidence	- uncommon
b.	pathology	- defect occurs during development of the ***AV canal*** ~ incomplete AV canal - defect is located low in the atrial septum - aortic leaflet of the ***mitral valve*** is usually cleft ± MR
c.	signs/symptoms	- usually asymptomatic and acyanotic ± dyspnoea on exertion - S_2 widely split and fixed - frequently apical SEM - diastolic flow murmur at lower LSE - CCF more common than with secundum defect
d.	ECG	* characteristic → LAD with frontal QRS ~ 0 to -60°
e.	complications	- mitral regurgitation & progressive CCF → major determinant of long term prognosis - infective endocarditis - paradoxical embolism - arrhythmias, increasing with age - progressive PVD and RV failure > ostium secundum

Complete Atrioventricular Canal

a.	pathology	- deficient atrial & ventricular septa - also deficient mitral & tricuspid valves - major shunting of blood at ventricular & atrial levels - usually with mitral regurgitation ± tricuspid regurgitation
b.	signs/symptoms	- biventricular heart failure common in infancy - loud S_2 with fixed splitting - blowing, pansystolic murmur ± other bruits - cardiomegally on CXR & examination
c.	catheter	- "gooseneck" deformity of mitral valve and LV outflow tract
d.	ECG	- LAD with frontal QRS ~ 0 to -60°
e.	complications	- progressive PVD, LV & RV failure are very common - severe CCF early requiring therapy - infective endocarditis, paradoxical embolism - arrhythmias, increasing with age
f.	postoperatively	~ 5% develop CHB - result depends upon AV valve tissue present - many with residual MI - late pulmonary vascular disease ± requiring mitral valve replacement

Ventricular Septal Defect

a.	incidence	**~ 25%** of CHD
b.	pathology	~ 85% occur in the ***membranous septum*** - conduction bundle is close to these ~ 10% are defects of the muscular septum - occasionally may have associated AI
c.	signs/symptoms	= those of pulmonary overcirculation ± dyspnoea on exertion, fatigue & poor weight gain ± CCF, frequent respiratory infections - often asymptomatic and acyanotic (small) - loud S_2 with fixed splitting - grade 2-6/6 pansystolic murmur → LSE - apical diastolic flow murmur - biventricular enlargement if large defect & hyperaemic lung fields
d.	ECG	± LBBB
e.	complications	- biventricular CCF - frequent respiratory infections - progressive PVD → operate earlier - infective endocarditis & arrhythmias

Patent Ductus Arteriosus

a.	incidence	**~ 17%** of CHD
b.	pathology	- failure of normal ductal closure - prematurity $\equiv^{t}$ ***persistent foetal circulation*** ± hypoxia, hypercarbia, acidosis
c.	signs/symptoms	± those of pulmonary overcirculation - often asymptomatic
	infants $\rightarrow$	± dyspnoea on exertion, fatigue & poor weight gain ± CCF, frequent respiratory infections - loud S_2 with fixed splitting - bounding peripheral pulses ($\downarrow$ SVR) - systolic ± continuous murmur at base - hyperaemic lung fields
d.	complications	- infants may $\rightarrow$ biventricular CCF - frequent respiratory infections * a large ductus & progressive PVD are ***unusual***
e.	risk of SBE	- lesions more common on the ***pulmonary side*** of the ductus
f.	R_X	- most close spontaneously without R_X - indomethacin inhibits synthesis of $\boldsymbol{PGE_1}$, works in ~ 1/52 - surgical ligation * ***no*** requirement for AB prophylaxis post-ligation

CHD WITH DECREASED PULMONARY BLOOD FLOW

• the combination of obstruction to RV outflow and a septal defect results in reduced pulmonary blood flow and R→L shunt
• the degree of shunt flow is inversely proportional to pulmonary blood flow
• common causative lesions include,

1. tetralogy of Fallot
2. pulmonary atresia
3. tricuspid atresia
4. Ebstein's anomaly

NB: less commonly this results from reversal of a left-right shunt,
2° to progressive PVD → ***Eisenmenger's syndrome***

• severe ***cyanosis*** stimulates red cell production, with ***polycythaemia***
• this may result in elevation of the Hct ≤ 80%
• up to ~ 60% this ***increases*** DO_2, however, increases in ***viscosity*** above this level result in decreased organ perfusion
• this also results in the reduction of ***fibrinogen & platelets***
• despite this, dehydration may lead to systemic and pulmonary venous ***thrombosis***
• ***clubbing*** of the fingers and toes develops due to proliferation of capillaries and small arteriovenous fistulae ? mechanism → PDGF
• ***hypoxic spells*** are due to acute cerebral hypoxia, 2° to decreased pulmonary blood flow
• ***spasm*** of the infundibular region is the most likely cause
• factors which lead to alterations of SVR/PVR are likely to precipitate spells, including,

1. physical exercise → ↓ SVR
2. hypoxia, hypercarbia, acidosis
3. hyperthermia, sepsis
4. drugs - vasodilators

• the reduction in pulmonary blood flow stimulates enlargement of bronchial and mediastinal arteries, which may provide the majority of blood flow
• at birth, the ***patent ductus*** provides a large contribution to PBF
• administration of $\boldsymbol{PGE_1}$, may prolong patency for up to days in some infants, allowing correction of the metabolic derangements prior to operation
• there are a number of anastomotic procedures to increase PBF,

a. Blalok-Taussig = subclavian to ipsilateral PA (end to side anastomosis)
   * now often done with a ***vascular patch*** to preserve the artery
b. Waterson = ascending aorta to right PA
c. Potts = descending aorta to left PA (*P*otts → *P*osterior)

• injection of the wall of the ductus with formalin 10% can delay closure for up to months in some infants

Tetralogy of Fallot

Def'n: ***pulmonary stenosis*** - with outflow obstruction
VSD - large, ***non-restrictive*** with R→L shunt
dextroposition of the aorta - over-ridding the septum
right ventricular hypertrophy ± failure

→ 10% of CHD and the commonest form of cyanotic CHD

plus atrial septal defect = ***pentalogy*** of Fallot

- ***Clinical Features***

a. symptoms
- syncope ~ 20%
- dyspnoea, exercise intolerance
- growth retardation

b. signs
- cyanosis, finger clubbing
- grade 1-3/6 PS bruit
* ***no VSD murmur***
- prominent RV impulse, single S_2
- murmur often absent during spell

c. ECG
- RAH, LVH
? RVH

d. CXR
- large aorta, small heart "boot shaped"
- small PA's, oligaemic lungs

e. complications
- cerebral abscess (~ 10%)
- other systemic emboli
- endocarditis
- thrombotic stroke (polycythaemia)
- epilepsy
- growth retardation
- increased risk/severity of "tet" spells if uncorrected

- ***Treatment***

• treatment varies with ***age*** and the ***severity*** of disease,

a. neonate
- maintain oxygenation
- maintain PDA, high SVR until shunt

b. severe infant - Blalok-Taussig shunt

c. child without shunt but increasing "spells" * β-blockers

NB: increasing trend toward ***primary repair***

• ***cyanotic spells*** are associated with self-perpetuating,

1. cyanosis
2. R→L shunt
3. hypoxic pulmonary vasoconstriction
4. subvalvular obstruction & ***spasm***
5. RV ischaemia ± failure

• mild to moderate attack,

1. 100% O_2
2. knee-chest position → ↑ SVR & reverse shunt
3. morphine 0.1 mg/kg → ↓ sympathetic drive

• severe attack,

1. 100% O_2
2. morphine 0.1 mg/kg - ↓ sympathetic drive
3. IPPV - ↑ P_{aO2} / DO_2
   - ↓ VO_2
4. paralysis - ↓ VO_2
5. hypocapnia - pulmonary vasodilator
6. maintain RV perfusion pressure
7. peripheral vasopressors - metaraminol
   - ↑ SVR
   * avoid β-agonists
8. pulmonary vasodilators - ***PGI_2*** ~ 0.1-0.2 µg/kg/min
   but, - also a systemic vasodilator
   - closes PDA (cf. PGE_1 maintains PDA)
   - fever
   - decreased platelet adhesiveness
   ? ***nitric oxide***

• b-***agonists*** may increase infundibular dynamic obstruction, reduce RV coronary perfusion and increase cardiac VO_2 (tachycardia)
• ***propranolol*** may therefore be used for prophylaxis
• providing the pulmonary vessels are of a reasonable size a corrective procedure is attempted
• the pulmonary outflow and annulus are frequently small, requiring insertion of a patch
• post surgery, greater volume work is required as PA flow is now normal, or often there is some incompetence of the valve
• therefore, these patients frequently have elevated heart rates and mild degrees of ***RV hypertrophy/failure*** postoperatively (↑ RBBB, sudden death)
• the overall success rate for surgical correction ~ 90-95%
• ~ 50% of these have near normal exercise tolerance

Transposition of The Great Vessels

- major diagnostic criteria,
    a. situs solitus, ***levocardia***
    b. ***cyanosis*** from birth ± hypoxic spells
    c. frequently in ***heart failure***
    d. cardiac enlargement and small PA segment on CXR *****narrow vascular pedicle***
    e. the presence of some pulmonary/systemic ***shunt***,
        → VSD (~ 30%), ASD, or PDA

- the lesion is more common in ***males***
- the aorta arises from the normally situated RV, and gives rise to the ***coronary vessels***
- the atria and ventricles are ***concordant***
- the systemic and pulmonary circulations are functionally separated, therefore, some abnormal shunt is required for existence
- patients with an intact ventricular septum and absent patent ductus have the worse clinical picture, as mixing occurs only at the atrial level
- however, these are the best candidates for surgery
- patients with large VSD's may die from excessive PBF and CCF from progressive PVD
- management includes,
    a. maintain high PVR
        - maintain RAP ~ LAP so that adequate ***mixing*** occurs, cf. one-way shunt flow
        - if LAP decreases (venous return / pulmonary afterload), then flow from RA → LA increases, with increased PBF and 2° LVF
    b. septostomy - ASAP
    c. vascular switch - 2 to 3 months

- ***corrected transposition*** is a rare anomaly where systemic venous blood reaches the lungs despite the presence of transposition
- commonly associated defects,
    1. pulmonary stenosis - ie. systemic inlet obstruction
    2. VSD

Cardiac Malposition

• ***situs inversus totalis*** is a rare anomaly where the stomach and other abdominal organs also occupy the mirror image of normal position
• except in asplenia, or polysplenia, the position of the abdominal organs determines the position of the atria
• thus, in ***situs inversus***, the atria are reversed and the heart is right sided
• the morphologic left ventricle is on the right and the atria and ventricles are ***concordant***
• severe anomalies may occur with situs inversus, dextrocardia and transposition of the great vessels,

a. the atria and ventricles are discordant
b. transposition of the great vessels is always present

• isolated ***levocardia*** is the remaining anomaly which may accompany situs inversus
• the heart is located in the left chest, there are severe cardiac anomalies and agenesis of the left lung
• in isolated ***dextrocardia*** the heart is in the right chest, the abdominal organs normal and there is agenesis of the right lung

• ***asplenia***, midline position of the stomach & liver (***situs intermedius***), distinct middle lobes of both lungs and Howell-Jolly bodies within RBC's are associated with severe cardiac anomalies

Miscellaneous Congenital Heart Lesions

a. congenital heart block
   - may be an isolated lesion, or with certain anomalies
   - especially - corrected transposition
     - 1º ASD or endocardial fibroelastosis

b. congenital mitral insufficiency

c. anomalous left coronary artery

d. pulmonary arterio-venous fistula
   ~ 50% have Rendu-Osler-Weber syndrome (multiple telangectasia)

e. pulmonary artery stenosis

f. persistent left SVC (connects LIJ & SC to ***coronary sinus***)

g. ***endocardial fibroelastosis***
   - ~ 1-2% of patients with CHD but may be sole anomaly
   - involves predominantly the left side
   - ? secondary to subendocardial ischaemia *in utero*
   - almost all die within the first year 2° to CCF

CHD - GA Considerations

1. ***prophylaxis for endocarditis***
   * all patient, ? except ligated PDA & secundum ASD without patch
2. ***air filters*** and meticulous removal of air from IV lines
   * all patients with intracardiac shunts, irrespective of the direction of the shunt
3. ***minimise myocardial VO_2***
   i. adequate premedication & a (? rapid) smooth induction
   ii. adequate analgesia
   iii. avoid hypertension / tachycardia
   iv. maintain normocarbia
   v. maintain NMJ paralysis
   vi. LV or RV afterload reduction
4. ***optimise cardiac output***
   i. avoid depressant agents
   ii. maintain filling pressures - minimise preoperative dehydration
   iii. avoid / manage arrhythmias
   iv. avoid hypocarbia
      - reduces CO, increases SVR
      - shifts HbO_2 curve left
      - decreases myocardial & cerebral blood flow
      - decreases K^+
      - increases arrhythmias
5. ***avoid alteration of shunt flow***
   i. avoid agents which alter SVR or PVR
   ii. be aware of the possible effects of IPPV/PEEP
   iii. factors which alter dynamic outflow obstruction
      - positive inotropes, sympathetic stimulation
   iv. avoid hypotension if dependent on systemic-pulmonary shunt flow for oxygenation
6. ***heparin*** has a larger volume of distribution and a more rapid plasma clearance in infants - larger loading doses and monitoring are often required
7. ***myocardial protection***, during CPB,
   i. cardioplegic solutions
      - different opinions
      - high K^+, Mg^{++}
      - high dextrose
   ii. hypothermia - repeated PRN
   iii. pre-CPB steroids ? controversial
   iv. optimal reperfusate solution
      - cool & alkaline
      - low ionised Ca^{++}
      - slightly high K^+

Post-operative Management Cardiac Surgery

- postoperative ***respiratory function*** is altered by,
  - a. anaesthesia
    - hypoventilation, atelectasis
    - reduced clearance of secretions
  - b. surgical incision
    - midline sternotomy or thoracotomy
    - poor cough and reduced FRC
  - c. effects of CP bypass
    - capillary leak and pulmonary oedema
    - damaged pulmonary capillary endothelium
      ? from endotoxin release
    - mechanical red cell damage
    - C' activation from exposure to oxygenator membrane
    - the effects of CPB on C', platelets etc. are ***greater*** than adults, due to the relatively greater SA of the circuit cf. body endothelial SA
  - d. ↑ LAP
    - left ventricular failure
    - mitral incompetence or stenosis
    - residual VSD
  - e. phrenic nerve palsy
  - f. pneumothorax

- postoperative ***cardiovascular function*** is altered by,
  - a. direct damage to myocardium from ventriculotomy
  - b. ischaemic damage because of hypoxia
  - c. effects of cardiopulmonary bypass
  - d. excision of hypertrophic muscle
  - e. changes to flow/load patterns, especially from central shunts, where repeat surgery may be necessary
  - f. hypovolaemia from insufficient venous filling from the bypass pump or haemorrhage
  - g. increased PVR
    - operative L→R shunts acutely increasing PBF
    - high PBF preoperatively
  - h. cardiac tamponade
    - bleeding
    - pericardial effusion
    - tension pneumothorax
  - i. HR abnormalities
    - surgical damage to conductive tissue
    - SA node with intra-atrial repairs
      (atrial baffles, patch closure ASD, repair A-V canal)
    - interruption of atrial pathways
    - distortion from atrial dilatation

• non-surgical postoperative ***bleeding*** results from,

a. consumption of platelets and clotting factors - bypass circuit
- intracardiac patches

b. residual heparinisation

c. citrate toxicity from large blood transfusion

d. preoperative hepatic insufficiency (2° to congestion)

• ***renal failure*** following cardiac surgery is caused by low cardiac output, and reduced renal perfusion while on bypass

Persistent Foetal Circulation

a. low lung volume states - hyaline membrane disease
- perinatal asphyxia

b. pulmonary hypoplasia
- diaphragmatic hernia
- Potter's syndrome - renal agenesis
→ - lack of amniotic fluid
- secondary failure of pulmonary development

c. meconium aspiration syndrome

d. chronic placental insufficiency

e. hypoxia or acidosis - any cause

f. sepsis - any cause

g. hyperviscosity syndrome

h. any increase in PVR → cyclic effect → ↓ P_{aO2} & pH

■ ***Clinical Features***

a. hypoxaemia >> the degree of respiratory distress

b. cyanosis - suggesting CHD
- may be differential with PDA

c. acidosis ± hypercarbia

- ***Management***

a. maintain a ***high*** $\mathbf{F_IO_2}$

b. correct low lung volume with **CPAP**

c. correct metabolic and respiratory acidosis

d. NMJ blockade + IPPV + deliberate ***hyperventilation***

→ generate a ***respiratory alkalosis*** (pulmonary vasodilation)

e. maintain systemic volume & pressure = plasma volume expanders ± inotropes

→ reduce the pressure gradient for shunting

f. isovolaemic haemodilution if hyperviscosity present

g. pulmonary vasodilators

i. inhaled nitric oxide

ii. others
- isoprenaline
- tolazoline
- SNP, GTN
- phenoxybenzamine
- PGE_1

→ variable response depending on underlying pathology

h. surfactant therapy

i. animal (bovine) surfactant

ii. recombinant human

CARDIAC ARREST IN CHILDREN

- the majority lack intrinsic cardiac disease, arrest being the end result of ***hypoxaemia & acidosis***
  → biochemistry is grossly abnormal ***prior*** to arrest

- ~ 70% or more of paediatric arrests occur **< 1 yr** of age

- ***Most Common Causes***

1. rapidly progressive ***upper airway obstruction***
2. SIDS
3. severe systemic illness
   i. pneumonia
   ii. gastroenteritis
   iii. septicaemia
4. major trauma / accidents
   i. MVA's
   ii. fire/smoke inhalation
   iii. near-drowning
   iv. NAI / abuse
5. congenital disorders
   i. heart disease
   ii. respiratory disease

- children invariably arrest in ***asystole*** (96% in one series) and this should be suspected if an ECG is unavailable
- ***ventricular fibrillation*** may be anticipated in the following situations,

1. congenital heart disease
2. cardiomyopathies / myocarditis
3. drug poisoning - TCA's
4. hereditary long QT - Romano-Ward syndrome
   - Jervelle-Lange-Neilsen

- EMD may occur from ***hypovolaemia*** but is rare from other causes
- presence of a pulse is best determined at the ***carotid***

Management

a. ***airway***

i. obstruction is more likely

ii. gastric distension is almost invariable → early ***ETT & NG*** tubes

b. ***cardiac massage***

- relative organomegaly etc. in the infant → used to advocate mid-sternal massage
- risks of trauma ***unfounded*** & lower sternal massage → more effective
- conventional CPR is more effective than simultaneous compression / ventilation

i.	< 1 year	2 fingers	100^{+}	bpm	1-2.5 cm
ii.	1-8 years	1 hand	80-100	bpm	~ 2.5 cm
iii.	adult	2 hands	80	bpm	~ 5.0 cm

c. ***drug access***- best by CVC lines, proximity to heart
- technically difficult, interferes with CPR
- percutaneous cut-down ± ***intraosseous needle***

d. ***asystole***

- SR can often be restored ≤ 45-60 min but high incidence of ***hypoxic brain damage***
- CPR alone is often successful
- in absence of AGA's → $NaHCO_3$ ~ 2 ml/kg stat
- ***adrenaline*** 1:10,000 → ~ 0.1 ml/kg stat (0.01 mg/kg) & repeat 3 minutely ≤ 2 ml/kg if required
- VF is uncommon & tachycardia well tolerated
- Ca^{++} should only be used for hyperkalaemia, hypocalcaemia & CEB toxicity due to role of Ca^{++} in ***reperfusion injury***

e. ***intracardiac injection***

- endotracheal administration of adrenaline, but ? effectiveness (use ~ 5x dose)
- HCO_3^- cannot be given via ETT
- thus, intracardiac injection may be justified in children
- either left ant. 4^{th} ICS or sub-xiphisternal (beware the liver)
- potential complications include,

i. intramyocardial injection & VF

ii. coronary vessel laceration

iii. pericardial tamponade

iv. pneumothorax - always with parasternal injection

v. interruption of CPR

f. ***ventricular fibrillation***

- spontaneous reversion may occur with CPR
  ~ 3-5 J/kg DC shock + repeat x1
  ± lignocaine 1 mg/kg IV ± 0.5 mg/kg
- adrenaline to improve coronary perfusion
- phenytoin 15 mg/kg if TCA overdosage & early HCO_3^-

Outcome

• important complications of paediatric cardiac arrest are,

1. brain failure
2. disseminated intravascular coagulation
3. splanchnic ischaemia mucosal sloughing

NB: in one study, patients who were resuscitated from absence of pulse or electrical activity showed ***no*** neurologically intact survivors

• neurologically intact survival is only seen in those paediatric patients who receive immediate resuscitation and respond promptly
• results are poor where cardiac arrest occurs in hospital wards or in paediatric and neonatal ICU's

→ ~ 9% long term survival

• outcome from near-drowning episodes may be good ***if*** the patient receives effective resuscitation at the scene and is gasping soon after
• where cardiac arrest occurs in the community, physician-staffed mobile intensive care units ***do not*** improve outcome

Arrhythmias in Children

- ***Causes***

a. hypoxia, hypercarbia, acidosis
b. electrolyte disturbance
c. hypotension
d. hypothermia
e. excessive vagal stimulation
f. cardiomyopathies, myocarditis
g. long QT syndrome
h. congenital
   - aberrant pathways
   - complex CHD
i. surgery
   - transplantation
   - cardiothoracic surgery
   - cardiac catheterization
j. drugs
   - TCA's
   - digoxin
   - organophosphates
   - suxamethonium
k. malignant hyperthermia

- ***Clinical Features***

a. ***sinus bradycardia*** - hypoxia, hypotension, acidosis
- raised ICP
- vagal stimulation, SCh
- post cardiac surgery (Mustard)

b. ***bradycardia-tachycardia*** - cardiomyopathy
- post cardiac surgery
(Mustard, Fontan & Senning operations)

c. ***A-V block*** - congenital
- cardiomyopathy
- post cardiac surgery
- myocarditis
- vascular disorders

d. ***SVT*** - WPW syndrome
- post cardiac surgery
- myocarditis, sepsis
- drugs, idiopathic causes

R_X infant
- DC shock, overdrive pacing
- neostigmine ~ 10 µg/kg (≤ 50 µg, atropine readily available)
- digoxin ~ 15 µg/kg
- amiodarone ~ 5 mg/kg/1 hr, then 5-15µg/kg/min

* avoid Ca^{++}-entry blockers

R_X child
- vagal stimulation
- neostigmine
- verapamil 0.1 mg/kg IV
- DC shock, overdrive pacing
- digoxin, amiodarone

• ***adenosine*** ? no controlled trials in children, but ? similar efficacy to adults
~ 0.0375-0.25 mg/kg

e. ***VEB's / VT*** - aortic stenosis
- other CHD
- myocarditis
- digitalis toxicity
- long QT syndrome
- TCA overdosage

R_X acute
- lignocaine ~ 1.0 mg/kg IV, then 20-50 µg/kg/min
- Mg^{++} ~ 0.05 mmol/kg/10 mins, then 0.2 mmol/kg/6 hrs
- bretylium ~ 5.0 mg/kg IV, then 5-15 µg/kg/min

* vasopressors for AS

R_X maintenance
- quinidine ~ 6.0 mg/kg q6h
- phenytoin ~ 4.0 mg/kg q8h

f. ***long QT syndromes***

i. pause dependent
- drugs: TCA's, phenothiazines
- metabolic: ↓ Mg^{++} | ↓ Ca^{++}
↓ K^{+} = "apparent long QT"

R_X
- correct cause
- DC shock & overdrive @ 120 bpm
± isoprenaline infusion

ii. adrenergic
- hereditary
- following SAH

R_X
- β-blockade
± phenytoin

g. ***TCA overdosage***
- multifocal VEB's
- VT / VF, torsade de pointes
- SVT
- CHB

R_X
- hyperventilate
- alkalinise blood to pH ~ 7.45-7.5
- $NaHCO_3$ ~ 1-3 mmol/kg
- phenytoin ~ 15 mg/kg slow IV
± lignocaine, Mg^{++}, or bretylium

Invasive Monitoring in Children

• excessive flushing of arterial lines may cause retrograde flow into cerebral vessels (especially temporal artery lines)
• ***normal saline*** is used as the fluid column to allow accurate glucose measurement from sampled blood
• central venous lines and pulmonary artery catheters are inserted as for adults
• cardiac output is described in terms of ***cardiac index*** (N ~ 3-3.5 l/min/m^2) to account for changes with weight and size, and is measured by,

a. thermodilution via PA catheter
- use limited in small patients
- not accurate where intracardiac ***shunts*** are present (systemic and pulmonary blood flows not equal)
- volume load in small patients

b. dye dilution
- CVC injection of dye and peripheral artery sampling
- not easily performed but demonstrates intracardiac shunts

• ***pulse oximetry*** monitoring is routine, and suitable probes are available for all age groups
• with end-tidal CO_2, in line sampling may be superior to side arm sampling techniques, especially with small tidal volumes at rapid rates, however, added dead space may be significant
• core-peripheral temperature gradients ***do not*** accurately trend changes in cardiac output

Circulatory Failure in Children

- the causes differ from the adult due to,

a. smaller ***fluid compartments*** → % changes are greater

b. immature ***immune system*** ≤ 2 years of age

i. ↓ IgG, C', opsonins (fibronectin)
→ susceptibility to ***bacterial*** infection

ii. ↓ interferon, lymphocyte cytotoxicity
→ susceptibility to ***viral*** infection

c. ***heart rate*** dependent CO
- little alteration of SV
- greater Ca^{++} dependency,

i. fewer sarcomeres/myofilaments per unit mass

ii. fewer mitochondria/myosin ATP'ase →
- higher diastolic volume, limited diastolic reserve
- less responsive to increases in preload
- augmentation of contraction is limited
- afterload induced increases in contraction are small
- VO_2 and CI are high with limited systolic reserve

→ less compliant ventricle & easily volume overloaded

d. ***autonomic immaturity***
- SNS << PNS innervation
- basal PNS tone is low
- insensitivity to β-agonists
- low myocardial NA stores

∴ ***stress response*** → bradycardia & less vasoconstriction

e. ischaemic tolerance
- greater than the adult
- ***cerebral plasticity***
- cardiac glycogen stores

f. factors peculiar to infancy
- SIDS
- haemorrhagic shock & encephalopathy syndrome

g. ***congenital abnormalities*** - cardiac, metabolic

h. dependency / inexperience

Causes of Shock in Childhood

- ***Hypovolaemic***

1. bleeding - bowel, body cavity, haematoma, external
   * scalp, intracranial
2. fluid/electrolyte loss
   - i. bowel - V&D, obstruction, 3rd spacing
   - ii. renal - diuretic use
     - diabetes insipidus
   - iii. skin - burns, heat stroke
3. plasma loss - sepsis, burns
   - pancreatitis
   - nephrotic syndrome

- ***Distributive***

1. septic
2. anaphylactic
3. drug induced - barbiturates, phenothiazines
4. neurogenic - brainstem, high Cx spine
5. ↑ intrathoracic press. - IPPV, CPAP, PEEP
   - tension pneumothorax
   - pericardial effusion/tamponade

- ***Cardiogenic***

1. congenital heart disease
2. hypoxia/ischaemia - global, near drowning
   - Kawasaki disease, anomalous LCA
3. cardiomyopathy - metabolic, glycogen storage diseases
   - muscular dystrophies
   - endocardial fibroelastosis
   - infective, Echo & Coxsackie
4. drug intoxication - barbiturate, chloramphenacol
5. loss of atrioventricular coordination
6. rate induced - bradycardia / tachycardia
7. sepsis

- ***Mixed***

  - eg. septis, drug, pancreatitis

Clinical Signs of Shock in Children	
Hypovolaemic	• signs of dehydration if severe H_2O loss • tachycardia, hypotension, narrow pulse • pallor, mottled & cyanosed skin • slow capillary refill • cool extremities • tachypnoea early, later hypoventilation • lethargy ± coma • oliguria
Cardiogenic	• tachycardia, hypotension, narrow pulse • pallor, mottled & cyanosed skin • cardiomegaly, hepatomegaly • faint heart sounds, gallop rhythm • pulmonary crepitations
Septic	• tachycardia, hypotension, oliguria • early: warm extremities, bounding pulse, lethargy • later: cool, cyanosed extremities narrow pulse, tachypnoea, coma
Other distributive	• tachycardia, hypotension, oliguria • bounding pulse, warm pink extremities • lethargy, stupor, coma

• septic neonates and infants ≤ 6 months generally present with a ***hypodynamic*** rather than hyperdynamic circulatory picture
• in hypovolaemia, BP is maintained until **~ 15-20%** of blood volume is lost
• subsequent signs of ***cellular injury*** include,

a. metabolic acidosis & hyponatraemia ∝ decrease Na^+/K^+-ATPase
b. increased catechols, tachycardia, glucose intolerance
c. falling platelet count & fibrinogen, increased clotting time
d. late: coagulopathy, bloody diarrhoea, fitting & coma

Age Related	BP (mmHg)	HR (bpm)	RR
birth	75 / 40	100-200	40-60
1-2 years	95 / 60	100-180	20-30
6 years	98 / 60	70-120	15-20
10 years	110 / 70		
14 years	118 / 75		

- ***Investigation***

a.	biochemistry	- U&E's, LFT's, BSL - AGA's
b.	haematology	- FBE, differential WCC, platelets - coagulation screen - group & hold serum
c.	microbiology	- blood cultures x 3 - M,C&S: sputum, pus, CSF, urine - viral studies: urine, stool, nasal - urinary bacterial Ag's
d.	imaging	- CXR ± AXR - ECG ± echocardiography
e.	drug screen	- urine, blood, gastric aspirate
f.	***metabolic screen***	- urinary amino acids/organic acids - serum ammonia

- ***Monitoring***

a.	HR, BP	- NIBP/intra-arterial, RR
b.	urine output	
c.	AGA's & pulse oximetry	
d.	CVP ± PAWP	- $\delta P/\delta V$ (compliance) better guide than absolute values - normal values ~ adults
e.	cardiac output	- signs/clinical examination - doppler - bioimpaedance - dye/thermodilution
f.	derived data (PA)	- PVR/SVR $\equiv^{t}$ afterload - CI, DO_2, VO_2
g.	core-toe temperature gradient	* ***does not*** correlate with CI
h.	clinical examination	- GCS

- ***Management - Priorities***

a.	brain and heart perfusion	~ 80% "normal" BP
b.	gas exchange	± IPPV
c.	renal & GIT perfusion	- adequate BP/CO ? low dose dopamine
d.	peripheral perfusion	

- ***Methods of Treatment***

a. optimise
   - i. preload — ~ 10 ml/kg colloid "challenges"
     - monitor as above
     * ***hypotension*** ~ 30 ml/kg deficit !
     * at 30 ml/kg consider rbc transfusion
   - ii. afterload — - short acting systemic agents, SNP
     ~ selective pulmonary agents (NO, PGE_1, GTN, tolazoline)
   - iii. contractility — - inotropic support
     - often need higher doses (per kg) than adults
     - myocardial NA stores easily depleted
     - receptor down-regulation
     ± try 10% Ca-gluconate (0.2-0.5 ml/hr)

b. correct metabolic acidosis with $NaHCO_3$

c. treat sepsis — - antibiotics, drainage

d. supportive measures
   - i. peptic ulcer prophylaxis — ?what
   - ii. platelets/FFP in coagulopathy
   - iii. steroids in Waterhouse-Friderichsen syndrome
   - iv. accurate fluid balance
   - v. thermal environment

e. controversial R_X
   - i. high dose steroids of ***no benefit*** in large trials
   - ii. plasma exchange
     - - positive animal work
     - ? anecdotal human reports
   - iii. granulocyte transfusion/exchange
     - positive case reports — - esp. newborns
   - iv. immunotherapy
     - E.coli J5 immune serum
     - anti-lipopolysacharrhide serum
     - phase III trials → ***no benefit***
   - v. acute phase reactant inhibitors: ***anti*** - phospholipase A_2
     - lipogenase
     - leukotrienes
     - PAF
   - vi. continuous ***haemofiltration*** ? middle molecule removal
   - vii. balloon counterpulsation — - effective but technically difficult

Heart Failure in Children

a.	congenital heart disease		
	i.	presenting at birth	- obstructive lesions - systemic AVM
	ii.	presenting 1st 4 months	- large left or right shunts
b.	post-cardiac surgery		
c.	asphyxia		- perinatal - near drowning - upper airway obstruction
d.	metabolic		
e.	arrhythmia		
f.	cardiomyopathy		- infective - infiltrative - metabolic
g.	endocarditis		
h.	rheumatic heart disease		
i.	severe anaemia		- eg. hydrops foetalis
j.	acute hypertension		- acute GN
k.	cor pulmonale		- cystic fibrosis - pulmonary vascular disease 1°/2°

Sepsis - Common Organisms

a.	neonates	- group B, beta haemolytic streptococci - Enterobacteriaciae - *Listeria monocytogenes* - *Staphylococcus aureus*
b.	infants/children	- *H. influenzae* - *Strep. pneumoniae* - *N. meningitidis* - *Staph. aureus* - Enterobacteriaciae
c.	immunocompromised	- Enterobacteriaciae - *Staph. aureus* - pseudomonas species - *Candida albicans*

Haemorrhagic Shock & Encephalopathy

• syndrome described in infants and children,

a. high mortality

b. shock, hyperthermia, watery diarrhoea, coagulopathy

c. impaired renal and hepatic function

d. cause has yet to be determined

Evaluation of the Cyanotic Neonate & Infant

• difficult to differentiate between pulmonary and cardiac causes of respiratory distress and cyanosis in neonates and infants because,

a. typical cardiac findings may be absent or obscured

b. central cyanosis, crackles and wheezes are caused by both intracardiac or intrapulmonary right to left shunting

c. noisy breathing interferes with auscultation

d. murmurs may not initially be present during transitional foetal/newborn circulation

• other causes of cyanosis are,

a.	2° to hypoventilation / apnoea	- prematurity - hypothermia - hypocalcaemia - hypoglycaemia - sepsis
b.	circulatory shock	- sepsis - obstructive cardiac lesions - hypoplastic left heart
c.	persistent foetal circulation	- elevated PVR

• ***cyanosis*** is clinically evident when $SpO_2 \leq 88\%$
• in neonates this corresponds to a $P_{aO2} \sim 30\text{-}85$ mmHg
• depending on foetal haemoglobin, pH, temperature and 2,3-DPG
• pulse oximetry is ***not reliable*** in this range of saturation

Intracardiac vs. Extracardiac Causes of Cyanosis

- blood gas with ***intracardiac shunts***,
  a. no significant improvement in P_{aO2} with increase in F_IO_2
  b. P_{aO2} < 160 mmHg with F_IO_2 = 1.0 (N: ~ 20-50 mmHg)
  c. no improvement in P_{aO2} with positive airway pressure
  d. P_{aCO2} is usually ***normal***

- note that P_{aO2} may also not rise when F_IO_2 is increased with ***intrapulmonary shunting***, when,
  a. the pulmonary lesion is severe, or
  b. where shunting occurs through foetal pathways
     i. patent ductus and foramen ovale
     ii. raised pulmonary vascular resistance

- CXR may help exclude non-cardiac causes but differentiation may be difficult,
  a. an ***enlarged*** heart equals cardiac disease
     - however, heart size may be normal with some cardiac conditions
  b. heart shape shows chamber enlargement and abnormally placed vessels
  c. lung fields show increased, reduced or normal pulmonary blood flow & vasculature
  d. classical appearances,
     i. transposition - cardiomegaly
        - increased vascular markings
        - narrow vascular pedicle
     ii. Fallot's - normal heart size
        - reduced pulmonary vascular markings
        - "boot-shaped" heart

- ECG may show increase in size of cardiac chambers (note that normal newborn ECG has right ventricular dominance) and arrhythmias
- other investigations for cyanosis include,
  a. FBE - Hb, *chronic cyanosis $\rightarrow$ polycythaemia
     - white cell count
  b. biochemistry - K^+, Na^+, HCO_3^-, Ca^{++}, glucose
     - ABG's
  c. temperature
  d. microbiology - MC&S: blood, urine, tracheal aspirate
     - CSF if no coagulopathy
  e. echocardiogram - in the presence of CHD
     ± cardiac catheter

Hypertension

1. elevated ***diastolic*** blood pressure,
   i. ≥ 90 mmHg < 6 years age N: 95 / 60
   ii. ≥ 95 mmHg ~ 6-12 years age N: 100 / 60
   iii. ≥ 100 mmHg > 12 years age N: 110 / 70
2. ECG or echocardiogram evidence of ***ventricular hypertrophy***
3. hypertensive ***encephalopathy***
   i. headaches, dizziness
   ii. seizures
   iii. hypertensive retinopathy / papilloedema

- causes in the paediatric age group are,
  a. essential hypertension
  b. renal disease
     - PSGN
     - GN - other causes
     - HUS
     - nephrotic syndrome
  c. coarctation of the aorta
  d. adrenal disease
     - phaeochromocytoma
     - Cushing's
     - Conn's

- Barrter's syndrome are usually normotensive

RESPIRATORY DISORDERS

▪ ***Respiratory Mechanics***

• a number of factors make respiration less efficient in the ***neonate***,

a. large V/Q mismatch
   i. large ***shunt fraction*** **~ 10%**
   ii. similar dead space but ~ **2-3x** VO_2 of adults
   iii. small FRC
      • ↑ VO_2 :: FRC ratio → rapid desaturation
      • ↓ FRC :: CC ratio → gas trapping & ↑ V/Q mismatch
      • loss of laryngeal brake with ETT & further ↓ FRC

b. small airway diameter $R_{AW} \propto 1/r^4$
   • compliant airways & increased narrowing 2° venturi (Bernoulli) effect
   • most resistance in the upper respiratory tract ~ 25% in the nasal passages,
     cf. ~ 60% in the adult

c. highly compliant/flexible airways & chest wall
   i. functional airway closure
   ii. inability to sustain a high negative P_{IP}
   iii. high compliance of chest wall / horizontal ribs
   iv. abdominal organomegaly/stomach

d. ↓ ***type I muscle fibre*** (oxidative phosphorylation) → less resistant to fatigue
   i. neonate ~ 25% diaphragm / 45% intercostal
   ii. adult ~ 60% in both
   • but, fast type II fibres are better suited to the neonates rapid respiratory rates
   • however, these are more prone to fatigue under conditions of increased load

• in the premature infant the basal ***work of breathing*** **~ 3x** that of adults without disease
• the pulmonary circulation at birth is characterised by the ***muscularity*** of the pulmonary arteries
• the response to hypoxia/stress is ***vasoconstriction*** and this may worsen the situation
• ***work*** of breathing is given by the volume of gas moved against respiratory compliance, and the work to overcome resistance to airflow,

$$W = V/C_{RS} + R_{AW}.\dot{Q}$$

• lungs of neonates with HMD or bronchitis may markedly differ from the above,

a. deficient ***surfactant***
b. ↑ ventilation/perfusion mismatch
c. ↓↓ compliance ~ 0.00025-0.001 l/cmH_2O ↓ **5-20x**
d. ↑↑ resistance ~ 100-250 cmH_2O/l/s ↑ **5-10x**
e. ↑ work of breathing
f. ↑ propensity to pneumothorax / barotrauma

■ ***Respiratory Control Centres***

• during infancy, central responsiveness to,

a. ↑ stimulatory inputs - hypoxia | hypercarbia | acidosis

b. ↓ inhibitory inputs - chest wall deformation | laryngeal stimulation

NB: → newborns have a ***biphasic*** response to ***hypoxia***
initial ~ 30% ↑ V_M, then ~ 30% ↓ V_M below baseline ± ***apnoea***

• response depends upon the thermal environment
→ ***hypothermic*** neonates responds only with respiratory ***depression***
• the ventilatory response to hypoxia becomes "adult-like" at ~ **3 *weeks***
• the ventilatory response to CO_2 increases with gestational & postnatal age
• this response is ~ 3x greater in 2-3 day term infants cf. 2-3 day prem's
• by ~ 1 month the response of a term infant is ~ adult

→ thus, both ***hypoxic & hypercapnic*** drives → adult at ~ ***1 month***

• in young infants, the increased ***apnoeic***,

a. ***incidence*** ∝ ↑ sensitivity to inhibitory inputs that trigger apnoea

b. ***duration*** ∝ ↓ central responsiveness to stimulatory afferents, which promote recovery from apnoea

■ ***Anaesthetic Considerations - Respiratory***

• Laylock (1988) found the incidence of ***hypoxaemia*** (SpO_2 < 80%) during induction to be,

a. infants ≤ 1 year ~ 28%

b. children 2-5 yrs ~ 2%

c. children 4-10 yrs ~ 4%

NB: the most commonly associated factor was a ***delay in intubation***

• recommendations for neonate/young infant,

1. set time sampling interval on oximeter to 2-3 cycles
2. intubate all infants ≤ 1 year unless procedure is very brief
3. pre-O_2 for 2-3 minutes prior to laryngoscopy
4. use the pulse oximeter to limit the duration of laryngoscopy
5. ?? assist ventilation during induction/emergence
6. control ventilation during maintenance (preserves FRC)

■ ***Neonatal Intubation***

• differences which make the neonate more difficult to intubate,

1. poor tone of the neck muscles and the large ***head*** → "floppy"
2. large size of ***tongue*** cf. oropharynx
3. the ***larynx*** is located higher in the neck C_{3-4} vs C_{4-5}
4. "V-shaped", short, stubby, highly mobile ***epiglottis***
   - adult is parallel to trachea cf. infant angled over
5. ***vocal cords*** are angled infero-anteriorly
   - blind ETT passage may lodge in the anterior commissure, rather than the trachea
6. the larynx is funnel shaped, being narrowest at the ***cricoid***
   - tubes easily passing the cords may result in subglottic oedema
     → use ***uncuffed tubes*** for ages < 10 years
7. the ***trachea*** only 4 cm long
   - ∴ ETT easily dislodged, or positioned in RMB, especially with head movement

■ ***Mechanical Ventilation***

• most neonates breathe at 30-60 bpm, I:E ratio of ~ 1:1, 5x the mean ***time constant*** being ~ 0.6s
• as the respiratory rate increases there is the potential for ***gas trapping***
• this may be beneficial at low lung volumes but detrimental in the face of increased airways resistance or high lung volumes

• majority of neonatal ventilation is with pressure-limited, time cycled ventilators
• these are used due to a reduced incidence of ***barotrauma*** and ***bronchopulmonary dysplasia***
• the major disadvantage is the lack of compensation for alterations in pulmonary mechanics, with subsequent changes in V_M

NB: ***oxygenation*** is predominantly determined by the mean airway pressure, ***normocapnia*** by alveolar ventilation

• Boros (1979) showed that the ratio PaO_2:F_IO_2 is proportional to the mean airway pressure
• however, at some point this becomes excessive and is detrimental (analogous to "best-PEEP")
• approximate guidelines are,

a. **PaO_2** **~ 50-70 mmHg**
b. SaO_2 ~ 87-93 % *this is oximeter dependent
c. $PaCO_2$ ~ 35-50 mmHg
d. pH ≥ 7.28
e. peak P_{AW} ≤ 30 cmH_2O

NB: by accepting these values the incidence of ***barotrauma*** is reduced

• PEEP increases mean P_{AW} and improves FRC at low lung volumes
• increasing PEEP without increasing the peak P_{AW} decreases the tidal volume & minute ventilation
• at rapid respiratory rates (> 60 bpm) significant gas trapping occurs
• time-cycled flow ventilators tend to more reliably deliver a constant tidal volume when the inspiratory time is ≤ 0.4 sec

• ***oxygen*** should only be administered to achieve a PaO_2 in the above range
• excessive administration is associated with an increased incidence of,

a. retrolental fibroplasia
b. bronchopulmonary dysplasia

• the aim should be to reduce the F_IO_2 to ≤ 0.6 ASAP
• there are few studies on the effects of gas flow rates
• the general aims of weaning should be to,

a. ↓ F_IO_2 ≤ 0.6 prior to other reductions
b. ↓ peak P_{insp} ≤ 20 cmH_2O
c. ↓ IMV rate
d. ↓ PEEP ≤ 5 cmH_2O

NB: most are extubatable at IMV ~ 5 bpm / PEEP ~ 3 cmH_2O

• if infants have periodic breathing or ***apnoeic spells***, weaning may be facilitated with ***theophylline***

• ***exogenous surfactant*** often has a dramatic effect upon neonatal respiratory function
• within 2-3 hours ventilation on room air with peak P_{AW} ≤ 20 cmH_2O is often seen
• changes may occur so rapidly that alteration of ventilatory parameters fails to keep pace with alterations in pulmonary mechanics
• this effect tends to be worse with bovine surfactant, as changes occur more rapidly than with synthetic surfactants
• despite this, these patients frequently require ventilation for several days
• early extubation is associated with a high incidence of re-intubation and deterioration of respiratory function
• occasionally 2-3 doses of surfactant are required

• other forms of ventilation, high frequency jet/oscillatory ventilation, ***have not*** been shown to be of any advantage in reducing,

a. the incidence of barotrauma or chronic respiratory disease

b. mortality

c. persistent PDA

• initial studies with these forms of ventilation were associated with,

a. a higher incidence of ***intraventricular haemorrhage***

b. higher requirements for ***vasopressors*** to maintain MAP

NB: 2° to interference with - cerebral autoregulation
- the baroreceptor reflex

HIFI study group, NEJM 1989 → widespread condemnation

• since then, improved knowledge of ***optimal lung volume*** strategies have resulted in ***improved outcomes*** in paediatric use of HFOV Review by Froese, Current Opinion in CC 1996
• aim is to institute ventilatory strategies maintaining open lung units, while preventing overdistension, ***early*** and thus preventing lung injury
• numerous neonatal/paediatric studies now support this view

• chronic lung disease, bronchopulmonary dysplasia, is managed with a combination of diuretics (frusemide) and steroids (dexamethasone)
• infants frequently relapse following response to steroids and multiple courses may be required

■ ***Postoperative Apnoea***

• postoperative ***apnoea*** occurs predominantly in former premature infants, and rarely in term infants ≤ 1 month of age
• in prem's the incidence is inversely proportional to the ***postconceptual age***
• incidence is very low ≥ 50-60 weeks postconception
• the ***apnoeic episodes*** usually commence within 2 hours of surgery and may be,

a. ***brief*** ~ 5-15 s

b. ***prolonged*** ≥ 15 s

• ~ 1/3 will have onset of apnoea at 4-6 hours, very rarely the onset may be at 8-12 hours
• the duration of apnoeic episodes also varies with postconceptual age,

a. ≤ 45 weeks - episodes may occur for up to 24-48 hours

b. > 45 weeks - episodes usually disappear within 12 hours

NB: most will admit ex-prem's < 60 weeks PCA for overnight monitoring

Upper Airway Obstruction		
	Neonate	Infant & Child
Nasal	• choanal atresia	
Oropharyngeal	• Pierre-Robin syndrome • Treacher-Collins • thyroglossal atresia • vallecular cyst	• macroglossia • retropharyngeal abscess • tonsillitis ± abscess • obstructive sleep apnoea
Laryngeal	• "infantile larynx" • vocal cord palsy • subglottic haemangioma • laryngeal cysts (cystic hygroma, teratoma) • laryngeal web • laryngomalacia • laryngeal spasm	• croup & spasmodic croup • epiglottitis • post-extubation oedema • teratoma / papilloma • haem/lymph-angioma • reflex (laryngospasm) • burns / smoke inhalation • caustic ingestion
Tracheal	• tracheomalacia • vascular ring • meconium aspiration • obstruction of ETT	• foreign body • tracheal stenosis • vascular ring • bacterial tracheitis • burns / smoke inhalation

- ***Progression of Obstruction***

1. ***Early***
   - i. stridor on exertion
   - ii. stridor at rest
   - iii. retraction on exertion → intercostal & suprasternal

2. ***Late*** = indications for ***intubation***
   - i. retraction at rest → tachycardia/tachypnoea
   - ii. exhaustion & tiredness
   - iii. cyanosis & bradycardia
   - iv. cardiorespiratory failure
   - v. cardiac arrest

- ***Upper Airway Obstruction Adult***

a.	foreign body / aspiration	
b.	infections	- adult epiglottitis - nectrotising fasciitis - Ludwig's angina - pharyngeal abscess, quinsy - infected epiglottic cyst
c.	neck / facial trauma	- gunshot wounds - burns - postoperative - acid/caustic ingestion - laryngeal fracture
d.	tumour	- tongue - larynx, trachea - thyroid - oesophagus - 2° nodes, mediastinal masses
e.	oedema	- angioneurotic oedema - pre-eclampsia - anaphylaxis
f.	neurological	- bulbar/pseudobulbar palsy - GBS, CIP - myasthenia - CNS depressants, drug overdose - CVA
g.	endocrine	- hypocalcaemia, acute hypoparathyroidism - goitre, myxoedema
h.	tracheal stenosis / tracheomalacia	
i.	post-surgical	- oedema - haemorrhage - throat packs - vocal cord palsy
j.	instrumentation	- ETT kinking - cuff overinflation - Minnesota tube - tracheostomy false passage

Respiratory Failure

- ***Predisposing Factors: Neonate***

a.	structural immaturity of the thorax	- high chest wall compliance - diaphragm fatigue - horizontal ribs - relative abdominal organomegaly
b.	immaturity of the respiratory system	- surfactant - alveolar instability - central drive
c.	airway size / resistance	
d.	high VO_2	
e.	high shunt fraction	
f.	relative immunoparetic state	
g.	the presence of developmental defects	
h.	perinatal asphyxia or other injuries	

- ***Clinical Presentation***

a.	young infants	- lethargy, pallor, apnoea - bradycardia, hypotension $\equiv^t$ CNS / CVS depression
b.	older child	- tachypnoea, tachycardia, hypertension - restlessness, confusion - ***prior*** to CNS / CVS depression ($\equiv^t$ adult)
c.	respiratory signs	- tachypnoea / apnoea - flaring alar nasi - chest wall retractions - expiratory grunting ± stridor - prolonged expiration ± wheezing - decreased or absent breath sounds - cyanosis
d.	cardiac signs	- tachycardia / bradycardia - hypertension / hypotension - cardiac arrest
e.	cerebral signs	- confusion, irritability, restlessness, combativeness - lethargy - seizures ± coma
f.	general signs	- sweating, pallor - fatigue

Causes of Acute Respiratory Failure		
	Neonate	Small Child
Airways obstruction (see preceding table)	• meconium aspiration • gastric aspiration • congenital abnormalities • tracheomalacia	• bronchiolitis • status asthmaticus • cystic fibrosis • foreign body • croup/epiglottitis
Alveolar disease	• HMD, BPD • CHD + high PBF/HT • pneumonia • aspiration • pulmonary oedema • 2° diaphragmatic hernia • interstitial emphysema • congenital lobar emphysema • congenital lung cysts	• trauma/contusion • CHD & pulmonary HT • pneumonia • near drowning • chemical pneumonitis • pulmonary fibrosis
External compression	• pneumothorax • diaphragmatic hernia • abdominal distension • abdominal wall defects (post repair)	• pneumothorax • haemo/chylothorax • pleural effusion • "TPN/IVT" thorax • thoracic trauma • burns
Neuromuscular disorders	• birth asphyxia • apnoea of prematurity • IC haemorrhage • convulsions • sepsis / meningitis • drugs ± maternal	• trauma • drugs/poisons (OP's) • IC haemorrhage • meningo-encephalitis • tumour • status epilepticus • kyphoscoliosis • Guillain-Barré • poliomyelitis • botulinism

- ***Neonate: General Causes***

1.	respiratory disease	- HMD, aspiration, etc.
2.	neurological disease	- birth asphyxia, ICH - seizures - phrenic nerve palsy, etc.
3.	cardiac disease	- CHD, PFC
4.	abdominal disorders	- diaphragmatic hernia - TOF - gastric distension, SBO

NB: R_X → controlled O_2 therapy
posture and physiotherapy
microbiology - NP swab, skin, NG tube, urine, blood
penicillin & gentamicin
thermoneutral environment
fluid monitoring and restriction
± intubation and IPPV
monitoring - clinical, SpO_2, AGA's, CXR

- ***Infant: General Causes***

1.	respiratory disease	- bronchiolitis, asthma - cystic fibrosis - pneumonia - airway obstruction
2.	cardiac disease	- CHD, myocarditis
3.	neurological disease	- GBS - meningitis, encephalitis - epilepsy - poisoning
4.	trauma	- head, chest, abdomen - Cx spine - drowning

- ***Causes - Specific***

a. ***transient tachypnoea*** - common, especially LSCS

b. ***hyaline membrane disorders*** - surfactant deficiency
- prematurity, maternal diabetes, intrauterine asphyxia, LSCS
- alveolar instability, atelectasis, increased shunt & WOB
- tachypnoea, retraction, expiratory grunting
- CXR: bilateral interstitial pattern & air bronchogram
- complications: severe respiratory failure, BPD
- CPAP → improved P_{aO2}, breathing pattern
  reduced disease progression, lower morbidity

c. ***acute viral bronchiolitis***
- cough, wheeze, low temp., tachypnoea, wheeze ± apnoeas
- $R_X = O_2$, IVT ± CPAP
- *no* benefit from steroids or bronchodilators

d. ***aspiration pneumonitis***
- meconium / gastric contents
- prematurity, birth asphyxia
- oesophageal atresia ± tracheo-oesophageal fistula
- oesophageal reflux
- intracranial haemorrhage
- gastric pH > 2.5, therefore ≠ Mendelsonn's syndrome

e. ***apnoea of prematurity*** > **20 sec** apnoeic spells
- immaturity of brainstem
- chemoreceptor dysfunction
- diaphragmatic fatigue
- ↑ REM sleep component

NB: ***exclude***
- hypoglycaemia
- HMD, aspiration
- sepsis, anaemia
- IC haemorrhage

R_X
- CPAP, IMV
- theophylline

f. ***spontaneous pneumothorax***
- barotrauma in the presence of HMD
- IPPV with aspiration syndrome, pneumonia
- especially lung hypoplasia (including diaphragmatic hernia), Staph. pneumonia, bronchiolitis, asthma, pre-existing PIE
- abdominal distension, unilateral chest hyperexpansion, transillumination of the chest

g. ***pneumonia***
- prolonged rupture of the membranes
- infected birth canal
- immunoparetic state, invasive procedures
- difficult to differentiate from HMD
- most are viral: RSV, influenza, parainfluenza
  * beware group B haemolytic streptococci
  * empyema, bronchopleural fistula, haematogenous spread

h. ***congenital diaphragmatic hernia***
- associated bilateral ***lung hypoplasia***
- ~ 50% mortality if present within 4 hrs of birth
- **> 4 hrs** almost all survive
- IPPV may $\rightarrow$ BPF or pneumothorax on either side
- pulmonary hypertension & persistent foetal circulation
- sample pre/post-ductal P_{aO2}
- respiratory alkalosis, high F_IO_2, avoid acidaemia

i. ***acute severe asthma*** - see below

j. ***congenital heart disease***

i. obstructive lesions

ii. lesions with increased pulmonary blood flow

iii. lesions with decreased PBF

iv. intercurrent infection - especially (ii)

v. post-surgical

k. ***near drowning***
- 2° to either aspiration pneumonitis or hypoxic/ischaemic encephalopathy
- pulmonary oedema ± necrotizing pneumonia may develop
- both fresh & salt water are usually hypovolaemic, hypoxic and acidotic on presentation
- thus, they require volume expansion, oxygen, inotropic support and correction of acidaemia
- associated ***hypothermia*** may afford some brain protection and should not be actively treated before volume resuscitation

l. ***convulsions***

i. newborn
- birth asphyxia
- trauma
- IC haemorrhage
- hypoglycaemia
- hypo-Ca^{++}/Mg^{++}
- pyridoxine deficiency, inborn errors of metabolism

ii. children
- fever
- idiopathic epilepsy
- meningitis, encephalitis
- drugs, poisoning

- respiratory failure 2° to airway obstruction, aspiration, apnoea & respiratory depression
- associated ↑ VO_2 and CO_2 production

m. ***trauma***
- majority are 2° to bicycle and motor vehicle accidents
- isolated CHI is common
- in the very young (< 2 yrs → open sutures), head injury alone may result in hypotension from hypovolaemia
- high cord lesions are difficult to detect with severe CHI
  (NB: rhythmical flaring of the alae nasi ***without*** respiration)
- major damage to the thoracic structures may occur without significant chest wall injury → ***CXR*** is mandatory
- acute gastric dilatation occurs almost invariably and may exacerbate failure
  → R_X nasogastric tube

n. ***poisoning***

o. ***Guillain Barré*** → IPPV if vital capacity is < 15 ml/kg
± early tracheostomy (children tolerate long-term ETT)
± management for muscle pains

p. ***acute respiratory distress syndrome***
- can occur at any age
- most common precipitating causes in children are,

i. shock, sepsis

ii. pneumonia, near drowning, aspiration pneumonia

iii. trauma

iv. ingestion

- management is similar to that for adults
- ***mortality*** in paediatric series is high (28-90%)
- this relates to the severity of the disease, secondary infection, or MOSF

Croup - Acute Laryngotracheobronchitis

Def'n: inflammation of the ***glottic & subglottic*** region (narrowest)

1. ***viral croup***
   - parainfluenzae viruses
   - occasionally RSV, rhinoviruses, or measles
   - coryzal prodrome, low grade fever
   - rare < 6/12, ? underlying lesion
   - commonest obstruction 6/12 to 6 yrs
   - median age of presentation 18/12
   - more common in autumn & winter

   ≤ 5% require intubation
2. ***spasmodic croup***
   - children with an allergic nature

   ? spectrum of asthmatic population
   - no coryzal prodrome / fever
3. ***bacterial tracheitis***
   - usually *Staph. aureus* ± *H. influenzae* group A Strep.
   - high fever, WCC, purulent secretions

   * risk of sudden obstruction

- ***Clinical Presentation***

a. signs of mild croup
   - URTI preceding 2-3 days
   - loud barking "croupy" cough
   - gradual onset ***inspiratory stridor*** which is high pitched
   - hoarse voice
   - no postural preference
   - mild fever
   - often a past history of croup

b. moderate
   - stridor on ***inspiration & expiration***
   - tachypnoea
   - flaring alar nasae
   - suprasternal/intercostal retractions

c. severe
   - restlessness caused by ***hypoxia***
   - exhaustion & listlessness
   - deteriorating conscious state
   - ***cyanosis*** on air

d. differential diagnosis
   - epiglottitis
   - aspiration of foreign body
   - bacterial tracheitis
   - retropharyngeal abscess
   - peritonsillar abscess

e. diagnosis
   i. history and examination * mainstay of diagnosis
   ii. radiology of the larynx (ESS or ICU) →
      • "steeple" sign - AP view
      • widened hypopharynx - lat. view, only ~ 40-50% of cases
   iii. direct laryngoscopy under GA

- ***Management***

a. minimal disturbance - ↓ V_M & VO_2
   - nursed by parent

b. adequate hydration
   • but propensity for ***pulmonary oedema***
   • hypo-Na^+ & convulsions have occurred 2° to ***SIADH*** with airway obstruction

c. ***oxygen*** therapy → SpO_2 > 90%
   • hypoxia from ***parenchymal infection*** ± increased interstitial water

d. humidification
   • mainstay for years but studies showing efficacy are lacking
   • now abandoned by many centres but anecdotal evidence ? otherwise

e. ***steroids***
   • dexamethasone ~ 0.6 mg/kg (≤ 12 mg) stat., then 0.15 mg/kg q6h
   • given on admission → ↓ intubation rate & duration of stay
     ↓ failed extubation rate
   • administer 24 hrs pre & 12 hrs post-extubation
   • may also be of use in spasmodic croup

f. nebulized ***adrenaline***
   • **1:1,000 ~ 0.5 ml/kg** ≤ 5 ml of 0.1% solution, nebulised 2 hrly
   • this dose is effective, has little systemic effect, and is less than the recommended dose for the racemic solution
   • subsequent doses → less effective
   • obstruction may be more severe after the effect has worn-off
     → ***rebound phenomenon*** ? progression of the disease process

   i. acute LTB - lasts ~ 1-2 hrs
      - doesn't alter course
      - may allow secretion expectoration
      - prior to ***intubation***, enhances induction
   ii. spasmodic croup - may obviate need for intubation
   iii. post ETT / endoscopy oedema where effect is often dramatic
   iv. prior to ***transfer*** if not for intubation
   v. prior to anaesthesia & intubation if tolerated

g. antibiotics - only for proven bacterial infection

h. ***intubation*** **~ 2-5%** of cases, nasotracheal
- use 1 mm less than "size for age"

- ***Indications for Intubation***

NB: essentially ***subjective assessment***

a. ↑ respiratory rate, HR, and chest wall retractions

b. cyanosis ***not*** responsive to oxygen

c. exhaustion and/or confusion

d. increased use of, and failure to respond to, nebulised adrenaline

e. need for transport to another hospital

- ***Method***

- spontaneously breathing, inhalational anaesthetic
- induction is ***prolonged*** ∝ ↓ tidal volume
  ↑ V/Q mismatch
- ETT ~ 1 size smaller for age to minimise trauma
- most safely passed ***orally***, then changed to a nasal
- small tubes are ***shorter*** and may be difficult to secure
- sedation ± arm splints to prevent self extubation
- stomach should be emptied with a ***nasogastric tube***
- ***CPAP*** or IPPV with PEEP to maintain oxygenation

- ***Extubation***

- extubation can be attempted when a ***leak*** is present with positive pressure or coughing, or when the disease has run its course at 5 to 7 days
- size limited to > 3.0 mm, due to requirement to pass a suction catheter to clear secretions
- reintubation may be required, but the incidence is reduced by administration of ***steroids*** prior to extubation → ***prednisolone*** ~ 2 mg/kg/day
- prior to steroid therapy intubation duration average 5 days, but now reduced to 2-3 days

Bacterial Tracheitis

- results in purulent secretions, ***pseudomembranes*** and ulceration of epithelium within the trachea
- death can result from upper airway obstruction, endotracheal tube blockage, and toxic shock
- either a ***primary bacterial*** infection or a ***superinfection*** on primary viral illness
- the causative organisms are,
    a. *Staphylococcus aureus*
    b. *Haemophilus influenza* type B
    c. *Streptococcus pneumoniae*
    d. *Branhamella catarrhalis*

Clinical Presentation

a. fever & toxaemia
b. respiratory distress
c. similar to epiglottitis except for
    i. the presence of a ***cough***
    ii. a subjective difference in quality of the ***stridor***
d. diagnosis
    i. CXR - may show tracheal membranes
    - narrowing & "fuzziness" are variable
    ii. ETT - absence of epiglotitis
    - suction following intubation
    → pus and membranes in the trachea

Management

- similar to that for epiglottitis (see over)
- if intubation is required, the ETT may block acutely with secretions
    → aggressive tracheal suction ± reintubation

- bronchoscopy to clear tracheal pus should be considered where the airway remains compromised after intubation, suction and reintubation
- initially, there may not be a leak around an appropriately sized endotracheal tube
- sputum should be sent for gram stain and culture, and urine for rapid antigen identification
- extubation is best performed when,
    a. the fever and secretions have settled, and
    b. a leak is present around the endotracheal tube

- initial antibiotic therapy → ***cefotaxime*** ~ 50 mg/kg q6h for 10/7
    then by MC&S

Epiglottitis

Def'n: ***supraglottic***, infective inflammatory lesion,
caused almost exclusively by *Haemophilus influenzae - type B*
± occasionally streptococci, staphlococci, or pneumococci

a. acute onset - short history (hrs)
- no preceding URTI

b. high fever & ***toxaemia***

c. stridor - low pitched, inspiratory ± ***expiratory snore***
- usually constant in nature

d. absence of ***cough*** and reluctance to ***talk***

e. characteristic ***posture*** - sitting forward
- mouth open
- drooling & dysphagia

f. ***diagnosis***
   i. direct laryngoscopy
   ii. urine latex antigen agglutination
   iii. ~ 80% blood culture (+)'ve
   iv. lateral XRay → "thumb print"

• most commonly children from 2 to 7 years but the disease can involve adults and infants
• due to ***septicaemia***, the severity of the illness is often out of proportion to the airway obstruction
• children less than 2 years of age may present with airway obstruction atypically accompanied by apnoea, URTI, low grade fever, and/or cough
• sudden total obstruction may be precipitated by,

a. instrumentation of the pharynx

b. painful stimuli - eg. IV insertion

c. supine posture

■ ***Management***

a. minimal disturbance - nurse in mothers arms, etc.
- ready access to intubation equipment

b. oxygenation - mask or nasal canulae
- if obstructs → CPAP/assist by bag

c. antibiotics
   i. ***cefotaxime*** ~ 50 mg/kg q6h
   ± chloramphenacol ~ 25 mg/kg q6h
   ii. ampicillin was used but high percentage of resistant strains

d. ***intubation*** - all but the mildest cases
- average duration ~ 18 hours

• may be required for longer in cases with,

i. pulmonary oedema

ii. pneumonia

iii. cerebral hypoxia

e. racemic adrenaline is of ***no use*** in this condition and can precipitate obstruction

■ ***Epiglottitis - Intubation Indications***

1. severe or progressive respiratory distress
2. prior to transportation to a tertiary centre
3. following diagnosis by direct laryngoscopy under GA

• patients can be managed without intubation if they remain in an area where appropriate personnel, equipment and supervision is available
• such patients are generally older, co-operative and are seen early in the day with minimal signs of obstruction
• diagnosis in these cases is made by lateral neck XRay
• an IV line can be inserted before anaesthesia, but should be delayed until after induction when the patient is distressed or obstruction is severe, in order to avoid sudden obstruction
• spontaneously breathing, inhalational GA is best tolerated in the sitting position
• agitation and distress at induction may be due to acute ***hypoxia***
• the patient can be laid flat on loss of awareness, and airway obstruction overcome by application of ***CPAP*** or assisted ventilation
• induction is prolonged, and laryngospasm may be precipitated if laryngeal stimulation occurs prior to surgical anaesthesia being achieved
• copious and persistent ***pulmonary oedema*** fluid may obscure the larynx, making intubation difficult
• an ETT of ***normal size*** for age or one size smaller should be inserted orally then changed to the nasal route once the child has settled
• positive pressure should demonstrate a leak around the tube
• the patient can be sedated ± restrained to prevent self-extubation
• muscle relaxants are ***not*** routinely required unless IPPV/PEEP is required to overcome hypoxia and hypoventilation from pulmonary oedema

- ***Complications***

a. respiratory failure / obstruction

b. pulmonary oedema ~ 7-10% of cases
- precipitated by intubation

i. hypoxia & SNS discharge - ↑ PAP

ii. vascular - endothelial injury & capillary permeability

iii. decreased intrathoracic pressure after intubation
- augmenting venous return, and increasing transmural pulmonary vascular hydrostatic pressure gradients

c. barotrauma

i. pulmonary interstitial emphysema (PIE)

ii. pneumothorax

iii. pneumomediastinum

d. septicaemia / pneumonia

- ***Extubation Criteria***

a. when the ***fever*** has settled

b. signs of ***inflammation*** subside → usually ~ 18 hours

i. pain subsided

ii. able to swallow

iii. free movement of the larynx

NB: exceptions are where hypoxia and reduced lung compliance persist
direct laryngoscopy prior to extubation is ***not*** required

Croup vs. Epiglottitis		
Parameter	Croup	Epiglottitis
Age	• 6-24 months	• 3-7 years
Aetiology	• parainfluenza • RSV, rhinovirus	• Haemophilus infleunzae type B • Group B Strep., Pneumococcus
Seasonal	• autumn, winter	• none
Onset	• few days • preceding URTI	• ***rapid***
Cough	• present, ***barking***	• ***absent***
Dysphagia	• no	• yes ± drooling
Appearance	• pale	• toxic, flushed, febrile
Temperature	• variable, ≤ 39°C	• ***high***, often ≥ 39°C
Posture	• variable	• sitting-up / forward
Stridor	• inspiratory • high pitched	• expiratory snore ± inspiratory • low-pitched
WCC	• usually normal	• often > 15,000
Neck X-Ray	• tracheal narrowing • "steeple sign"	• "thumbprint sign"
Treatment	• nebulized adrenaline	• Cefotaxime 50mg/kg q6h, or • Chloramphenacol 25mg/kg q6h
Intubation • frequency • duration	 • ~ 1-5% • days	 • majority • ~ 1 day
Complications	• obstruction • pneumonitis • "asthma"	• obstruction • pulmonary oedema • septicaemia • meningitis

Supraglottic Obstruction - Other Causes

NB: these may all present in a similar fashion,

i. retropharyngeal abscess
ii. tonsillitis, peritonsillar abscess
iii. infectious mononucleosis
iv. Ludwig's angina

- airway management is essentially the same ± antibiotics
  ± surgical drainage

- the conservative approach to tonsillectomy & adenoidectomy has led to an increased frequency of hypertrophy and chronic upper airway obstruction
- these children may present with an acute exacerbation with intercurrent infection
- removal is generally contraindicated in the acute setting due to the risk of ***haemorrhage***

Foreign Body

- most common between 6 months and 3 years age
- clinical presentation depends on the ***site*** of lodgement,

1.	pharynx / larynx	- respiratory distress - gagging, persistent cough - stridor, dysphonia - sudden total obstruction
2.	tracheal / bronchial	- cough, stridor, wheeze - persistent pneumonia, lobar collapse
3.	oesophageal	- dysphagia, drooling - stridor from tracheal compression

- diagnosis is best made from the ***history***, usually choking while eating, and examination
- AP and lateral XRays only demonstrate radiopaque objects
- inspiratory and expiratory films may show localised ***air trapping***
- management for respiratory arrest includes,

1. holding the child upside down while supporting the airway
2. backblows
3. finger sweep of the pharynx
4. chest thrusts, and abdominal thrusts (Heimlich manoeuvre) in the older child
5. direct laryngoscopy, bronchoscopy, and emergency intubation.

Obstructive Sleep Apnoea

- characterized by ***intermittent*** upper airway obstruction during sleep, with,
    a. heavy snoring & stertorous breathing
    b. an abnormal, irregular respiratory pattern
    c. ***hypopnoea*** → chest wall motion with ***inadequate*** airflow
    d. ***obstructive apnoea*** → chest wall motion with ***no*** airflow

- these episodes occur most frequently in ***REM sleep***, which constitutes,
    a. pre-term infant ~ 65%
    b. 6 months ~ 20%

- the episodes are accompanied by varying degrees of ***arterial desaturation***
- these may be accompanied by cardiorespiratory decompensation
- chronic hypoxia/hypercarbia may lead to progressive ***pulmonary vascular disease***, hypertension and cor pulmonale

- ***Associated Findings***

    a. obesity
    b. enlarged tonsils/adenoids
    c. a large uvula or long soft palate
    d. macroglossia
    e. retrognathia
    f. various neurological abnormalities

    NB: severely affected children may be ***growth retarded***

- ***Surgical Management***

    a. tonsillectomy & adenoidectomy - even if normal size
    b. ± uvulopalatopharyngoplasty
    c. ± tracheostomy

    NB: long term nasopharyngeal intubation or nocturnal nasal CPAP is not feasible in the young child

Pierre-Robin Syndrome

Def'n: congenital syndrome associated with,

1. posterior cleft palate
2. retrognathia & relative macroglossia
3. chronic upper airway obstruction
4. feeding difficulties & failure to thrive in the newborn

- differential growth generally ***reduces*** the significance of the deformity
- acute obstruction may be managed by nursing ***prone*** or the passage of a naso-pharyngeal tube
- intubation is rarely required
- tongue/lip anastomosis is sometimes beneficial

Other Subglottic Lesions

a. burns
b. subglottic stenosis
c. subglottic haemangioma
d. foreign body

Anaesthetic Considerations - Airway Obstruction

NB: → inhalational induction with halothane & 100% O_2
+ ***skilled assistance*** if available

a. adequate preparation - reliable suction, tube sizes, stylets, etc.

b. inhalational induction is ***slow*** with obstruction
- small tidal volumes
- parenchymal lung disease - infection, increased lung water
- if oxygen saturation is adequate, N_2O reduces induction time

c. use the sitting position ± the parent with epiglottitis

d. ***CPAP*** / assisted ventilation will aid induction, but may result in ***abdominal distension***

e. laryngoscopy should only be attempted once a deep plane of anaesthesia is reached

f. ***orotracheal*** intubation is safest & may be performed first
- replacement with nasotracheal intubation following adequate tracheal toilet

g. placement should be at ~ $\mathbf{T_2}$, or the aortic arch/medial claviclular heads on CXR
~ 13 cm + age for children ≥ 1 year (at the naris)
~ (age + 17)/4 ETT size

h. ***humidification*** is difficult → lightweight heat/moisture exchangers

i. require regular ***toileting*** due to inspissated secretions

j. ***sedation*** is rarely required once the obstruction is relieved
- arm restraints may be required to prevent self-extubation
- incidence of spontaneous extubation is 8% to 12%

- if obstruction occurs prior to anaesthesia, immediate ***oral*** intubation should be performed
- emergency cricothyroidotomy and tracheostomy are rarely indicated, except for failure of oral or nasal intubation
- cricothyroidotomy can be performed using a 14G intravenous cannula, with ventilation performed via a 15 mm standard connector from a 3.5 mm ETT
- percutaneous tracheal ventilation requires short inspiratory times and long expiratory times to minimize the risk of ***barotrauma***
- nasal intubation allows secure fixation and greater comfort
- ***subglottic stenosis*** may result from too large a tube,

a. incidence ~ 2% ventilated neonates

b. may be related to duration, reintubation rate, infection and age

- low lung ***compliance*** may produce an excessive leak
- this can be overcome by placing the endotracheal tube tip lower in trachea (not endobronchial), inserting a larger endotracheal tube, or considering a low pressure cuffed tube (the smallest is 4.5 mm ID)
- problems with cuffed tubes include larger outside diameter, trauma and tracheomalacia

Severe Acute Asthma

Def'n: severe asthma ***unresponsive*** to conventional therapy

• incidence is increasing, frequently triggered by ***viral infection***
• patients presenting with one episode of acute respiratory failure are at higher risk of presenting with another

▪ ***Clinical Features***

a. air hunger, tachypnoea, wheeze ± silent chest, cyanosis
   → unreliable for assessment, use ***AGA's***

b. P_{aCO2} - hypocarbia 2° hypoxic drive is usually present
   - normocarbia/hypercarbia ≡^{t} fatigue & failure

c. pulse paradox - should be < 20 mmHg
   - may be low with severe disease & fatigue

d. best assessment of need to intubate → clinical picture

▪ ***Management***

a. supplemental O_2 - hypoxia presumed on presentation

b. IVT - hydration is important for inspissated secretions
   - beware SIADH & oedema
   - total lung water is increased

c. nebulized salbutamol
   • 0.5% solution, 0.05 ml/kg q2-4h
   • can be given neat (undilute) continuously with ***less*** side effects of tremor, tachycardia, hyperglycaemia, and hypokalaemia cf. IV administration
   • < 2 yrs little airway muscle & relatively unresponsive to bronchodilators

d. ***steroids***
   • hydrocortisone 2-4 mg/kg q4h
   • significant benefit at 12 hrs

e. IV salbutamol
   • may obviate need for intubation ~ 1.0 µg/kg/min
   • increment ≥ 20 minutely to 14 µg/kg/min maximum → ↓ P_{aCO2} ≥ 10%
   • equally effective & less side-effects cf. adrenaline
   • indications,
   i. progressive deterioration
   ii. O_2 flows too high for effective nebulisation
   iii. no response to nebulised salbutamol
   iv. patients in extremis

f. ***aminophylline***

- bronchodilator also improves respiratory muscle function and stimulates the respiratory centre
- increased clearance of theophylline < 9 years
- loading dose ~ 10 mg/kg - less if recent administration
- infusion ~ 1.1 mg/kg/hr - cf. adults ~ 0.5-0.7 mg/kg/hr
- serum levels must be monitored, especially when symptomatic
  → vomiting, tremors, convulsions
- * isoprenaline & theophylline may override HPV
  → ↑ shunt, ∴ salbutamol is preferable
- * ***salbutamol & aminophylline*** precipitate, use separate IV's

g. ***intubation / ventilation***

i. progressive exhaustion and hypercapnia despite aggressive therapy

ii. where the patient presents in a terminal state

- usually not required, and morbidity from IPPV is low
- intubation technique should be rapid
- use either a large uncuffed, or a cuffed ETT to minimise leak with high inflation pressures
- ***IPPV*** → low rates with prolonged expiratory times
  minimal peak airway pressures
  volume cycling
  ± adequate V_M *lesser requirement
- ventilation is aimed at correcting ***hypoxia***, not normocapnia
- PEEP may minimise hypoxia, but the use of PEEP for reversal of airway obstruction is not proven
- paralysis and sedation → maximise compliance & ↓ VO_2
- drugs which release histamine are best avoided (eg. morphine, but no evidence)
- complications include barotrauma and muscle weakness

h. ***bronchoalveolar lavage***

- indicated where hypoxia is associated with persistent lobar collapse or localised hyperexpansion
- requires a fibreoptic bronchoscope with a suction channel, and it's use is limited by endotracheal tube size

• ***mortality*** is low and thus extraordinary measures such as anaesthesia (inhalational agents, ketamine) and extracorporeal CO_2 removal are rarely indicated

• there is a high incidence ***metabolic acidosis*** in severe asthma, and HCO_3^- has been advocated to improve bronchodilator responsiveness (ie. adrenergic function), however,

a. ↑ morbidity from untreated acidosis is not proven

b. HCO_3^- does not significantly change pH in asthma unless large doses

c. HCO_3^- → ↑ CO_2 production

d. some don't believe improves adrenergic response anyway - eg M. Fisher

Bronchiolitis

Def'n: acute lower respiratory tract infection of infants

- effects ~ 2% of all infants
- the ***most common*** severe lower respiratory infection
- more frequent in winter months

• age distribution from 6 months to 2 years age (same as croup) is attributed to,

a. loss of protective maternal antibodies
b. aspiration of infected nasopharyngeal secretions
c. small calibre of peripheral airways

▪ ***Aetiology***

a. respiratory syncitial virus (RSV) **~ 70%**
b. influenza, parainfluenza types I and III
c. rhinovirus
d. adenovirus
e. mycoplasma

▪ ***Pathology***

a. lymphocytosis in peribronchiolar spaces
b. inflammation & oedema of submucosa and adventitia in small airways
c. necrosis and ***desquamation*** of small airways epithelium
d. airway obstruction from oedema, cellular debris, and secretions in small airways
e. ***hyperinflation***, atelectasis, ventilation/perfusion inequality
f. ↑ resistance, ↓ compliance and ↑ work of breathing

• ventilation is a compromise between the work required to breathe at high lung volumes and the required minute volume
• this results in ***hypercapnia*** which is tolerated in order to minimise work of breathing
• further progressive increases in P_{aCO2} denote decompensation
• ***mortality*** (≤ 1%) is associated with other serious disease,

a. congenital heart disease
b. bronchopulmonary dysplasia
c. cystic fibrosis
d. congenital lung disease
e. immunosuppressive disorders

- ***Clinical Presentation***

NB: broad clinical spectrum,
from mild URTI → severe pneumonia and respiratory distress

a. preceding URTI

b. symptoms usually last ~ 5-10 days

c. acute onset with rhinorrhoea, cough, dyspnoea, and wheezing
   - copious thick nasal & pharyngeal secretions
   - may have high fever

d. occasional progression to severe respiratory distress

e. ***infants*** present with tachypnoea, hyperinflation, and fine crepitations

f. ***premature infants*** & neonates may present with ***apnoeic spells***, 2° to,
   - hypoxia
   - respiratory muscle fatigue
   - immaturity of respiratory muscle control

g. ***immunofluorescent*** techniques of nasopharyngeal secretions allow rapid virus identification

- ***Complications***

1. acute respiratory failure
2. pneumonia
3. interstitial emphysema, pneumothorax
4. ***obliterative bronchiolitis*** < 1% of cases
   - chronic hyperinflation, collapse, and abnormal small airways
   - usually results from ***adenovirus infection***
5. RSV bronchiolitis can lead to ***asthma*** in older children,
   - ~ 75% have symptoms of wheezing in the subsequent 2 years
   - ~ 22% in the next 10 years

- ***Investigations***

a. CXR - ***hyperinflation*** ± diffuse patchy infiltrates
   - flat diaphragms, horizontal ribs, 'air under heart', etc
   - increased abdominal gas ∝ air swallowing

b. AGA's - hypoxia
   - frequently hypercarbic

c. ***immunofluorescence*** of nasopharyngeal swab

d. serology - 4x rise in RSV titre

- ***Management***

a. supplemental O_2 - head box, nasal cannula or face mask
   - monitor by SpO_2 ± arterial cannula for serial AGA's
   - ? warmed, humidified gases
   - mist inhalations may induce bronchospasm
   - physiotherapy and handling may increase respiratory distress

b. IVT ± mild fluid restriction

c. warmed, thermoneutral environment

d. steroids are of ***no benefit***

e. antibiotics are of ***no benefit***
   - infiltrates on CXR are common
   - there is no increased incidence of bacterial infection

f. bronchodilator therapy
   - trials assessing the effect of bronchodilator therapy have been unpredictable
     → either no response, or improvement
   - a trial of nebulized salbutamol, or IV aminophylline may prove beneficial (especially if apnoea is associated)

g. respiratory stimulation ? aminophylline, caffeine

h. ***riboviron***
   - antiviral agent, limits RSV replication within cells
   - aerosol (~ 1.3 μm) for 3-7 days
   - increases elimination of the virus and resolution of symptoms, and improves oxygenation
   - given orally it is teratogenic in pregnant rodents
   - it precipitates in ventilator circuits
   - ***no evidence*** for earlier discharge or effects on mortality
   - expensive & disease has low morbidity, therefore only considered early in the infection and where there is severe pre-existing cardiorespiratory disease

i. ***nasopharyngeal CPAP***
   - proved helpful in one series but not in another
   - if commenced early, it may reduce incidence of tracheal intubation

j. ***intubation / ventilation***
   - tend to be younger, smaller, and more premature
   - endotracheal CPAP may correct apnoea
   - IPPV is required - bradycardia
     - persistent hypoxia, rising P_{aCO2}
     - exhaustion
   - IPPV is well tolerated few require paralysis
   - sedation may aid synchronisation, and does not prolong weaning provided dose is adjusted to clinical response
   - potential problems - air trapping, barotrauma, ETT obstruction

Cystic Fibrosis

• ***autosomal recessive*** disorder, most common genetic abnormality in Caucasians,

a. gene frequency ~ 1:25

b. incidence ~ 1:2500 live births
~ ¼ of $^{1}/25^{2}$

• median survival (1990) ~ 28 years
• most common molecular basis is deletion of 3 base pairs from long arm of chromosome 7
• eliminates phenylalanine from membrane protein, ***cystic fibrosis transmembrane conductance regulator*** **CFTR**, which permits apical membrane conductance of water
• major organ systems affected,

1. respiratory
   - i. upper airway - chronic sinusitis, polyposis
   - ii. lower airways
     - bronchial hyper-reactivity
     - inflammatory cell activation and tissue destruction
     - bronchiectasis, abscess formation, empyema
     - colonisation - *H.influenzae, S.aureus, P.aeuroginosa, P.cepacia*
     - pneumothorax
     - haemoptysis - bronchial artery errosion/rupture
2. pancreatic insufficiency
   - i. exocrine - malabsorption syndromes
   - ii. endocrine ~ 75% have glucose intolerance
     ~ 15% → ***diabetes mellitus***
3. gastrointestinal
   - i. meconium ileus ~ 12% of presentations at birth
   - ii. gastro-oesophageal reflux
   - iii. recurrent constipation
   - iv. rectal prolapse
4. hepatobiliary
   - i. fatty liver ~ 40%
   - ii. focal cirrhosis ~ 25%
   - iii. cholelithiasis ~ 12%
5. malnutrition - multifactorial
6. immune suppression

Respiratory Failure - General Management

1.	thermoneutral environment	- humidicrib - overhead heater - room temperature control $\rightarrow$ minimise VO_2
2.	diaphragmatic movement	- abdominal contents - prone or head-up position - NG tube
3.	cease feeding	- diaphragmatic movement - microaspiration
4.	minimal handling	- dynamic airways collapse - reduces VO_2
5.	monitoring	- HR, RR, SpO_2, P_{aO2} and P_{aCO2} - routine CXR's

- ***Complications of Oxygen Therapy***

a. retrolental fibroplasia (retinopathy of prematurity)
   ? absolute duration
   ? level of hyperoxia
   $\rightarrow$ P_{aO2} ~ 50-80 mmHg
   - retinal receptors mature from the centre to the periphery of the retina
   - pattern results from high O_2 consumption during development, $\therefore$ ordered formation from centre $\rightarrow$ out
   - hyperoxia allows proliferation in multiple regions simultaneously, $\therefore$ results in a disorganised vascular pattern
   - ? frequency reduced by vit.E and other antioxidants

b. bronchopulmonary dysplasia $\propto$ peak inspiratory pressures
   $+$ other evidence of barotrauma

c. resorption atelectasis

d. ? acute lung injury / O_2 toxicity

Intubation - Disadvantages

a. risks / complications of intubation procedure

b. bypasses the humidifying action of the nose

c. ***increases*** total airway resistance

d. risk of subglottic stenosis

e. interference with cough reflex

f. loss of physiological PEEP - "laryngeal braking"

g. impairment of pulmonary defence mechanisms
   - increased incidence of ***nosocomial pneumonia***

- the subglottic area is relatively narrow, and an ET tube small enough to be passed through the larynx may be too large to be inserted into the trachea
- the ETT is easily malpositioned because,

a. the trachea is short ~ 4-5 cm in neonates

b. the tube changes position with head and neck movement
   → in with flexion
   out with extension

- the smaller airways and endotracheal tubes are easily blocked with secretions

   → patients require frequent ***suctioning*** and constant ***humidification***, by servo-controlled humidifiers or moisture exchangers

- the correct size tube is one which allows a small ***leak*** with IPPV ~ 25 cmH_2O
- exceptions to this rule are,

a. neonates - absence of a leak rarely causes problems
   - problems correlate with duration & re-intubation frequency

b. croup - the appearance of a leak $\propto$ disease resolution

c. IPPV with low compliance lung disease

d. Down's synd. - often have subglottic narrowing & require a smaller tube

CPAP

- ***Benefits***

1. ***increases FRC***, stabilises alveoli, reduces shunt fraction

   → allows a reduction of F_IO_2
2. promotes both small and large ***airways stability***
   - airway obstruction
   - bronchomalacia, tracheomalacia
   - croup, bronchiolitis, asthma
3. decreases the ***work of breathing***
4. reduces auto-PEEP
5. may abolish ***apnoeic spells*** in neonates & improves the respiratory pattern

   → small (physiological) levels should be applied wherever possible
   ≤ 3-5 cmH_2O, to prevent airway closure

- requires a fresh gas flow,
   a. ~ 2-3x minute ventilation
   b. ≥ peak inspiratory flow rate
   c. or requires use of a reservoir bag

- nasotracheal intubation is the safest means of administration
- however, a nasal mask or a single nasopharyngeal tube may be used

- ***Complications***

1. ↑ incidence of ***barotrauma*** *potentially
2. ↓ cardiac output
3. ↓ GFR
4. ↑ secretion of ADH → fluid retention
5. ↑ PVR and ***RV afterload***
   - this is balanced against the ↓ PVR which follows opening of small airways and expansion of areas of atelectasis

Indications for Mechanical Ventilation

1. general anaesthesia with muscle relaxation
2. cardiopulmonary resuscitation
   i. respiratory / cardiac arrest
   ii. severe LV failure / acute pulmonary oedema
      • as a form of circulatory support
3. acute / chronic respiratory failure
   i. maintenance of adequate ***gas exchange*** → parenchymal failure
      • to maximise DO_2 - reduce work of breathing
      - paralysis, reducing VO_2
   ii. minimise ***work of breathing*** → pump failure
4. manipulation of CO_2 excretion
   i. induced hypocapnia - metabolic / respiratory acidosis
      - raised ICP, acute head injury
   ii. ∝ ↑ CO_2 production - MH, thyroid storm
   iii. manipulation of PVR - pulmonary hypertension ± cor pulmonale
      - CHD with R→L shunt
      - transitional circulation in the newborn
5. "prophylactic" ventilation - severe flail chest
   - major, chest & upper abdominal surgery
   - unstable patients for transport

• ***time-cycled, pressure limited*** ventilation is used for neonates and infants less than 10 kg weight
• this compensates for leak around the ETT and overcomes the problem of a relatively large circuit compliance and compressible volume compared to the small tidal volume
• however, this form of ventilation has problems,

a. the inspiratory waveform pattern is dependent on,
   i. the ***flow*** through the circuit
   ii. the ***resistance*** of the circuit
   iii. the performance of the ***expiratory valve***
b. tidal volume varies with pulmonary compliance & resistance
c. in patients spontaneously breathing or receiving IMV, stability of inspiratory and expiratory pressures is not maintained with varying flows in the respiratory cycle, resulting in suboptimal work of breathing
d. on older ventilators there is no ability to synchronise ventilation, or calibrate PEEP and CPAP → these problems have been overcome in modern ventilators with acceptable flow heads at the patient T-piece and digitally controlled valves

Mechanical Ventilation - Complications

a. airway trauma
   i. nasal passages
   ii. mouth & pharynx
   iii. tracheal trauma - subglottic stenosis
   - ulceration

b. barotrauma
   i. pulmonary interstitial emphysema (PIE)
   ii. pneumothorax
   iii. pneumopericardium, pneumomediastinum
   iv. pneumoperitoneum

c. raised mean intrathoracic pressure
   i. ↓ cardiac output
   ii. ↓ GFR
   iii. fluid retention - ↑ ADH / ↓ ANF

d. equipment related
   i. disconnection
   ii. ETT obstruction
   iii. ventilator malfunction

e. nosocomial infection

f. microaspiration / macroaspiration

Indications for Tracheostomy

a. failure to achieve intubation by the oral or nasal route

b. congenital or traumatic upper airway obstruction

c. following craniofacial surgery

d. long term ventilation in children - GBS
   - quadriplegia
   - neuromuscular diseases

• paediatric patients can be managed for long periods with nasotracheal tubes ***without*** long term sequelae and tracheostomies are rarely performed
• percutaneous tracheostomy has not been described
• ***cricothyroidotomy*** is preferable in emergencies for small children and infants

Extracorporeal Membrane Oxygenation (ECMO)

- pulmonary bypass procedures for neonates has been used in the U.S.A.
- limited to those patients with acute, potentially reversible pulmonary failure, who fail to respond to conventional therapy
- attempts to identify this group remain difficult

a. ***neonates*** - need to fulfil the following criteria:

i. acute reversible disease - eg. meconium aspiration

ii. ³ **80%** predicted mortality by statistical analysis

iii. no other abnormality incompatible with life

iv. body weight > 2.5 kg

- limitations in body size and the risk of haemorrhage

b. ***children***

- attempts have been made to identify those with predictably high mortality, and it's use has been extended to include,

i. bypass dependence following cardiac surgery

ii. catastrophic post cardiac surgical events

iii. reversible lung disease - aspiration pneumonia
- uncontrolled air leak

- the ***advantages*** of ECMO are,

a. lost lung function is directly replaced

b. technical success is independent of disease severity

c. further lung damage is limited

- complications include,

1. bleeding from ***heparinisation***, as completely heparin bonded circuits are yet to be developed

2. the effects of large vessel cannulation and ligation (EJV & ICA)

3. platelet & WBC activation

- side effects of vessel ligation appear acceptable and reconstruction techniques are now available
- outcome from ECMO for neonates is good, with impressive survival figures

~ 75 to 80% survival in those patients with 80% predicted mortality

- however, no adequate controlled trials have been performed

Surfactant

- a phospholipid produced by alveolar type II cells
- trials of surfactant administered via the trachea have shown ***improved outcome*** in neonates susceptible to hyaline membrane disease
- sources of exogenous surfactant are,

    a. modified natural surfactant
        - lipid extract of animal lung - ***bovine*** most commonly used

    b. human surfactant recovered from ***amniotic fluid***

    c. ***synthetic*** dipalmitoylphosphatidylcholine

- ***indications*** have not been standardised but are based on,

    a. age

    b. $P_{A\text{-}a}O_2$ gradient

    c. positive inflation pressure

    d. duration of ventilation

- results, when given prophylactically, show significant decreases in acute complications of neonatal respiratory distress syndrome,

    a. ***mortality*** 30% $\rightarrow$ ~ 12%

    b. ***barotrauma*** 40% $\rightarrow$ ~ 8%

RENAL SUPPORTIVE THERAPY

• renal failure in the critically ill patient is prevented by,

a. maintaining or improving ***RBF*** despite other organ failure

b. careful monitoring/avoidance of ***nephrotoxic drugs***
   ± vigorous use of loop diuretics (frusemide) and inotropes (dopamine)
   → normal or high output failure

• high output ARF being easier to manage than oliguria, and may not require renal replacement therapy

• the choice between peritoneal dialysis (PD), haemodialysis (HD), or continuous arteriovenous haemofiltration (CAVH) is governed by a number of factors,

a. no modality has been demonstrated superior in ***outcome*** in ARF

b. HD is more effective than PD in highly catabolic patients

c. PD clearance is impaired in
   - microangiopathies
   - heatstroke

d. advantages of PD include
   - technically simpler
   - widespread availability
   - useful for infants
   - useful post CPB

Continuous Haemofiltration

• haemofiltration is either arterio-venous (CAVH) with flow from the arterio-venous pressure difference, or veno-venous (CVVH) requiring flow from an extrinsic pump
• the ***ultrafiltrate*** formed is proportional to,

a. the hydrostatic pressure gradient
b. the membrane area & mean pore size

• this UF is then replaced IV with a solution of desired composition
• haemodiafiltration is where dialysate is perfused across the filter
• indications for haemofiltration are,

a. acute renal failure
b. fluid overload / pulmonary oedema
c. metabolic derangements - hepatic failure
   - severe electrolyte or acid-base imbalance
d. fluid volume limitations that restrict nutrition
e. drug and poison removal

• haemofiltration is most useful for fluid removal in cardiovascularly unstable patients, but is less rapid and effective than haemodialysis
• it removes middle molecular weight vasoactive peptides that may lead to capillary leakage & contribute to the "sepsis syndrome"

• problems of continuous haemofiltration in children are,

a. additional arterial ± venous lines
b. blood flow and UF flow are dependent on,
   i. arterial blood pressure (which is lower in children),
      or, blood flow through the pump (CVVH)
   ii. haematocrit
   iii. position, size and length of catheters - greater dead space
c. greater circuit::blood volume ratio
   i. dilution
   ii. heat loss
   iii. hypo / hypervolaemia with pump failure
d. regional heparinisation may cause bleeding
e. ***platelet sequestration***, especially at low blood flows in paediatric patients
f. microaggregates are flushed into the venous circulation

• CAVH is simpler because the A-V pressure gradient drives blood through the filter

→ this provides safety and haemodynamic stability

• however, with small paediatric cannulae and lower blood pressure, blood flow rates are low urea clearance is reduced
• blood flow can be improved by,

a. correcting hypovolaemia
b. increasing blood pressure
c. reducing blood flow resistance
   i. reducing cannula length
   ii. increasing cannula size
   iii. changing cannula position

• continuous arterio-venous diafiltration improves urea clearance
• CVVH via a central venous dialysis catheter must be pump driven, but provides higher blood flow and ultrafiltration rates, with better urea clearance
• CVVH is technically more difficult than CAVH in infants
• haemodialysis allows controlled ultrafiltration and dialysis
• it requires relatively large central vascular access, specialised personnel and regional heparinisation, and is expensive
• it may cause rapid osmotic shifts and haemodynamic instability

Peritoneal Dialysis PD

• peritoneal dialysis is inexpensive and provides smooth changes in fluid volume
• a soft, purpose-designed catheter is inserted into the peritoneal cavity using a Seldinger technique
• ***respiratory function*** may be affected in infants because raised intra-peritoneal pressure impairs diaphragm function
• complications include,

a. infection
b. catheter blockage
c. leakage of dialysate fluid and bowel perforation

• it is contraindicated where abdominal pathology is present or recent surgery has been performed

NEUROLOGICAL EMERGENCIES IN CHILDREN

- these are the most common causes of life-threatening injury & death in children
- ***SIDS*** outranks all other causes of death in ***infants*** by ~ 10x
- after the first year, ***trauma*** accounts of ~ 50% of all deaths
- ***primary brain injury*** results from,
    a. trauma
    b. ischaemia
    c. infection
    d. metabolic disturbance

- ***secondary injury*** results from,
    a. oedema - acute vasogenic cerebral oedema
    b. altered cerebral autoregulation
    c. tissue hypoxia, reperfusion injury
    d. other cytotoxic events

- factors pertinent to the paediatric population include,
    a. ***diffuse cerebral swelling***
        - occurs commonly and early in severe CHI
        - may be progressive with development of ***vasogenic oedema***
    b. ***cerebral blood flow***
        - ICP & MAP vary with age
        - autoregulation is easily disrupted
        - with vasogenic oedema, hypertension may worsen ICP
    c. ***hypovolaemia***
        - commonly occurs 2° to scalp or intracranial bleeding
    d. anatomical differences
        - large head, weak neck muscles, short stature
            → ***isolated*** severe head injury is common
        - under 2 years the ***sutures*** are open and the vault may expand
        - high cervical cord damage may occur ***without*** bony damage (SCIWORA)

Causes of Coma in Children	
Structural	Metabolic
trauma • accidental • child abuse	infection • meningitis • encephalitis
hydrocephalus blocked CSF shunts	hypoxia / ischaemia circulatory shock / arrest
tumours	drugs / toxins
intracranial haemorrhage	postictal / status epilepticus
infection • meningitis • encephalitis • abscess	biochemical - Na^{+}/H_2O - Mg^{++}/Ca^{++} - pH - hypoglycaemia
	hyper / hypothermia diabetic ketoacidosis hepatic failure Reye's syndrome complication of haemodialysis haemolytic uraemic syndrome hypertensive encephalopathy inborn errors of metabolism

Intracranial Pressure

a. 2 years of age ≤ 5 mmHg
b. 5 years of age ≤ 10 mmHg
c. > 10 yrs / adults ≤ 15 mmHg

- elevation *per se* is ***not*** an indicator of poor ***outcome***, unless persistently > 40 mmHg
- symptoms and signs of raised ICP are,
  a. depressed ***conscious level***
  b. vomiting, headache and ***papilloedema***
  c. strabismus
  d. changes in blood pressure, heart rate and respiratory pattern
  e. in infants with ***open sutures***,
     i. the fontanelle is full
     ii. head circumference increases
     iii. papilloedema is uncommon

- physiological compensations for raised ICP are,
  a. displacement of CSF → spinal canal
  b. ↑ CSF resorption | ↓ CSF production
  c. compression of intracranial veins → may worsen ICP
  d. increase in head size

- in the ***infant***, gradual increases in volume of intracranial contents are accommodated by an increase in head circumference, and this can delay clinical signs and diagnosis
- the limiting factor on whether the ICP rises quickly or there is an increase in head size is the elasticity of the ***dura***
- acute increases in head circumference is limited to children £ ***18 months***
- over this age, any additional intracranial volume must be accommodated by displacement of blood, CSF and brain
- signs of ***cerebral herniation*** are,
  a. abrupt changes in level of consciousness ± coma
  b. irregular respiratory pattern
  c. peripheral weakness / focal neurological signs
  d. cranial nerve palsies - including pupillary dilatation
  e. decorticate or decerebrate posturing
  f. cardiorespiratory failure

Cerebral Perfusion Pressure

$$CPP = MAP - ICP \quad \text{(when ICP > CVP)}$$

• dependence on blood pressure is important in the younger age group because physiological blood pressures are low and autoregulation is disturbed
• normal ***systolic*** blood pressure, 50th percentiles,

a. 1-6 months ~ 85 mmHg
b. 2 years ~ 95 mmHg
c. 7 years ~ 100 mmHg

• in younger age groups, CPP is more easily encroached upon, and relative ***hypotension*** has a significant effect on CPP and outcome
• ***hypotension*** may be the principle cause of cerebral circulatory failure and infarction, resulting in complete cessation of CBF
• ***CPP < 40 mmHg*** reduces the likelihood of good outcome, and is critical for a range of paediatric management
• if vasogenic oedema is present (trauma, hypoxia/ischaemia, infection), hypertension may worsen oedema

Cerebral Blood Flow

• metabolism requires constant supply of oxygen **~ 3.3 ml.O_2/100g/min**
• CBF is maintained at 50-60 ml/100g/min over a range of MAP by autoregulation

→ 50 ml/100g/min ~ 10 ml.O_2/100g/min → O_2 ER ~ 35%

• abnormal CBF is caused by,

a. gross changes in P_{aCO2} and P_{aO2}
b. convulsions
c. head injury
d. drugs - eg vasodilators
e. ↑ temperature

• regional pressure, regional perfusion and total blood flow are not absolutely linked, and ***focal oedema*** can effect local cerebral blood flow despite an adequate CPP
• attempts to improve monitoring have led to measurement of cerebral blood flow as a clinical indicator of changes in regional perfusion, but this is technically difficult and subject to significant errors

Management

• the aims of therapy are to,

1. reverse the 1° disease processes
2. maintain CBF to prevent 2° ischaemic injury
3. prevent herniation from raised ICP

NB: there is ***no evidence*** that therapies aimed at reducing ICP, maintaining cerebral blood flow, and improving cerebral perfusion pressure (CPP) improve ***outcome***

• however, monitoring these parameters allows for assessment of effects of therapy and routine clinical interventions, and for ***outcome prognostication***

a. initial
- assessment/management of ABC
- venous access, blood for routine tests
- 0.5 ml/kg 50% dextrose if ? hypoglycaemia
- history & examination

b. controlled ventilation

i. apnoea, respiratory failure, or poor airway control

ii. rapidly worsening coma GCS < 9

iii. evidence of advancing IC hypertension

- following this the stomach should be drained via NG tube
- hyperventilation ± 15-30° head up (?? CPP better flat)
  ± mannitol 0.25 g/kg
  ± frusemide 1 mg/kg
  ± NMJ blockade
- beware excessive diuresis → ***hypovolaemia***

c. circulation
- frequently hypotensive / hypovolaemic
- support MAP for age
- non-hypoosmotic fluids

d. CT scan
- coma & localizing signs
- no diagnosis

e. LP
- suspicion of meningitis, encephalitis
- no evidence of raised ICP
- defer until ***after*** CT scan if in doubt
- IC haemorrhage better defined by CT

f. ultrasound
- when the fontanelle is open
- ventricular size & IC haemorrhage

g. EEG
- focal or non-specific global abnormalities

h. other I_X
- U&E's, AGA's
- metabolic screen (LFT's, NH_3, amino and organic acids)
- blood, urine & gastric fluid for toxicology
- blood cultures and urine antigen screen
- virology for HSV, enteric viruses, CMV, measles, and rubella

Head Injury

- majority are from road trauma (MVA, pedestrian, cyclist)
    a. age < 1 yr → 3rd leading cause behind SIDS & congenital anomalies
    b. age > 1 yr → leading cause of death

- presence of early hypoxia, hypercarbia or hypotension with severe CHI confers a bad prognosis
- factors in initial assessment peculiar to paediatric patients,
    a. GCS modified for age
    b. acute gastric distension → NG tube
    c. significant liver, lung, spleen & kidney trauma may occur ***without*** bony trauma
    d. major blood loss with ***hypotension*** may be ***concealed***
    e. higher incidence of
        i. seizure activity
        ii. mass lesions
        iii. white matter tears - frontal and temporal lobes
            - especially infants < 6 months
        iv. subdural haematomas - especially NAI

- indications for further monitoring include,
    a. ***CT scan***
        - all children with modified **GCS £ 8**
        - presence of focal neurological deficit
        - less severe injuries prior to prolonged anaesthesia / procedures for other injuries
    b. ***ICP monitoring***
        - GCS ≤ 8 with cerebral swelling on CT scan
        - following drainage of cerebral collections
        - ? best method but intraventricular catheter allows CSF removal
        - where NMJ blockade obscures signs of ICP

Modified Glasgow Coma Scale			
	≤ 1 year	> 1 year	Score
Motor Response	 localises pain[1] withdrawal decorticate[2] decerebrate[3] flaccid	obeys localises pain withdrawal decorticate decerebrate flaccid	**6** 5 4 3 2 1
Eye Opening	spontaneous to voice / noise to pain nil	spontaneous to command to pain nil	**4** 3 2 1
Verbal Response			
0-2 years	2-5 years	**> 5 yrs**	
appropriate smile/cry	appropriate smile/cry	oriented/converses	**5**
crying	inappropriate words	disoriented	4
irritable crying	irritable crying	inappropriate words	3
grunts	grunts	incomprehensible	2
nil	nil	nil	1
		Total Score	3-15

[1] some score GCS/14 for ages < 1 year

[2] decorticate = abnormal flexion, flexion/extension & crossed patterns

[3] decerebrate = extension ± clonus

- ***Prognosis - Coma***

a. in large series
- variable figures
~ 3% mortality
~ 2% severe disability
~ 95% normal

b. severe CHI (GCS ≤ 8) ~ 20-40% mortality cf adults ~ 40-50%

c. poor prognostic factors
   i. initial GCS ≤ 4
   ii. apnoea
   iii. absent pupillary/vestibular reflexes
   iv. subdural or multiple IC haematomas
   v. intractable high ICP

Management - Head Injury

NB: maintain CBF, DO_2 & avoid hypercarbia

a. IPPV + muscle relaxation & sedation
→ $P_{aO2} \geq 100$ mmHg / $P_{aCO2} \sim 35$ mmHg

b. prevent rises in ICP - head-up ~ 30° & neutral position
~ 30% of maintenance fluids
(no evidence that this works)

c. treatment of ICH > 20 mmHg ICP persistently
? R_X at > 15 mmHg better prognosis

i. hyperventilation → $P_{aCO2} \sim 25\text{-}30$ mmHg
- effect wanes over hours
- excessive may decrease CBF
* RAH study in adults showing $\downarrow S_{jb}O_2 \propto \downarrow P_{aCO2}$
- rebound on cessation

ii. diuretic therapy - mannitol 0.25 g/kg
- may be repeated 1-2 hrly
- ≤ 325 mOsm/l maximum effect ? also decreases viscosity
- frusemide 0.5 mg/kg IV ? also decreases CSF formation
- synergistic with mannitol

iii. CSF removal - situate drain at set height above the tragus

iv. barbiturate therapy
- decrease $CMRO_2$, blood volume and ICP with bolus injection
- ***no*** studies show morbidity or mortality reduced with infusions

v. surgical decompression → bifrontal craniectomy
- rarely used for high ICP
- some American centres use this in adults

d. hypovolaemia - occurs more commonly in children
- especially scalp & intracranial
- associated intra-abdominal

e. seizure prophylaxis **~ 7.2% *risk*** with severe CHI in child
- phenytoin ~ 20 mg/kg IV + 3 mg/kg q8h

f. steroids??

g. surgery for mass lesions

Prolonged Seizures

• the ***common causes*** of prolonged seizures are,

a. known epilepsy + - withdrawal of anticonvulsants
- intercurrent infection & fever

b. CNS infection - meningitis
- encephalitis

c. febrile convulsion **atypical***
- usually → short duration ≤ 15 minutes
absence of focal signs
absence of post-ictal features

d. metabolic disturbance - hyponatraemia
- hypocalcaemia
- hypoglycaemia

e. trauma

f. NAI

■ ***Management***

a. ABC

b. diazepam ~ 0.1-0.2 mg/kg IV/PR, up to 0.5 mg/kg

c. phenytoin ~ 20 mg/kg IV then 3-4 mg/kg q8h (minimal sedation), or,
phenobarbitone ~ 20 mg/kg IV

d. thiopentone ~ 2-5 mg/kg IV, then 1-5 mg/kg/hr by infusion
- seizures are only controlled by anaesthetic doses
- intubation and IPPV are therefore ***mandatory***

e. management of metabolic / respiratory acidaemia

f. LP / CT scan

• in neonates, seizures may be subtle and difficult to diagnose, with signs being irregular breathing, apnoea, nystagmus and "bicycling" movements

NB: ***HSV encephalitis*** is frequently atypical in children, thus the early use of ***acyclovir*** in febrile patients with unknown cause is justified

→ early therapy is associated with a markedly reduced morbidity & mortality

Bacterial Meningitis

- the major route of spread is ***haematogenous*** from the nasopharynx
- it may result as a local complication of,
    a. head trauma involving the paranasal sinuses
    b. neural tube defect
    c. dermoid sinus
    d. middle ear infection
- the causative organisms are usually,
    a. *Haemophilus influenzae* - type B
    b. *Neisseria meningitidis*
    c. *Streptococcus pneumoniae* - sickle cell anaemia
    - post splenectomy
- the classical clinical findings,
    a. fever
    b. headache, painful stiff neck
    c. photophobia
    d. altered conscious state

    NB: these may be absent in young children or following seizures, and may be obscured by partial treatment
- there may be over-ridding features of ***septic shock***
- ***petechiae / pupura fulminans*** may be seen not only associated with meningococcus, but also with pneumococcus and Haemophilus
- an atypical history with lower cranial nerve signs may represent TB

    NB: the common pathogens can frequently be discerned using ***latex agglutination antigen*** testing of the CSF or urine

- pathophysiology includes,
    a. early transient ventricular dilatation
    b. cerebral oedema - cytotoxic and vasogenic
    c. vasculitis - resulting in thrombosis/infarction
    d. arterial spasm
    e. cortical vein thrombosis

- ***Differential Diagnosis***

a. infection - viral encephalitis
- fungal / tuberculous meningitis
- cerebral abscess

b. tumour - cerebral neoplasm, meningeal carcinomatosis
- leukaemic infiltration of meninges

c. subarachnoid haemorrhage (uncommon in children)

- ***Investigation***

a. FBE - ↑ WCC, ↑ ESR
± anaemia, thrombocytopaenia

b. INR / APTT ± DIC screen

c. E,C+U, CaP, LFT, BSL

d. urine ***antigen screen***

e. blood cultures ± fluid from other suppurative foci

f. CXR ± SXR if sinisitis / otitis are origin

g. lumbar puncture
- ↑ WCC - usually > 1000/ml
- ↑ protein - marked rise in TB
- ↓ glucose

- organisms on ***gram stain*** ± bacterial antigen determination
- increased lactate > 4 mmol/l → ↑ morbidity
- LP ***should not*** be performed when,

i. the diagnosis of meningitis is clear
ii. the patient is seriously ill, or
iii. there is evidence of raised ICP

- ***Complications***

a. profound coma → - 2° complications

b. uncontrolled seizures

c. persistent focal signs
- hemiparesis
- hearing loss (esp. pneumococcus)

d. suppurative lesions
- pericarditis
- septic arthritis
- pneumonia

e. immune complex disease
- arthritis
- glomerulonephritis

f. SIADH & hyponatraemia

• these complications may occur in the presence of,

a. infarction
b. cerebral oedema
c. subdural effusion - persistent fever & signs
d. cerebral abscess
e. venous sinus thrombosis

■ ***Management***

a. ABC
b. IVT
   • ~ 1/3 normal maintenance H_2O, once ***normovolaemic***
   • ***SIADH*** almost always occurs
   • hypotonic fluids may → hyponatraemia & cerebral oedema coma, fitting ± death
c. ABx
   • for community acquired → 3rd generation cephalosporin ***cefotaxime*** ~ 50 mg/kg tds
   • once sensitivities known continue R_X for 10 days
d. ***prophylaxis***
   • every case of *Strep. pneumoniae*
   • *H. influenzae* with another child ≤ 5 years
   i. infants/children → rifampicin ~ 20 mg/kg/day (max 600) for 4 days
   ii. neonates → rifampicin ~ 10 mg/kg/day for 4 days
   iii. pregnant women → ceftriaxone ~ 25 mg/kg stat
e. ***dexamethasone***
   • 0.15 mg/kg q6h for 4 days → ↓ ***deafness*** with *H. influenzae*
   • given with the first dose of antibiotics when the diagnosis is proven or strongly suspected

Neonatal Meningitis

- typically present with a paucity of clinical findings,
    a. poor feeding
    b. weight loss, failure to thrive
    c. loss of thermoregulation
    d. respiratory distress, apnoea
    e. metabolic disturbances - hypoglycaemia
       - hypocalcaemia

- causative agents include,
    a. group B haemolytic streptococci
        - most common, often associated with ***fulminant sepsis***
    b. *E. coli* & gram negative rods
    c. *Listeria monocytogenes*

    NB: (a + b) were the causative agents in > 70% of cases in one large review
    (c) responsible for ~ 5%

- ***ventriculitis***, with surrounding cerebral oedema and communicating ***hydrocephalus*** occurs more commonly in neonates
- therapy is similar to that for older children, initial AB_X cover,
    a. amoxicillin ~ 100-200 mg/kg/day, ***plus***
    b. cefotaxime ~ 150-200 mg/kg/day, or
       gentamicin ~ 2.5 mg/kg q12h

    NB: although aminoglycosides have poor penetration into CSF,
    direct instillation SA or intraventricular in neonates is of ***no benefit***

    3rd generation cephalosporins have good activity against most GN enteric organisms but not against *Pseudomonas spp.*, or against *L. monocytogenes*

Herpes Simplex Virus Encephalitis

NB: the most common cause of severe, often fatal encephalitis

a. wide range of symptoms from mild illness to sudden deterioration and death

b. usually a non-specific acute systemic illness
   → fever, headache, nasopharyngitis, & screaming spells in infants

c. progressive symptoms
   i. nausea and vomiting
   ii. lethargy, stupor
   iii. neck stiffness, photophobia
   iv. bizarre movements
   v. focal neurological signs
   vi. convulsions ± coma

- ***Investigations***

a. CT Scan
   - localised or generalized changes
   - may be normal in the first 2-3 days

b. LP
   i. ICP - universally raised in encephalitis
   ii. CSF
   - ↑ WBC (predominantly lymphocytes)
   - ↑ protein & ↓ glucose
   - often blood stained

c. ***EEG***
   - focal changes
   * the most common abnormal neuroradiological test

d. viral studies
   - isolation from peripheral sites is unhelpful
   - Ab responses are not always positive at time of infection
   - the virus is ***rarely*** isolated from CSF (PCR takes 2 weeks)
   → these may be ***normal*** early in the disease

- ***Management***

a. ***acyclovir*** ~ 10 mg/kg 8 hrly IV reduces mortality
   - commence empirically ***without*** brain biopsy
   - phosphorylated by viral ***thymidine kinase***
     → inhibition of HSV ***DNA polymerase***
   - side effects
     - nephrotoxicity
     - encephalopathy, agitation, seizures & coma

b. general
   - maintenance of cerebral blood flow
   - monitoring and reduction of ICP

Hypoxic-Ischaemic Encephalopathy

• the commonest causes in children are,

1. SIDS
2. immersion - salt/fresh water
3. accidents - drug ingestion
   - child abuse
   - strangulation

• anaerobic glycolysis produces 1/19th the ATP and requires the conversion of pyruvate to lactate to provide NAD^+ for ongoing glycolysis
• if ischaemia accompanies hypoxia, there is also a failure of substrate removal which amplifies the cellular insult
• ischaemia produces coma in ~ 10 seconds and cellular injury in as little as 2 minutes

▪ ***Management***

• same principles of ABC as for other arrest/brain injury scenarios
• large volumes of air/water may be in the stomach after immersion & resuscitation
• in 10-15% of immersion, early ***laryngospasm*** prevents aspiration → ***dry drowning***
• common problems after prolonged arrest,

1. cardiac dysfunction requiring inotropic support
2. hypovolaemia from GIT fluid loss & ischaemic diarrhoea

• comatose patients with a GCS < 8 should be ventilated for several days, though, this is of ***unproven*** benefit in outcome
• barbiturate coma & induced hypothermia are of ***no*** proven value and increase the risk of sepsis
• ***hyperglycaemia*** should be actively treated as this has been shown experimentally to worsen prognosis

▪ ***Prognosis***

• the onset of ischaemia may be delayed by bradycardia with preferential cerebral blood flow, the ***diving reflex***, in young children
• survival from out-of-hospital arrest presenting in ***asystole*** is poor
• the exception is ***hypothermia*** following immersion, where prolonged resuscitation is justified
• recovery is likely in comatose patients who respond to ***pain*** → flexion or extension
• normothermic patients who are flaccid & apnoeic are unlikely to survive
• in contrast to isolated head injuries, defects present at the end of 1 week are unlikely to recover further

Guillain-Barré Syndrome

- the most common cause of ***acute motor paralysis*** in children, the usual presentation being,
  - → ascending symmetrical areflexic weakness

- may present insidiously with apparent lethargy and failure of motor milestones in young children
- ***sensory loss*** is usually minimal or transient
- muscular back & leg ***pain***, presumably neurogenic in origin, is common
- papilloedema and encephalopathy occasionally occur
- DVT and thromboembolism are not as significant a problem in children
- ***admission*** criteria to ICU include,
  1. respiratory failure ≤ 30% of patients will require mechanical ventilation
  2. severe autonomic disturbance
  3. bulbar palsy
  4. rapidly progressive weakness

- early indications for elective ***ventilation*** include,
  1. ↑ work of breathing
  2. fatigue with a poor cough
  3. arterial hypoxaemia - $SpO_2 \leq 90\%$
  4. progressive bulbar palsy

- ***hypercarbia*** is a late sign and should be avoided
- FVC is difficult to assess in children but successful weaning is unlikely unless,
  1. vital capacity ≥ 12 ml/kg , or
  2. peak inspiratory pressure ≥ -20 cmH_2O

- ***Differential Diagnosis***

a.	botulism	- clostridium toxin in blood
b.	tick toxin	- presence of a tick bite
c.	OP poisoning	- reduced serum cholinesterase levels
d.	myasthenia gravis	- deep tendon reflexes present
e.	transverse myelitis	- presence a sensory level
f.	motor neurone disease	- weakness is asymmetrical
g.	dermatomyositis	- presence of rash, muscle pain and increased CPK
h.	periodic paralysis	- history of previous episodes - increased or reduced potassium
i.	posterior fossa tumour	- spinal long tract signs

- CSF findings,
    1. normal pressure & clear
    2. ≥ 90% have ***increased protein*** ≥ 400 mg/l → mainly ***albumin***
    3. cell count / mm^3 < 50 lymphocytes
       < 2 PMN's
        - ≤ 10% have mild ***lymhpocytosis***

- nerve conduction studies show
    a. normal, slow or non-existent nerve conduction
    b. reduced amplitude of motor potentials

- ***autonomic dysfunction*** may be a serious problem, especially with airway manipulation or other procedures,
    a. cardiac arrhythmias
    b. hyper / hypotension
    c. urinary retention
    d. GIT dysfunction

- however, autonomic dysfunction is ***uncommon*** in children

- ***plasmapheresis*** within 7 days of onset may reduce the period of ventilation and reduce the time to recovery (no controlled trials - only adults)

- ***gammaglobulin*** may be of benefit in severe cases and in cases of ***relapsing polyneuropathy***
- steroids and other immunosuppressives are of ***no*** proven benefit
- other problems peculiar to long-term IPPV in the paediatric patient include,
    a. emotional immaturity
    b. speech failure
    c. fear of procedures
    d. family disruption

NB: the prognosis for GBS is ***better*** in the paediatric group

full recovery is likely if the time from maximal deficit to start of recovery is less than ***18 days***

Reye's Syndrome

Def'n: severe ***metabolic encephalopathy*** with cerebral oedema and fatty degeneration of the viscera, especially the liver

- occurs almost exclusively children, usually ≤ 15 years
- the ***aetiology*** is unknown, however,
  a. incidence is higher during epidemics of ***influenza*** or ***varicella***
  b. relationship to ***salicylates*** is controversial
  c. children with ***juvenile RA*** taking salicylates are at risk →
     i. ? viral
     ii. ? drugs (aspirin) / toxins cf. post-vaccination encephalitis
  d. abnormal ***mitochondrial function*** in hepatocytes
     → disturbed ***carnitine*** / coenzyme-A metabolism

- liver histology shows ***non-inflammatory*** microvesicular fat deposition
- EM studies show swollen and disrupted mitochondria

- the ***toxic encephalopathy*** is characterised by,
  a. progressive, generalized CNS damage
  b. severe, refractory cerebral oedema (usual cause of death)
  c. neuronal damage

▪ ***Clinical Picture***

a.	prodromal URTI	± exanthem
b.	intractable ***vomiting*** is often the first symptom	
c.	***encephalopathy***	- progressing over hours to days - personality change / agitation ± convulsions / coma - normal CSF (if no coagulopathy)
d.	hepatic failure	- from mild to fulminant - hepatocellular enzyme elevation - hyperammonaemia - coagulopathy & prolongation of PT - hypoglycaemia rare unless ≤ 2 yrs * mild jaundice, bilirubin seldom > 50 µmol/l
e.	MOSF	- cardiac failure - pancreatic failure
f.	mortality	~ 50% (T.OH states ~ 25% overall) ~ 100% for stages ≥ 4 (see below)

- ***Treatment***

a. control of raised ICP

b. manage liver failure
   i. coagulopathy
   ii. prevention of ***hypoglycaemia***
   iii. minimise ammonia load

c. support renal function

d. high dose ***l-carnitine*** ? may prevent progression in stage 2

Staging in Reye's Syndrome - Lovejoy			
Stage	**Coma**	**Pain response**	**Reflexes**
1	lethargy	normal	normal
2	combative	variable	pupils slow
3	coma	decorticate	pupils slow
4	coma	decerebrate	pupils slow
5	coma	flaccid	no δ pupils no occulo-cephalic

- ***Differential Diagnosis***

a. meningitis

b. encephalitis

c. fulminant hepatic failure

d. pancreatitis

e. inborn errors of metabolism

f. drugs or poisons

SPINAL TRAUMA

- paediatric spinal trauma is relatively rare → ~ 5% of all spinal injuries
- of children with severe trauma ~ 5% will have a cervical spine injury
- injuries will occur at more than one spinal level in ~ 16% of cases
- the commonest causes are,

    a. road trauma - MVA, pedestrian, cyclist

    b. falls - especially diving

- anatomical differences include,

    a. interspinous ligaments & joint capsules are more flexible

    b. uncinate articulations are poorly developed & slide forward

    c. the facet joints are flat

    d. the vertebral bodies are wedged anteriorly & slide forward with flexion

    e. the head is relatively large
    → greater angular momentum can be generated with flexion / extension

- ***normal*** radiological variations include,

    a. anterior displacement of C_2 on C_3 ~ 40% < 7 yrs
    ~ 20% ≤ 16 yrs
    ± 3 **3mm** movement

    b. increased distance between the dens and anterior arch of C_1 ~ 20% of children

    c. skeletal ***growth centres*** may resemble fractures

    d. basilar ***odontoid synchondrosis*** appears as a radiolucent line at the base of the dens (especially ≤ 5 years)

- spinal cord injury without radiographic abnormality, ***SCIWORA*** is almost unique to the paediatric age group

    a. ~ **20-60%** of all SCI

    b. ~ 30-50% of these the lesion is complete

- SCI in the ***first decade*** of life is,

    a. almost exclusively ***high cervical*** ~ $C_{1/2}$

    b. either subluxation or SCIWORA and severe cord injury

    c. rarely associated with fractures

• a high proportion of children who die in MVA's, or suffer cardiorespiratory arrest prior to reaching hospital have cord trauma above C_3, particularly at the ***cervico-medullary junction***
• this is difficult to diagnose in the unconscious patient, signs including,

a. flaccid immobility & areflexia

b. hypoventilation with paradoxical chest movement

c. apnoea and rhythmic flaring of the alae nasi (above C_3)

d. hypotension with
- inappropriate bradycardia
- peripheral vasodilatation
± priapism

Spinal Shock

• the syndrome of spinal shock occurs more commonly in children,

a. SCI lesion resolves after 2-3 days

b. progressive return of reflexes → bulbocavernous & anal first

c. incomplete lesions may then become apparent
   i. Brown-Sequard hemisection
   ii. anterior cord lesion
   iii. central cord lesion

Non-Accidental Injury

a. physical
b. sexual and emotional abuse
c. ***deprivation*** of medical care and nutrition

- children are also intentionally poisoned, and endure the consequences of inadequate supervision
- diagnosis of children who suffer from abuse or neglect is difficult
- NAI should be suspected where,

a. an injury is unexplained
b. the history is not consistent with the type of injury
c. it is alleged that the injury was self-inflicted
d. relatives delay in seeking medical aid
e. there are repeated suspicious injuries

- the history is rarely volunteered by the child
- the pattern of physical findings can be helpful,

a. bruises and scars on the back and buttocks in different stages of development and of unusual shapes
b. burns from cigarettes or forced immersion in hot water
c. ***retinal haemorrhages*** occur with head shaking, but also have other causes
d. head injury - skull fractures
- subdural haematomas
e. overt bone fractures or ***healing fractures***

- when non-accidental injury is suspected, referral to a specialised child protection unit to enable appropriate counselling and intervention is helpful
- safety of siblings must be considered